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AUTOMATED GEOCODING OF ROUTINELY COLLECTED HEALTH DATA IN NEW SOUTH WALES

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Almost every record of an individual's contact with the NSW health system contains some form of spatial information, whether a street address or postcode. This information can be used to assign a geocode to the record, which in turn can be used to examine the spatial distributions of disease and health service utilisation. This article describes a study that compares two different methods of geocoding addresses from routinely collected administrative health data to enable small area analysis.

WHAT IS GEOCODING?

Geocoding is the process of allocating geographical coordinates (such as latitude and longitude) to an address, thus defining the position of the address on the Earth's surface. The geocode itself can be used in the analysis or, alternatively, the geocoded record can be assigned to a spatial unit, such as a census collection district, that is smaller than other spatial units generally available (for example, postcodes), and then analysed. Spatial analysis of routinely collected health data in Australia has generally been limited to larger spatial units such as local government areas or area health service boundaries.[1,2]

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While spatial analysis at this level can be useful, many interesting spatial features that occur at a smaller geographical level can be lost in the aggregation of data to larger units.

GEOCODING PACKAGES

FEBRL (Freely extensible bio-medical record linkage)

NSW Health and the Australian National University have recently developed FEBRL 'freely extensible bio-medical record linkage'.[3] Before geocoding, FEBRL firstly 'cleans' the data by transforming the original text address into a standardised format that corrects for missing, erroneous or abbreviated data (for example, 'st' is transformed to 'street', 'pde' is transformed to 'parade'). The data are then 'parsed' by separating the address into individual standardised elements (for example, the element for 'street' or 'parade' is 'wayfare type'). FEBRL can then match the cleaned address data to the Australian Geocoded National Address File (G-NAF)[4], using a probabilistic algorithm, and allocate a geocode. The G-NAF contains 12.6 million unique geocoded addresses derived from a variety of national and state-based datasets. The geocode is provided for the centre (or centroid) of a property parcel. The G-NAF is updated every quarter.

MapMarker

MapMarker is a commercial geocoding package developed by MapInfo Australia[5]. MapMarker cleans and parses the address data and then links the cleaned address to an Address/Co-ordinate Dictionary, using fuzzy logic and Soundex indexing. Soundex is an algorithm for phonetic name encoding which indexes names by their English pronunciation to overcome minor differences in spelling. Secondly, the Address/Co-ordinate Dictionary, which is MapInfo's StreetWorks Australia database, is based on street centerlines and town/postcode centroids. Each street is broken into linear segments with a coordinate pair at both endpoints. Linear interpolation between the endpoints provides geocode coordinate values for a given address. Figure 1 illustrates the allocation of geocodes using the MapMarker street centre line interpolation method compared with the allocation of geocodes using the G-NAF land parcel centroid method.

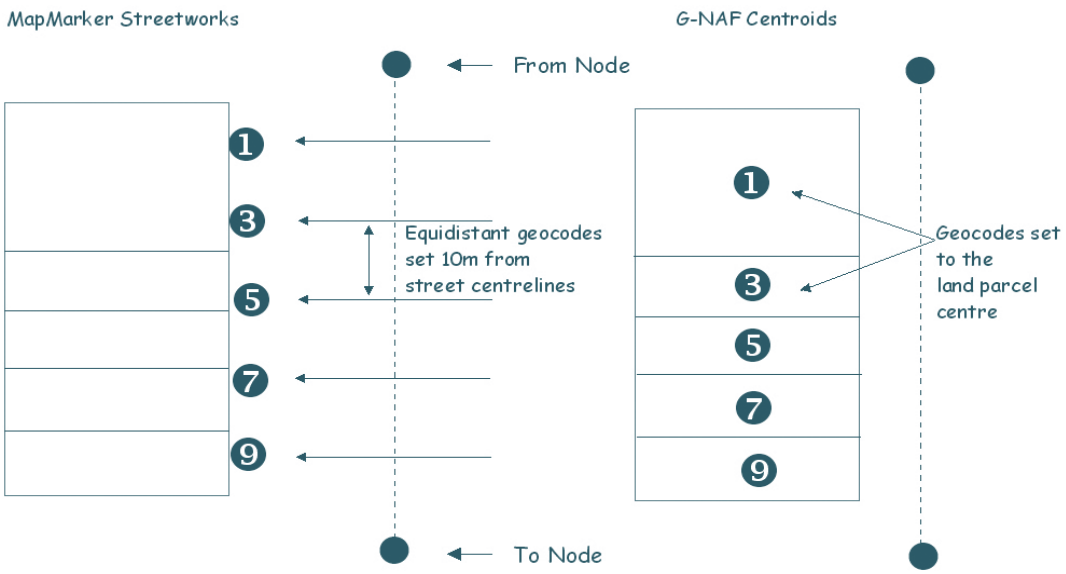
APPLYING GEOCODING

There is growing recognition of the value of geocoding administrative datasets[6] to enhance their usefulness for service planning and resource allocation.[7,8] Geocoded health events can also be used to investigate the possible effect of environmental hazards (often using proximity of residential address to a source as a proxy for exposure) such as: congenital malformation and proximity to a hazardous waste sites[9]; childhood asthma and proximity to roads[10]; and childhood leukaemia and exposure to electromagnetic fields. [11] Geocoded health data is also being used to investigate the epidemiology of specific diseases including adverse birth outcomes and childhood leukaemia.[12]

While a small number of outcomes can be geocoded individually, geocoding large numbers of records in routinely collected health databases requires well-defined geocoding procedures. Australia has lagged behind internationally in

FIGURE 1

EXAMPLE OF MAPMARKER INTERPOLATED HOUSE NUMBER WITH AN OFFSET OF 10 METRES FROM STREETLINE CENTRE, COMPARED TO A G-NAF LAND PARCEL CENTROID FOR THE SAME ADDRESSES.



developing standards for defining a geocoded address.[13] Limitations for accurate geocoding include: errors in the address/street reference data; imprecision in the algorithms used by geocoding software[14]; and errors in the original addresses such as post box addresses or property names.[15] Lack of precision in addresses, especially in rural areas, can bias urban and rural comparisons and limit the opportunity for small area or point source analysis.[16] Studies generally find that 60–80 per cent of addresses can be assigned a geocode; however, few studies have examined the accuracy of these geocoded addresses.[17,18]

This study compares the performance of FEBRL and MapMarker in the geocoding of addresses using routinely collected administrative health data from the NSW Central Cancer Registry.

METHODS

The authors obtained ethics approval for use of NSW Central Cancer Registry data, including residential addresses, on 888 cases of childhood leukaemia in children aged up to 14 years and diagnosed in NSW between 1990 and 2002. Using both FEBRL (version 0.3 and G-NAF version May 2005, MapInfo 2005 postcode boundaries) and MapMarker (version 7.0, MapInfo 2003 postcode boundaries), we geocoded these cases and compared the match status for each case.

Both products assign a geocode 'match status' category that indicates the precision of the assigned geocode, summarized in Table 1. FEBRL has several non-geocoded categories for addresses that have multiple possible streets or localities. MapMarker provides a geocoded result for all addresses, at least to the postcode centroid. We developed a hierarchical protocol to identify the most accurately geocoded address between the two products, where an exact address match in FEBRL and MapMarker was considered the most accurate and a 'many' match in FEBRL and a postcode centroid in MapMarker the least accurate. The least accurately geocoded cases (addresses that were not allocated a geocode or were allocated only a locality centroid in FEBRL and only a postcode centroid in MapMarker) were then considered for clerical review.

Clerical review is an essential part of all automated geocoding procedures for verifying and improving the accuracy of geocoding. The options for clerical review are largely dictated by the resources available and the degree of spatial precision required. We used the following approach for clerical review:

1. The addresses of the most imprecise cases were checked for misspelling using the Internet site Whereis[19], a street address mapping website based on UBD digital street map data. Potentially misspelt addresses were amended and resubmitted to FEBRL for geocoding.
2. If FEBRL did not return an improved match status we used Whereis to get an approximate location for the address. This Whereis location was often a local name for a locality or wayfare, or an address not yet included in G-NAF. We then used GIS-Epi, a mapping tool developed by the NSW Department of Health, to try to find this locality or wayfare using StreetPro 8.5 (which displays street names below 1:20,000 scale).
   a. Where the local wayfare address coincided with a G-NAF address or vice versa (for example, a Les Darcy Drive Maitland address in the G-NAF coincides with an address on New England Highway Maitland), we assigned the G-NAF geocode.

TABLE 1

DEFINITIONS OF MATCHING STATUS IN FEBRL AND MAPMARKER GEOCODING SOFTWARE

FEBRL	
Exact Address	Matched to wayfare number, name, type and locality—unique latitude and longitude
Exact Street	Matched to wayfare name, type and locality—latitude and longitude based on the street centroid
Average Address	Multiple addresses found close enough in space to produce an average match (eg units in an apartment block)—unique latitude and longitude based on average address
Exact Locality	No matching on the street level—latitude and longitude of the locality (postcode) centroid
Many Addresses[a]	Multiple addresses found but not close enough in space to produce an average match—no geocode
Many Streets[a]	Match on street name but no other item and the streets appears in many localities—no geocode
Many Localities[a]	Match only on locality but there may be many localities with same name—no geocode
MapMarker[b]	
Exact street address match (S5)	Single close match—the record has been geocoded to an interpolated house number offset from the street
Street centroid match (S4)	Single close match—the record has been geocoded to the street centroid
Locality centroid (Z1)	No street address match—the record has been geocoded to the suburb or postcode centroid

a. This match status does not produce a geocoded result in FEBRL (beta version)

b. MapMarker (v7.0) further classifies S5 and S4 by subcodes: H (House number match), P (Street prefix match), N (Street name match), T (Street type match), S (Street suffix match), C (Town name match), Z (Postcode match)

b. If GIS-Epi found the wayfare, we identified the address, a nearby address, or wayfare centroid to estimate the geocode.
c. If GIS-Epi found the locality, we assigned the geocode to the estimated locality centroid.

RESULTS

The geocoding results for MapMarker and FEBRL are summarised in Table 2. FEBRL assigned a geocode for an 'exact' or 'average' address match to 719 (81 per cent) cases. Street centroid interpolation within a locality was given to 73 (8.2 per cent) cases as an exact street match. Where a locality (postcode or suburb) could be identified but no unique address matched, the locality's centroid was allocated to a further 58 (6.5 per cent) cases. The 38 (4.3 per cent) remaining cases that could not be matched by FEBRL were not assigned a geocode. MapMarker assigned a street level interpolated geocode to 766 (86.3 per cent) cases. Street centroids were allocated to 72 cases (8.1 per cent). The remaining 50 (5.6 per cent) cases were assigned the postcode centroid.

The major reasons for addresses not being geocoded in FEBRL or assigned only a postcode in MapMarker were due to errors in the original health record, such as incomplete addresses (eight records), post boxes (five records) or lot/property names (16 records).

The hierarchical protocol developed to identify the most accurately geocoded addresses between the two products is summarized in Table 3. This protocol can be used to select the most accurate geocode assigned between the two products depending on the positional accuracy required by a study. It also helps people to understand the level of spatial imprecision and the limitations this may have in any spatial analysis.

TABLE 2

GEOCODING MATCH STATUS FOR FEBRL AND MAPMARKER FOR 888 CASES OF CHILDHOOD LEUKAEMIA DIAGNOSED IN NEW SOUTH WALES FROM 1990 TO 2002

Geocoding method	MapMarker match status							
FEBRL match status	Interpolated Exact Address (S5)[a]		Street Centroid (S4)[a]		Postcode Centroid (Z1)[a]		Total	
	n	Row %	n	Row %	n	Row %	n	Row %
Exact Address	659	94.7	19	2.7	18	2.6	696	100.0
Average Address	23	100.0	0	0	0	0	23	100.0
Exact Street	41	56.2	29	39.7	3	4.1	73	100.0
Exact Locality	20	34.5	18	31.0	20	34.5	58	100.0
Many Addresses[b]	17	85.0	2	10.0	1	5.0	20	100.0
Many Streets[b]	1	16.7	3	50.0	2	33.3	6	100.0
Many Localities[b]	5	41.7	1	8.3	6	50.0	12	100.0
Total	**766**	**86.3**	**72**	**8.1**	**50**	**5.6**	**888**	**100.0**

a Codes used in MapMarker (v7.0)

b No geocode produced in FEBRL

TABLE 3

HIERARCHICAL APPROACH TO REFINING THE GEOCODING OUTPUT USING FEBRL AND MAPMARKER SOFTWARE

FEBRL output	Approach
Exact Address	Use FEBRL output.
Average Address	Use FEBRL output.
Exact Street Address	Use MapMarker result of Exact Address (S5); otherwise use FEBRL output.
Exact Locality	Use MapMarker output for cases with a MapMarker result of Exact Address (S5) or Street Centroid (S4). Depending on accuracy required, the remaining cases coded to postcode centroid (Z1) are either excluded depending on the required spatial resolution for the study or undergo clerical review.
Many Addresses, Many Streets or Many Localities	Use MapMarker output for cases with a MapMarker result of Exact Address (S5) or Street Centroid (S4). Depending on accuracy required, the remaining cases coded to postcode centroid (Z1) are either excluded, depending on the required spatial resolution for the study, or undergo clerical review.

The hierarchical protocol allocated a FEBRL geocode to 771 cases (87 per cent), a MapMarker exact address or street centroid geocode to a further 108 cases (12 per cent), leaving 29 cases (3 per cent) for clerical review. These 29 cases were considered the most imprecise, having either no FEBRL geocode but a MapMarker postcode centroid (9 cases) or only a postcode/locality centroid provided by both products (20 cases). These cases then underwent clerical review, resulting in a geocode being assigned to all 29.

DISCUSSION

Both products gave above average results for exact address matches (a match result of 70 per cent is often considered acceptable). [6]

For environmental epidemiological studies examining very small areas or point event analysis, where the address of individual cases of a disease can be modeled as the data unit rather than an area[20], the positional accuracy required is more likely to be met with FEBRL than MapMarker because of the use of property centroids in FEBRL compared to street interpolation in MapMarker.

There can be enough difference between the actual position and a street centreline interpolation for an address to be assigned to the wrong spatial unit, especially if the study uses small areas.[14] In a previous Australian study using a street centre-line product to compare property geocodes, an estimated 5-7.5 per cent of addresses were misclassified to another census collection district. [21] The size and shape of a study's spatial unit can influence the estimated disease rate (referred to as the Modifiable Area Unit Problem) and assignment of area level covariate/exposure data. [22–24] The use of property parcel centroids could reduce this misclassification. Verifying the actual positional accuracy of the geocode with ground proofing of the address was beyond the scope of this study.

Our evaluation of two geocoding products suggests both are acceptable for use with large data sets, providing geocoding cheaply and quickly, with each having trade-offs. FEBRL accurately assigned an exact address geocode to 81 per cent of subjects while MapMarker geocoded slightly more (86 per cent).

Our study was limited to 888 subjects with a particular diagnosis, but there is no reason to suspect that these results would not be applicable to other outcomes. Indeed, the authors recently geocoded over one million records for the Midwives Data Collection using a protocol similar to the one described above with similar results.

The G-NAF is the most complete, up-to-date and accurate coverage of Australian addresses available and is updated quarterly. When the FEBRL probabilistic mapping algorithm is complete, it will be one of the most sophisticated freely available geocoding software products in Australia.

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SHORT QUESTIONS FOR SURVEYS ABOUT BREAD AND CEREAL INTAKE: COMPARING MEASURES OF QUANTITY VERSUS FREQUENCY

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The use of short questions in population surveys can provide valuable information on food habits and how these habits change. In order to monitor trends in diet and make comparisons, standardised survey questions must be maintained over time. However, occasionally it may be desirable to replace older questions with improved ones. This study was commissioned by the NSW Adult Health Survey Program to compare the responses provided to older and more recent versions of questions to improve the data collected in health surveys on food habits.

INTRODUCTION

Information about population food consumption patterns and how these are changing is important for planning and improving nutrition-related health programs and services. Although a national system of on-going nutrition surveillance, based on detailed assessment of dietary intakes, has been recommended for Australia, standardised short questions about food habits, which can be included in population surveys at minimal cost, can provide valuable information on some indicators of diet relevant to dietary guidelines and other nutrition policies.[1] The advantages of using these types of questions are:

- they impose a low respondent burden and yield higher response rates than other dietary assessment methods
- they can be administered by telephone or mail
- responses can be readily and rapidly analysed and reported.[2]

However, short questions supply only limited information on aspects of food consumption and they are therefore used mainly to classify people into broad groups of 'higher' and 'lower' intakes and 'frequent' or 'infrequent' consumption, and to classify types of foods within a category. Short questions cannot provide accurate quantitative estimates of food or nutrient intake; they are mainly useful for surveying groups, not individuals; and they may not be sensitive enough to detect small but important changes over time.[2] Short questions are increasingly being used by state and territory health departments, national data collection and information agencies, community dieticians, public health nutritionists and epidemiologists.

The validity of several short questions about food habits has been evaluated in adults.[2,3] Among short questions relating to bread and cereal intake, only those about frequency (but not quantity) have been previously validity-tested.[3] The results showed that, compared to 3-day weighed records, reported frequency of intake was relatively accurate, although subjects at the upper end of the distribution for quantity tended to overestimate their frequency of consumption while subjects at the lower end tended to underestimate their frequency of intake.

An important principle in monitoring trends in diet is to maintain standardised survey questions, so as to enable comparisons over time.[2] However, experience suggests that some survey questions are more difficult for respondents to answer than others, and therefore it is necessary, on occasions, to revise the questions. When replacing one short question with another, it is recommended there be a period of overlap so that the agency doing the survey can obtain comparable data, while calibrating the old question to the new.

Information about bread and cereal consumption is useful in monitoring population adherence to the Australian Dietary Guideline that recommends 'eating plenty of cereals (including bread, rice, pasta and noodles)'.[4] The 1995 National Nutrition Survey, using a more detailed dietary assessment, found that only a small proportion of people met the recommended number of serves (adjusted for age, sex and energy intake) of bread and cereals.[5] It is therefore appropriate that health surveys include short questions in an attempt to monitor the direction of change in habits relating to these foods.

Prior to 2002, the NSW Adult Health Survey Program[6] assessed bread and cereal intake using three short questions relating to the quantity consumed. These questions were replaced after a period of overlap with three new short questions relating to frequency of intake (Box 1). The reason for changing the questions was that frequency questions appeared easier for respondents to answer, and would not compromise validity, given study results showing a correlation between frequency and quantity of intake.[3] This paper reports on the comparison of the responses to three short survey questions on the frequency and quantity of bread and cereal consumption used in the 2002 NSW Adult Health Survey.

METHODS

Data used in this study were from all adults who participated in the 2002 NSW Adult Health Survey. This survey is part of an ongoing survey of the health of people in NSW using computer-assisted telephone interviewing (CATI) of a random sample of NSW adults aged 16 years and older, including those who speak a language other than English.[6] All survey participants were asked three short questions relating to their 'frequency' of bread and cereal consumption, as well as one of the three 'old' short questions, relating to quantity of consumption of either

BOX 1

SHORT SURVEY QUESTIONS ABOUT THE QUANTITY AND FREQUENCY OF BREAD AND CEREAL CONSUMPTION USED IN THE NEW SOUTH WALES ADULT HEALTH SURVEY 2002

'OLD' QUESTIONS RELATING TO QUANTITY OF INTAKE

1. How many slices of bread do you usually eat each day? (one slice of bread is equal to 1 small bread roll or 1 bagel or ½ a large bread roll or ½ bread muffin or 1 scone or ½ a pita bread)
2. How many cups of breakfast cereal do you usually eat each day? (one cup is equal to 1 cup of cornflakes or other flake-based muesli; 2 weetbix; ½ cup of cooked porridge; 1/3 cup of oat-based muesli or ½ cup of allbran)
3. How many cups of cooked pasta, rice, noodles or other cooked cereals do you usually eat each week? (not including cooked breakfast cereals)

Response options:

Number of slices/cups per day
Number of slices/cups per week
Don't eat bread/breakfast cereal/cooked cereal
Don't know
Refused

'NEW' QUESTIONS RELATING TO FREQUENCY OF INTAKE

1. How often do you usually eat bread? (include bread rolls, flat breads, crumpets, bagels, English or bread type muffins)
2. How often do you eat breakfast cereal? (ready-made, home-made or cooked)
3. How often do you eat pasta, rice, noodles or other cooked cereals? (not including cooked breakfast cereals)

Response options:

Number of times per day
Number of times per week
Times per month
Rarely or never
Don't know
Refused

bread, breakfast cereals or cooked cereals (see Box 1). Direct comparisons were made for each pair of questions dealing with quantity and frequency of bread, breakfast cereal and cooked cereal intake.

The distribution of responses for each short question was determined, using SPSS to recode the responses, to provide daily (for bread and breakfast cereal) or weekly (for cooked cereal foods) estimates of the frequency or number of serves. Spearman's correlation coefficients were used to assess the relationships between the non-parametric continuous data on quantity and frequency of intake. To assess agreement between categories on both questions a weighted kappa was calculated for each of the three comparisons. A weighted kappa is used for ordinal data, with higher weights given to categories furthest away from perfect agreement. For this analysis, responses for each of the quantity and frequency questions were classified into four groups using cut-points based on standard serves (quantity) and suggested by the distribution (frequency). Subgroup analyses were conducted for males and females and three age groups (16–34 years, 35–54 years, and 55 years and over).

SPSS 12.0.1 for Windows (SPSS Inc) was used for all statistical analyses except weighted kappas, which were calculated using SAS version 8 (SAS Institute).

RESULTS

The study population consisted of a total of 12,622 adult respondents, aged between 17 and 95 years (mean age was 52 years). Males were slightly under-represented (42.1 per cent) and females over-represented (57.9 per cent) in the survey sample, compared to 49.7 per cent males and 50.3 per cent females in the NSW population.[6] The sample was older than the NSW population with 46.5 per cent aged 55 years or over compared with 21.9 per cent, but the number of people born in Australia was similar (74 per cent and 70.5 per cent, respectively). The sample characteristics are further described in the Adult Health Survey 2002 report.[6]

The 'new' short questions on frequency of bread and cereal consumption were answered by 12,491 adults, giving an item-response rate of 99.0 per cent. Of these people, 12,389 also responded to one of the 'old' survey questions about quantity of consumption: 4074 for bread consumption, 4172 for breakfast cereal consumption, and 4143 for cooked cereal consumption.

Bread consumption

The comparison between reported quantity and frequency of daily bread consumption is presented in Table 1. The following examples illustrate how to interpret this table. Among respondents who consumed bread less than once a day, 71.9 per cent consumed less than two slices per day. Among those who reported consuming bread three or more times a day, 52.7 per cent consumed six or more slices per day.

The weighted kappa for all persons was 0.50, which suggests moderate agreement between the short questions on quantity and frequency of bread intake.[7] The lowest level of agreement between the two questions (that is, low kappa values) were observed for males and people aged 55

TABLE 1

DAILY BREAD CONSUMPTION: COMPARISON OF RESPONSES TO TWO SURVEY QUESTIONS, ONE MEASURING QUANTITY CONSUMED AND THE OTHER MEASURING FREQUENCY OF CONSUMPTION, FOR ADULT MALES AND FEMALES, NEW SOUTH WALES (N=4074)

Quantity: Number of slices per day	Sex	Frequency: Number of times per day 0-<1/day %	1-<2/day %	2-<3/day %	≥3/day %
<2 slices	Male	61.8	10.1	1.0	0.8
	Female	77.3	16.6	1.2	3.1
	All	71.9	14.1	1.1	1.7
2-<4 slices	Male	20.6	52.6	26.6	9.7
	Female	18.5	72.3	49.7	19.9
	All	19.2	64.4	39.3	13.7
4-<6 slices	Male	14.2	29.8	46.5	22.3
	Female	3.7	10.3	44.7	46.6
	All	7.4	18.0	45.6	31.9
≥6 slices	Male	3.4	7.5	25.9	67.2
	Female	0.5	0.8	4.4	30.4
	All	1.5	3.5	14.0	52.7
Total	**All**	**100.0**	**100.0**	**100.0**	**100.0**

Source: New South Wales Adult Health Survey 2002

TABLE 2

DAILY BREAD CONSUMPTION: COMPARISON OF RESPONSES TO TWO SURVEY QUESTIONS, ONE MEASURING QUANTITY CONSUMED AND THE OTHER MEASURING FREQUENCY OF CONSUMPTION, FOR THREE AGE GROUPS, NEW SOUTH WALES (N=4074)

Quantity: Number of slices per day	Age group (years)	Frequency: Number of times per day 0–<1/day %	1–<2/day %	2–<3/day %	≥3/day %
<2 slices	16–34	69.1	12.6	1.1	3.6
	35–54	74.3	14.6	1.3	1.8
	>55	70.8	14.3	1.0	0.9
2–<4 slices	16–34	16.8	59.0	29.1	6.0
	35–54	18.0	64.2	32.2	7.1
	>55	22.8	67.2	47.0	20.3
4–<6 slices	16–34	12.8	23.7	48.1	18.1
	35–54	6.1	17.3	47.9	28.3
	>55	4.8	16.0	43.2	39.2
≥6 slices	16–34	1.3	4.7	21.6	72.3
	35–54	1.6	4.0	18.6	62.8
	>55	1.6	2.5	8.8	39.6
Total	**All**	**100.0**	**100.0**	**100.0**	**100.0**

Source: New South Wales Adult Health Survey 2002

years and over (0.44–0.46), while the highest agreement was found for those in the younger age groups (0.53–0.55). In the age group 55 years and over, respondents who consumed bread three or more times a day, ate fewer slices of bread than those in younger age groups (Table 2). The Spearman's correlation coefficient was 0.67 for all persons, and ranged between 0.62 (males) and 0.72 (35-54 years) for subgroup analysis.

Breakfast cereal consumption

The comparison between reported quantity and frequency of daily consumption of breakfast cereal is presented in Table 3. The weighted kappa for all persons was 0.77, with the lowest agreement (kappa values) being for those aged 16–34 years (0.71) and the highest for females (0.79). These kappa values suggest good agreement between the two

TABLE 3

DAILY BREAKFAST CEREAL CONSUMPTION: COMPARISON OF RESPONSES TO TWO SURVEY QUESTIONS, ONE MEASURING QUANTITY CONSUMED AND THE OTHER MEASURING FREQUENCY OF CONSUMPTION, FOR ADULT MALES AND FEMALES, NEW SOUTH WALES (N=4172)

		Frequency: Number of times per day			
Quantity: Number of cups	**Sex**	**0/day**	**0–<1/day**	**1–<2/day**	**≥2/day**
		%	**%**	**%**	**%**
0 cups	Male	99.9	10.4	0.1	0.0
	Female	98.4	9.5	0.0	0.0
	All	98.7	9.8	0.0	0.0
0–< 1cup	Male	0.8	52.1	2.6	7.7
	Female	0.9	56.8	6.3	0.0
	All	0.8	55.0	4.8	5.6
1–<2 cups	Male	0.2	29.3	66.1	7.7
	Female	0.7	29.7	80.6	60.0
	All	0.5	29.5	74.6	22.2
≥2 cups	Male	0.0	8.2	31.1	84.6
	Female	0.0	4.0	13.1	40.0
	All	0.0	5.7	20.6	72.2
Total	**All**	**100.0**	**100.0**	**100.0**	**100.0**

Source: New South Wales Adult Health Survey 2002

TABLE 4

DAILY COOKED CEREAL CONSUMPTION: COMPARISON OF RESPONSES TO TWO SURVEY QUESTIONS, ONE MEASURING QUANTITY CONSUMED AND THE OTHER MEASURING FREQUENCY OF CONSUMPTION, FOR ADULT MALES AND FEMALES, NEW SOUTH WALES (N=4143)

		Frequency: Number of times per week			
Quantity: Number of cups per day	**Sex**	**0/week**	**0–<3/week**	**3–<7/week**	**≥7/week**
		%	**%**	**%**	**%**
0 cups	Male	96.5	8.6	0.4	1.9
	Female	95.6	7.6	0.6	0.4
	All	96.1	8.0	0.5	1.0
0–< 3 cups	Male	2.0	75.1	6.1	12.2
	Female	2.0	79.6	7.9	8.6
	All	2.0	77.7	7.2	10.0
3–<7 cups	Male	0.5	12.4	84.7	9.6
	Female	1.0	9.3	84.7	16.0
	All	0.7	10.6	84.7	13.5
≥7 cups	Male	1.0	4.0	8.8	76.3
	Female	1.5	3.5	6.8	74.9
	All	1.2	3.7	7.6	75.5
Total	**All**	**100.0**	**100.0**	**100.0**	**100.0**

Source: New South Wales Adult Health Survey 2002

questions.[7] The Spearman's correlation coefficients ranged from 0.85 (55 years and over) to 0.91 (35–54 years).

Cooked cereal foods consumption (pasta, rice, noodles, other)

The agreement between the reported quantity and frequency of cooked cereal foods consumed was similar to that of the breakfast cereal consumption, with an overall weighted kappa of 0.75 (Table 4). Weighted kappas were similar (0.73–0.76) for males and females and all three age groups. Spearman's correlation coefficient was 0.84 for all persons, and ranged between 0.81 (16–34 years and 35–54 years) and 0.85 (55 years and over and all females) for subgroup analyses.

DISCUSSION

This study found moderate to good agreement between responses to the three short survey questions about the quantity and frequency of consumption of breads and cereals that were included in the New South Wales Adult Health Survey 2002. Those who reported higher frequency of intake also reported eating greater quantities. The sub-group analysis showed a similar pattern of moderate to good agreement for both males and females and for the three age groups. Agreement between responses to the quantity and frequency questions was generally higher for females than males, and higher for younger compared with older respondents. This was most pronounced for the questions about bread. This finding may reflect real differences in consumption patterns. For example, males reported consuming more slices of bread per eating occasion than females, and respondents aged 55 years and older tended to eat bread more frequently but in smaller amounts, than younger respondents. Thus, some measurement error will be introduced by substituting a short question about quantity with a question about frequency of bread intake for these sub-groups. Extrapolation about quantities should be made with caution and separate estimates made for males and females and for people aged over 55 years. Agreement between quantity and frequency was higher for the breakfast cereals and cooked cereals questions than for the bread question among sex and age subgroups.

A limitation of the analysis was that we could not compare the total number of bread and cereal serves. Respondents were asked only one of the three 'quantity' questions about breads and cereals because the survey team was concerned about respondent fatigue. Another limitation of this study was the absence of a gold standard to enable simultaneous validity-testing of both the quantity and frequency questions. However, the validation study by Riley et al[3] comparing frequency and quantity of bread and cereal consumption, measured by short survey question and 3-day weighed food records, provides support for the use of frequency questions as a proxy for questions about quantity. Their findings suggest that frequency and quantity are related and that short questions about frequency can discriminate between higher and lower consumers, but the point estimates should not be interpreted literally. Notable difficulties in quantifying bread and cereal intake are that people find it difficult to identify all the foods they consume that belong to the breads and cereals group (for example bagels, pizza and pie crust, risotto, etc), and that usual portion sizes consumed are difficult to estimate with accuracy.[8]

Based on the findings of this study, we recommend that the three frequency questions be used as the standard short questions for assessing bread and cereal consumption among adults. Further validity-testing of short questions relating to bread and cereal consumption, along with questions about other food groups of public health interest, is recommended. In particular, we suggest that the short questions on frequency of intake be tested for validity with more detailed dietary intake assessments to quantify the extent of measurement error at various levels of consumption. This is particularly relevant due to the sex and age differences in the relationship between frequency and quantity measurement of bread consumption. It is likely that short survey questions will not provide accurate quantitative estimates of bread and cereal consumption for individuals, but may be able to provide interim information between detailed dietary surveys about changes in the frequency of consumption, and an indication of the direction of change in quantities.

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A TUBERCULOSIS CONTACT INVESTIGATION INVOLVING TWO PRIVATE NURSING HOMES IN INNER WESTERN SYDNEY IN 2004

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Australia has one of the lowest incidence rates of tuberculosis (TB) in the world, approximately five per 100,000 per year [1], although in some parts of the country the incidence is considerably higher. In the former Central Sydney Area Health Service the incidence rate in 2003 was 14 per 100,000 per year.[2] The incidence is also higher in those aged 65 years and over[1], a population that has declining immunocompetence due to a variety of factors.[3] Furthermore, the subpopulation of this group (and, indeed, of people of any age) living in residential institutions such as nursing homes and hostels are at even greater risk of TB infection and disease due to their chronic ill health and multiple medical problems.[3] Despite this, there are no guidelines in NSW regarding TB screening of the elderly, either in response to potential exposure or with regard to screening at entry to a residential facility. There are also very few reports in the literature of TB contact investigations in residential facilities. Those that have been published come from the United States and are concerned with TB in hospitals or correctional facilities.[3,4,5,6] There has been one report of TB transmission in a school setting in Sydney[7], but there is a paucity of Australian literature that clarifies what is required for contact investigations in the local residential care setting.

This paper describes a contact investigation resulting from a case of active TB in a health care worker employed by a number of nursing homes in inner-western Sydney and highlights the need for policies with regard to TB screening of the elderly residing in nursing homes.

METHODS USED IN THE INVESTIGATION

The index case

The index case was an overseas-born female health care worker residing in Sydney. Prior to the woman's diagnosis of TB in January 2004 she was asymptomatic. She was diagnosed as a result of contact investigation of another case of TB infection. Microscopic examination of sputum demonstrated 1+ acid-fast bacilli, and polymerase chain reaction was positive for *Mycobacterium tuberculosis*. Sputum culture grew *M. tuberculosis* sensitive to all first-line anti-tuberculous medications. Chest CT scan demonstrated no pulmonary cavities but did show areas of consolidation and mediastinal lymphadenopathy with partial collapse of the left lower lobe, indicating significant lymph node disease. Because the sputum was direct-smear positive, the woman was assessed by an expert panel as potentially having been infectious for at least one month prior to her diagnosis. We identified that she had worked as an assistant-in-nursing in two nursing homes in the inner western suburbs of Sydney during this period.

Screening of nursing home residents and staff

All staff and residents in both nursing homes were considered contacts and evaluated. It was considered too late by the time of notification to undertake baseline screening of either the residents or staff. All staff were given a screening questionnaire to obtain information regarding: their country of birth; previous Bacille Calmette-Guerin (BCG) vaccination and tuberculin skin testing ('TST'); any history of TB; previous contact with patients or others with TB; and overseas travel history. Staff then underwent TST on-site at the two residential facilities, which was administered by chest clinic staff. If the TST was positive for latent TB infection (greater than 10mm if no previous BCG or 15mm if previous BCG), the staff member was referred to their local area health service chest clinic for a follow-up chest x-ray and review by a respiratory physician to investigate for TB disease and infection.

After interviewing the management of both residential facilities to determine the infected health care worker's patient care schedules, it was concluded that all residents of both facilities had had significant contact with her and therefore should be screened. Initial screening included review of the residents' medical histories specifically for the presence of risk factors for TB, symptoms of TB and any previous chest x-ray or TST results. Residents then underwent TST and chest x-ray (regardless of the TST result). This was based on the assumption that some of the elderly residents with multiple medical conditions might have depressed cell-mediated immunity, and therefore might have a negative TST despite being infected with TB.[8] The timing of screening was set at approximately five to six months following the diagnosis of TB in the health care worker, at which time her contact with the residents had ceased. Based on the little literature available[9], this was the optimal time to detect TB disease by chest x-ray before infectivity set in.

RESULTS

Review of the medical records of the nursing home residents' revealed that none had a previous TST recorded. Some residents did have previous chest x-rays, however, and none of these were reported as having findings consistent with TB. The results of initial TST screening at nursing home 1 (NH1) and nursing home 2 (NH2) are presented in Table 1. There was a notable difference between the two nursing homes in the number of positive TST results for residents, with more at NH2, and the proportion of TST

TABLE 1

RESULTS OF TUBERCULIN SKIN TESTING (TST) SCREENING FOR THE STAFF AND RESIDENTS OF TWO NURSING HOMES IN SYDNEY IN 2004

	Nursing Home 1		Nursing Home 2	
	n	%	n	%
Staff				
Staff potentially exposed	30	100.0	40	100.0
Staff screened	26	86.7	40	100.0
Staff with positive TST*	15	58.0	22	55.0
Residents				
Residents potentially exposed	75	100.0	83	100.0
Residents screened	74	98.7	83	100.0
Residents with positive TST*	5	6.7	14	16.9

*positive TST = greater than10mm if no previous BCG or 15mm if previous BCG

positive staff at both nursing homes was quite high (more than 50 per cent).

The infected woman had worked for the majority of the time in NH1. There were no changes indicative of TB or any other pathology found on follow-up chest x-ray of staff with positive TST. Of the four staff members who did not undergo TST, three did not attend for testing and the remaining person had a previous severe reaction with TST and therefore underwent chest x-ray only, which was negative for TB. One resident of NH1 refused to undergo TST but had a chest x-ray that was negative for TB, as were the chest x-rays for all other residents.

The TB-infected woman worked only six shifts at NH2 during the month in which she was determined to be infectious. Follow-up chest x-ray of NH2 staff with positive TST did not identify any who were likely to have acquired TB. Four (5 per cent) nursing home residents were found to have chest x-ray changes that required follow-up with a chest physician. On further investigation, one resident was diagnosed with carcinoma of the lung, two were discharged from follow-up after review by a chest physician (as chest x-ray changes were not consistent with TB) and one underwent follow-up chest x-rays to monitor for development of TB disease (now complete).

The sociocultural demographic characteristics were similar for both facilities in that the majority of the residents were of Anglo-Saxon descent and born in Australia. Therefore, one would expect a similar proportion of positive TST among the residents. NH2, however, had a disproportionate number of positive TST results, which were distributed throughout all three sections of the nursing home. As the TB-infected woman had only worked a small number of shifts at this facility, therefore it was hypothesised that there might be another staff member acting as a source case in NH2. According to NH2 management, only night shift and agency staff worked in all three sections of the facility. All night-shift staff had been screened (with negative results), so after consultation with experts in the field (necessary because of the paucity of literature on the subject), screening was expanded to include casual agency staff who had worked three or more shifts at NH2 in the previous six months, to exclude the possibility of another source case. In consultation with the management of NH2, it was found that all these staff were employed through the one nursing agency. Only 47 per cent of staff from this agency attended for screening, and of these 80 per cent had positive TSTs. These agency staff all had previous BCG vaccination and none had chest x-ray findings consistent with active TB disease. All but one of the agency staff were born overseas in TB-endemic countries. Reasons for non-attendance at screening by other agency staff are unknown due to reluctance on the part of the nursing agency to co-operate.

DISCUSSION

Whilst no further cases of active TB disease were detected and a second source of infection within NH2 was not found, this screening is noteworthy for raising a number of issues for which there is no clear policy direction, or local or international literature, to support decision-making.

In this contact investigation, there were no baseline TST results available for the residents. In NSW, there is no requirement or recommendation that nursing home residents are screened for tuberculosis upon entry to a residential facility. In contrast, the Northern Territory Centre for Disease Control[10] and the United States Centers for Disease Control (CDC)[3] recommend that all residents in long-term residential care be screened for TB with TST upon entry to the facility. In Victoria it is recommended that new residents undergo a baseline chest x-ray and TST.[11] The Victorian guidelines state that TST has a low predictive value in the elderly.

Consideration could be given to formulating policy in NSW that is similar to that of these other Australian states and of the CDC. This may be made easier to implement with

the development of a new in-vitro test, QuantiFERON®-TB Gold (Cellestis Limited, Carnegie, Victoria, Australia), for the diagnosis of both latent TB infection and TB disease, which the CDC recommends can be used in all circumstances in which the TST is currently used, including contact investigations.[12] However, this novel test should be validated for use in the elderly, and its cost effectiveness evaluated, before widespread implementation.

There were also no baseline TB screening results for staff of the nursing homes. The screening identified a reasonably high proportion of TST-positive staff, most likely related to the high level of staff born in TB-endemic countries. It is NSW Health policy that all health care workers in public health facilities must be screened for infectious diseases, including TB.[13] However, this policy is not mandatory for private health care facilities, though it is strongly recommended. There is a need for the introduction of strategies that will assist in the screening and management of TB contacts in private health care facilities in the future.

Because there was no baseline data, much consideration was required to decide on the most appropriate time, after potential exposure, for screening, especially in the elderly. During these deliberations it was found there was a paucity of literature, either international or Australian, addressing this question. There were also few reports of contact investigations following potential exposure to TB in the elderly. Reports such as these, with relevant outcome data, would assist in decision-making at the local level during future screenings as well as in formulating policy.

In addition, there are few published studies on the best means of screening in the elderly (TST versus chest x-ray), and the literature that is available is contradictory. International guidelines vary depending on the jurisdictions they cover. The CDC has no specific guidelines on the most appropriate course of action in the event of exposure to TB within the nursing home.[3,14] Even within the United States there are widely differing policies and practices regarding contact investigations.[15] The British Thoracic Society recommends chest x-ray to investigate for TB disease in nursing homes in the United Kingdom but does not recommend screening upon entry to a residential facility.[16] There are no national guidelines in Australia.

Consideration should be given to addressing this gap in policy, particularly in NSW. These policies and guidelines need to be produced using sufficient levels of evidence as outlined by the National Health and Medical Research Council.[17] A possible reason for not having specific guidelines for TB screening in the elderly is that this issue could be addressed on a case-by-case basis, through the use of an expert panel. However, if this were to occur (and be established in policy), published reports of the results of such contact investigations should be made available to assist with decision-making for future screenings.

CONCLUSION

While this particular TB contact screening did not result in further cases being found, the relevant and recurring issues described above were highlighted. These should be addressed through further research to collect process and outcome data on TB screenings in those aged 65 years and over, and that guidelines based on expert opinion (in the absence of available literature) be developed and uniformly applied, with continuous monitoring and evaluation. In this way, the best outcomes for the elderly put at risk of TB infection will be achieved through evidence-based best practice.

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NEW 'AIR POLLUTION ALERTS' WARN OF HEALTH RISKS

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Research has shown for some time that high levels of air pollution can exacerbate illness. More recently, evidence has emerged about how air pollution at much lower levels can also affect the health of susceptible people, such as those with asthma, chronic obstructive pulmonary disease or coronary artery disease. NSW Health and the Department of Environment and Conservation have started Australia's most comprehensive air quality warning system to ensure residents of Sydney, the Illawarra and the lower Hunter can take action to stay healthy at times of low, medium and high air pollution.

As different patient groups are sensitive to different types of air pollution, the warnings are tailored to draw attention to this – for example, people with asthma can be affected by several pollutants such as ozone and nitrogen dioxide from car exhaust and industry, whilst people with heart disease may be more affected by fine particle pollution.

The air quality alerts are based on information routinely collected by the Department of Environment and Conservation. In Sydney, where routine air pollution forecasting is also in place, an alert will be issued when high air pollution days are forecast. Alerts are distributed to the media and posted on the Department of Environment and Conservation's website. In the Illawarra and lower Hunter high monitored air pollutant levels will trigger the alert system.

The alert predicts the level of risk to sensitive individuals and suggests simple ways to reduce exposure and manage impacts. Under the new system, people are able to ring a free-call help line for all the latest information on air pollution levels, forecasts and alerts.

It is anticipated that the Air Pollution Health Alerts will be used by primary care providers to help reduce the effects of air pollution on sensitive individuals with chronic disease.[1] The brochures inserted into this edition of the *Bulletin* support this and provide broader advice about reducing the adverse impacts of air pollution.

In much the same way we look at the daily weather forecast and plan our day, the air quality alerts will help sensitive people plan activities around expected air pollution levels and take action to minimise health impacts.

The NSW Department of Health has established a web page with information about the alert system at the following address: www.health.nsw.gov.au/living/airpollution.html. This webpage provides a link to any current air pollution health alerts; information about air quality and health; and information brochures for the general public as well as health professionals.

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THE CHANGING EPIDEMIOLOGY OF PERTUSSIS IN THE HUNTER NEW ENGLAND AREA AND POTENTIAL IMPLICATIONS FOR THE IMMUNISATION SCHEDULE

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Notifications of pertussis in adults have recently shown an upward trend in the Hunter New England Area, while rates in adolescents have decreased. This article presents trends in pertussis notifications and rates for the Hunter New England region for the period 1998–2005, demonstrating a shift in pertussis incidence to older age groups that peak in the 50–64 age group.

A complete understanding of the epidemiology of *Bordetella pertussis* is complicated by imperfect diagnostic methods and incomplete recognition, testing and notification of the disease. It is generally agreed that notification rates represent an under-reporting of actual incidence.[1] However, Australia has improved surveillance for pertussis since the early 1990s and currently has the highest reported pertussis incidence rates of any country in the world. Between 4,000 and 10,000 cases have been reported each year since 1993, an incidence of between 22 and 58 per 100,000 population.[2]

In most countries, infants are given three doses of pertussis vaccine within the first six months of life. But despite high immunisation rates in infants and children, pertussis remains endemic, with epidemics also occurring every two to five years.[3,4] This pattern has not changed since the pre-vaccine era prior to the 1940s, and is probably caused by the continued transmission of pertussis among adolescents and adults, with infection of susceptible infants. Five countries—Australia, Austria, Canada, France and Germany—have responded to the rising incidence of pertussis in adolescents and adults by incorporating an adolescent booster dose in their immunisation schedules.

Preventing pertussis in children is one of the aims of the NSW Immunisation Program. In 2004, high school students were also targeted for a pertussis booster as this group was most affected by the epidemics that occurred in 1997 and 2000–2002.[5]

The Hunter New England Area Health Service provides health services, including immunisation services, to approximately 826,000 people across an area of 130,000 km^2 in northeastern NSW. In Hunter New England, infant immunisation coverage against pertussis has consistently exceeded 90 per cent during the past decade. Coverage of approximately 70 per cent of adolescents was achieved in Hunter New England during 2004. The impact of this initiative on the epidemiology of the disease is unknown. In addition, the immunity that follows pertussis disease is not long lasting.[6] It is therefore necessary to consider the changing epidemiology of pertussis, which in turn should inform the development of future immunisation strategies to limit disease transmission.

METHODS

Pertussis is a statutory notifiable disease in NSW, with cases notified by doctors, hospital chief executives, laboratories, school principals and directors of childcare facilities. In Hunter New England, pertussis notifications are routinely followed-up by the public health unit in partnership with general practitioners and other health workers. The follow-up serves to limit the spread of disease through appropriate education, contact tracing, and providing antibiotic prophylaxis to contacts.

For notification purposes in NSW, a probable case of pertussis is defined as a person with a coughing illness lasting for two or more weeks and paroxysms of coughing or inspiratory 'whoop' or post-tussive vomiting. A confirmed case requires isolation of *B. pertussis* or detection of *B. pertussis* by nucleic acid testing from a nasopharyngeal specimen. All suspected, probable and confirmed cases of pertussis within NSW are entered into the NSW Notifiable Diseases Database.

Notification data of confirmed pertussis cases from the NSW Notifiable Diseases Database for the Hunter New England Area for the period 1 January 1998 to 31 December 2005 was reviewed. Pertussis notifications were analysed for the date of onset of illness and the age group of the case. The following age groups were used in the analysis: 0-4 years, 5-19 years, 20-34 years, 35-49 years, 50-64 years, and 65 years and over. The narrower 0-4 year age group was used, as this group is particularly prone to serious illness.

For each year between 1998 and 2005, the proportional contribution of each age group to the overall number of pertussis notifications was calculated. To account for demographic changes in the Hunter New England population over this period, annual pertussis notification rates per 100,000 were also calculated for each age group for the years 1998-2005, using annual Census data estimates for the Hunter New England population. Chi square tests for linear trend were used to explore variations between age groups.

RESULTS

Between 1 January 1998 and 31 December 2005 there were 4,447 notifications of pertussis ranging from 313 in 1999 to 1,233 in 2000 (Figure 1).

There has been a change in the age distribution of pertussis notifications during this period. The proportional contribution of the 5–19 age group to total notifications

FIGURE 1

PERTUSSIS NOTIFICATIONS BY MONTH, HUNTER NEW ENGLAND AREA HEALTH SERVICE, NEW SOUTH WALES: JANUARY 1998 TO DECEMBER 2005

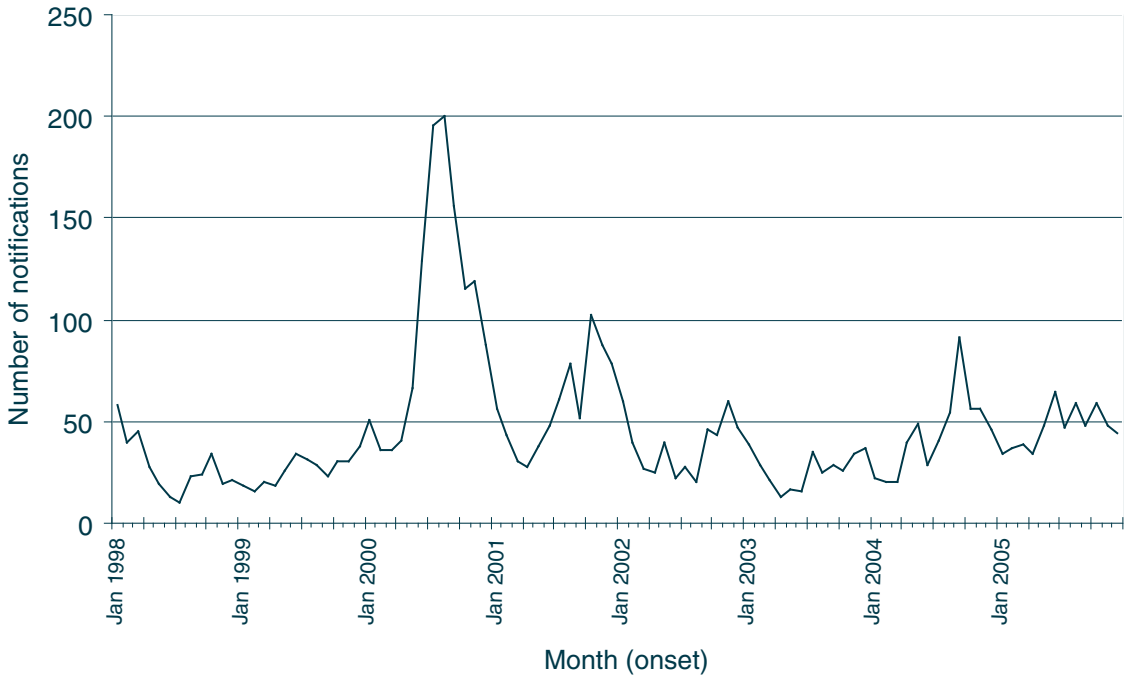


FIGURE 2

PROPORTIONAL CONTRIBUTION TO PERTUSSIS NOTIFICATIONS FOR SIX AGE GROUPS, HUNTER NEW ENGLAND AREA HEALTH SERVICE, NEW SOUTH WALES: JANUARY 1998 TO DECEMBER 2005

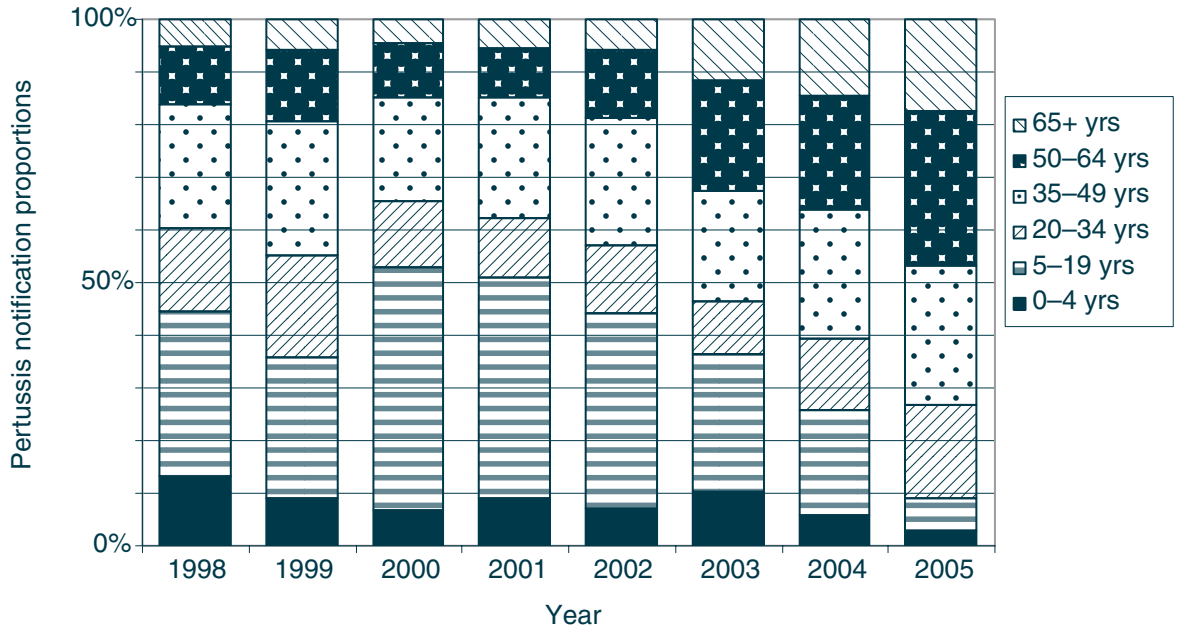
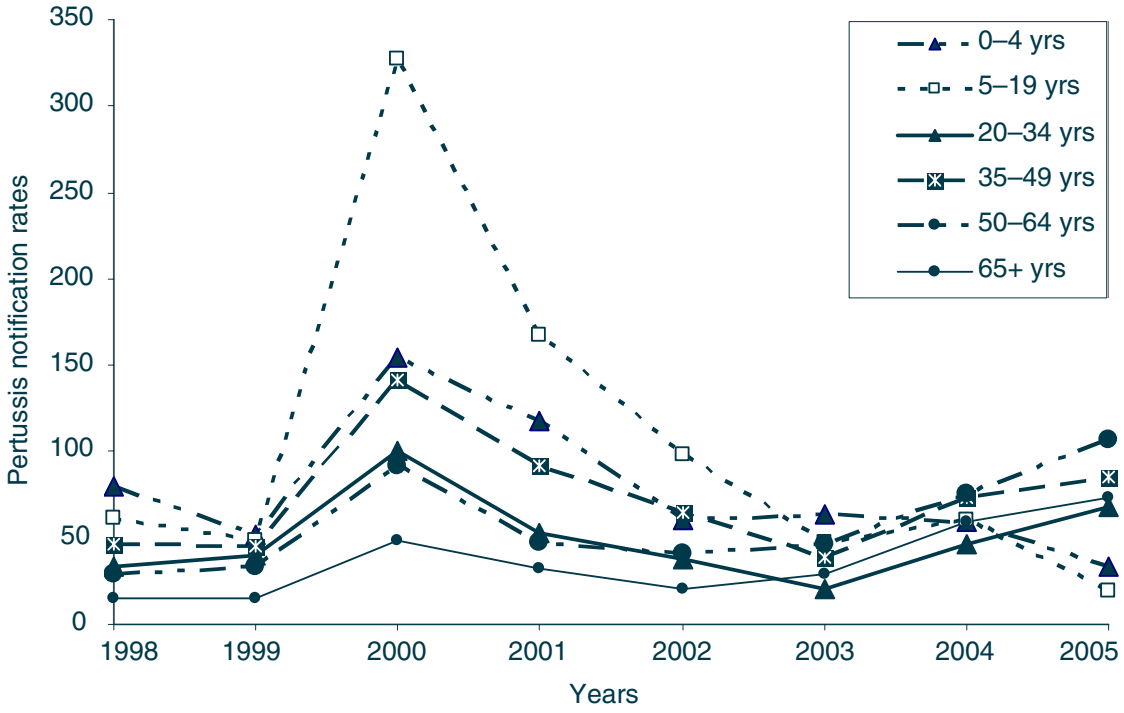


FIGURE 3

PERTUSSIS NOTIFICATION RATES PER 100,000, FOR SIX AGE GROUPS, HUNTER NEW ENGLAND AREA HEALTH SERVICE, NEW SOUTH WALES: JANUARY 1998 TO DECEMBER 2005



decreased from 46.3 per cent to 5.9 per cent during the period 2000 to 2005 (Figure 2). During the same period the proportional contribution of the 50–64 year age group increased from 10.1 per cent to 29.4 per cent (Figure 2).

Consideration of age group specific notification rates in Hunter New England from 1 January 1998 to 31 December 2005 demonstrated that the notification of pertussis in children aged 0–4 years peaked during 2000 at approximately 150 per 100,000, it then stabilised at approximately 60 per 100,000 per annum over the years 2002–2004, and has decreased further to 33.4 per 100,000 in 2005 (Figure 3). There has also been a decrease in notification rates in the 5–19 year age group from the epidemic level of 328 per 100,000 in 2000, to a low of 19 per 100,000 in 2005, a rate which is less than a third of the 2004 rate.

There have been important changes in the notification rates in older age groups since the 2000 epidemic, with a 6.9 per cent decrease in notified cases in the 35–49 year age group between 2001 and 2005, and a 26.3 per cent increase in notified cases in the 20–34 year age group during the same period. However, the greatest relative increase occurred in the 50–64 year age group, where there has been a 150 per cent increase in notified cases over this period, giving a notification rate of 107 per 100,000 in 2005—currently the highest of any age group (χ^2 linear trend = 128; $p<0.0001$).

DISCUSSION

Hunter New England notification data suggests that in this Area Health Service adults may be emerging as an important reservoir of pertussis. During the period studied, there were no changes in diagnostic criteria or particular initiatives to encourage physicians to increase testing for pertussis amongst adults with respiratory symptoms. Although the adolescent immunisation program appears to have reduced the burden of disease in this age group, cases still occur in the vulnerable 0–4 age group. These findings, however, should be compared with other NSW Area Health Services and other Australian jurisdictions.

Humans are the only known host for *B. pertussis*, with direct transmission of the organism presumed to occur through airborne contact with aerosol droplets from the respiratory tract of an infected, coughing individual. Clinical cases are highly infectious, with a primary case resulting in a mean of 17 secondary cases in an immune-naïve population.[7]

The occurrence of pertussis disease is affected by the waning nature of pertussis immunity, with immunity due to natural disease declining after 4 to 20 years and

protective immunity after vaccination declining after 4 to 12 years.[5] As disease becomes less prevalent in a population, opportunities for natural boosting are also reduced.[8]

The importance of this disease in adults has previously been neglected as it is much less severe than in infants, although not inconsequential.[9] There has recently been acknowledgement that a greater understanding of pertussis epidemiology in adults must inform control strategies as transmission in older age groups may expose vulnerable infants and children to infection.[10] Older age groups are recognised as playing an important role in transmitting *B. pertussis* infection to incompletely immunised infants.[11]

The administration of acellular pertussis vaccine boosters to adolescents and adults, if appropriately timed, should decrease the circulation of *B. pertussis* in the community.[12] Although the newly available pertussis vaccine is currently not on the Australian Standard Vaccination Schedule for adults, the National Health and Medical Research Council recommends a booster dose of dTpa (diphtheria and tetanus toxoids, and acellular pertussis vaccine) on a single occasion for any adult expressing an interest and suggests that dTpa may be considered at 50 years of age instead of ADT (adult diphtheria tetanus toxoids).[13] The high pertussis notification rates in adults, persisting severe disease, hospitalisation and deaths in infants despite high childhood and adolescent immunisation coverage, and availability of adult-formulated pertussis vaccines, make alternate strategies for vaccine control of pertussis, which are informed by mathematical modeling, of critical importance in Australia.[14] Control efforts need to take account of the dynamic epidemiology of pertussis, with due consideration of the cyclical epidemic pattern.

CONCLUSIONS

To optimally control pertussis in our community and reduce the risk of infection in the most vulnerable groups, it is essential not only to maximise pertussis immunisation rates in children and adolescents but also to consider expanding pertussis booster immunisation to include adults. This should be preceded by careful modeling of the impact of adult vaccination strategies on pertussis epidemiology, utilising techniques that take account of heterogeneous population mixing.

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LABORATORY DIAGNOSIS OF COMMUNICABLE DISEASES —PITFALLS AND PROSPECTS

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This is the first in a series of articles from the Centre for Infectious Diseases and Microbiology – Public Health (CIDM-PH) describing the clinical and microbial epidemiology of communicable diseases; and new developments in the identification and typing of pathogens of public health importance. The CIDM-PH is a research group based at Westmead Hospital that is closely allied to clinical and laboratory service divisions of CIDM and the Institute of Clinical Pathology and Medical Research (ICPMR).

Part one of this article provides an overview of current laboratory diagnostic methods, and part two illustrates how these tests are applied in practice. A glossary of terms is included to assist readers.

PART 1:

AN OVERVIEW OF CURRENT LABORATORY DIAGNOSTIC METHODS

The diagnosis of communicable diseases involves laboratory tests that either detect the presence of a potential pathogen (live organisms, antigen or nucleic acid) or a host response to a pathogen. Sensitivities, specificities (and so predictive values) vary between test categories, individual assays and laboratories. Interpretation requires knowledge, expertise and an understanding of the technical idiosyncrasies of assays. The turnaround times for results can vary from hours to weeks. This article is offered as a guide for the users of diagnostic laboratories, both clinicians and public health practitioners. Like any form of evidence, laboratory test results should be interpreted in light of other relevant information and questioned if they seem implausible.

SETTING THE SCENE

Clinical microbiology and serology are cottage industries compared with the rapid, automated systems used in clinical chemistry and haematology. Methods that rely on microbial culture are often too slow to influence the choice of treatment or disease outcome; serological testing often provides only a retrospective diagnosis—more useful for describing the epidemiology than a diagnosis. Microscopy is rapid, but neither sensitive nor specific, unless combined with an immunological stain for a particular antigen.

The development of rapid nucleic acid amplification tests (NAAT) has partially changed this scenario. NAAT are most widely used to detect organisms that grow slowly or not at all *in vitro*, require special facilities or are dangerous to handle. There is reluctance to replace the culture of rapidly growing bacteria with NAAT, because isolates are required for antibiotic susceptibility testing or subtyping. This could also change, with development of real-time multiplex methods that allow simultaneous amplification and identification of many microbial gene targets and could potentially provide all the data needed for patient management, disease surveillance and outbreak investigation.

The introduction of new methods does not overcome the need for careful interpretation of results, which requires an understanding of microbial virulence, microbial-host interactions—colonization, infection versus disease, and opportunistic infection—and the patient's current and relevant past medical and social history. Serological test interpretation must also take into account the timing of serum collection in relation to symptom onset, potential cross-reactions and the type of antibody being assayed. The purpose, limitations, sensitivity, specificity and predictive values of the test and the pretest probability of a particular diagnosis must be considered. Thus the interpretation of a test depends on whether it was done for population screening or case detection in someone who is asymptomatic but at risk due to, for example, exposure to an illness. A negative result rarely, if ever, excludes a diagnosis and a positive result should not be accepted as diagnostic proof in the absence of plausible clinical features or a history of exposure.

CULTURE—OPEN-ENDED OR SELECTIVE?

Despite new developments, culture remains the mainstay of diagnosis for many (mainly bacterial) infections. Bacterial culture can be broadly categorised according to the type of specimen collected and type of media used.

Culture of normally sterile specimens on enriched, non-selective media

Specimens that are normally 'sterile', for example blood, CSF and other fluids from enclosed body spaces, are routinely cultured in enriched, non-selective media on the assumption that anything isolated from a patient with a clinical infection is likely to be significant. Enriched liquid media, such as those provided in commercial, automated blood culture systems, will support the growth of most rapidly growing pathogenic bacteria—*Staphylococcus* species, *Streptococcus* species, *Neisseria meningitidis, Listeria moncytogenes,* enterococci, *E.coli,* salmonella, other Gram negative bacilli, and some highly fastidious species, such as *Brucella* species—which are responsible

for most cases of community and hospital-acquired septicaemia and meningitis.

Blood cultures will usually signal as positive in automated systems within 8 to 24 hours or occasionally longer, depending on the bacterial species, number of organisms in the specimen and whether they have been damaged by exposure to antibiotics. A Gram stain will give clues to the organism's identity, but full identification and antibiotic susceptibility testing takes another 1–3 days, or more, if further testing is required. Interpretation is usually straightforward, but contamination with skin commensals (normal flora) such as coagulase negative staphylococci, diphtheroids, or environmental bacteria, is common. Careful clinical assessment is needed, because these same organisms can cause septicaemia, for example in patients with indwelling devices or who are immunosuppressed.

Culture of specimens with normal flora

a) On selective media (such as faecal specimens)

Specimens with a copious normal flora, such as faeces, are cultured on media designed to inhibit growth of these bacteria relative to target pathogens. Several different media are needed. Most laboratories routinely culture only salmonella, shigella and campylobacter from faeces, which takes 48–72 hours or more if further identification or typing is required. Additional selective media (for example, *Vibrio* species) or antigen or NAAT (see below) for specific pathogens (for example, rotavirus, norovirus, *Cryptosporidium, Giardia* etc) or toxins (for example *E. coli* shigatoxin; *C. difficile* toxins) can be added if there is a relevant history or the specimen is watery or bloody, but this greatly increases the cost and time to diagnosis. Added to the fact that there are, undoubtedly, many still unknown and probably unculturable enteric pathogens, this means that no pathogen is identified in a high proportion of cases of infective diarrhea.

Despite many technical and interpretive obstacles, this is an area in which real-time multiplex NAAT, targeting a wide range of microbial species and virulence genes, would greatly enhance diagnosis and epidemiological investigation of diarrhea.

b) On nonselective media (such as respiratory specimens)

Assessing the bacterial causes of respiratory infection is difficult, because the most common bacteria (for example *Streptococcus pneumoniae, Haemophilus influenzae* and *Moraxella catarrhalis)* can occur as normal respiratory flora, and colonization with antibiotic-resistant bacteria often precedes hospital-acquired pneumonia. Selective media can be used only for a few bacteria (for example *M. tuberculosis, Legionella* species), and viruses. The quality of the specimen is crucial—it must be from the lower respiratory tract (expectorated or induced sputum, bronchalveolar lavage) and uncontaminated, as far as possible, with saliva. The specimen is assessed by microscopy for the presence of pus cells and the predominant bacterial type and discarded if unsatisfactory. A positive result is generally only reported if a) the specimen was satisfactory and b) both Gram stain and culture show a single predominant bacterial type/species, in excess of normal respiratory flora.

ANTIGEN DETECTION

Rapid antigen tests, such as immunfluorescence (IF) or enzyme immunoassay (EIA) have been used for many years, especially for slowly growing organisms such as viruses, *Chlamydia trachomatis* and *Legionella pneumophila,* and are generally highly specific. The sensitivity of IF depends on the skill of the microscopist and the quality of the specimen, which can be assessed by noting the presence of epithelial cells. Respiratory virus IF is useful for rapid diagnosis of infections due to influenza or respiratory syncytial virus, for which infection control measures may be required. If no virus is identified, the specimen can be cultured. The urinary antigen test for *Legionella pneumophila* serogroup 1 has greatly improved the speed, sensitivity and convenience of diagnosis of legionellosis, and serum testing for hepatitis B surface antigen (HBsAg) is used routinely to identify chronic hepatitis B virus carriers. Antigen tests alone are often relatively insensitive because they do not involve amplification and many (for example for *C. trachomatis*) have now been replaced by NAAT.

NUCLEIC ACID AMPLIFICATION TESTS (NAAT)

NAAT, of which the polymerase chain reaction (PCR) is the most widely used, have revolutionised diagnostic microbiology. Briefly, they involve the use of two primers: short, single stranded nucleic acid sequences, which are complimentary to segments at each end of the target deoxyribonucleic acid (DNA) (for example, a gene that identifies a microbial species, a virulence factor such as a toxin, or an antibiotic resistance marker). When mixed with a supply of the four nucleotide DNA building blocks and DNA polymerase, these primers amplify target DNA, which is then identified, often with a labelled probe specific for the target gene. Primer design is facilitated by the availability of rapidly expanding Internet databases of microbial genome and individual gene sequences as well as free software to identify appropriate sequences unique to the target of interest.

Sensitivity depends on, among other things, the number of copies of the gene in an organism, the number of organisms in the specimen, the efficiency of DNA extraction and the presence of inhibitors in the specimen. False negative results are most likely to be due to inadequate specimen quality or amount. False positive results are usually due to contamination with "foreign" DNA, which is usually detectable by use of "no DNA" controls. The presence of dead organisms (for example after treatment), or colonisation, may complicate interpretation of a positive result.

Many NAAT are commercially available in kit form, although they are still relatively expensive. The field is progressing rapidly. Soon it will be possible to simultaneously and rapidly test a single specimen for tens or even hundreds of target gene markers. This will potentially enable the application of NAAT testing for:

- detecting numerous potential pathogens in a faecal or respiratory sample
- distinguishing nonpathogenic from pathogenic bacterial strains (for example, of *E.coli*) by detecting the prevalence of virulence genes
- detecting epidemiologically important antigenic markers such as influenza virus haemagglutinins or *Salmonella* serotypes
- detecting any of a large number of antibiotic or antiviral resistance genes.

SEROLOGICAL TESTS

Serology is widely used for the diagnosis of acute or chronic infection and identification of immune status. There is a wide range of different types of serological assay, which measure either a) the ability of the antibody to bind to a specific antigen (for example enzyme-linked immunoassay, EIA, which it the commonest type used) or b) a functional antibody effect (for example complement fixation, viral neutralisation, or inhibition of viral haemagglutination). Each type of assay and each specific antibody test must be evaluated against some "gold standard" diagnostic method. Antibody assays usually measure IgG or IgM, less commonly IgA or total antibody. Results of serological assays are usually expressed semiquantitatively as titres (the reciprocal of the highest doubling dilution of serum in which the result is positive) or qualitatively as positive/reactive, negative or equivocal. For semiquantitiative tests, a fourfold change in titre between serum specimens, tested in parallel (in the same run) is regarded as a significant change. Changes in antibody levels are more difficult to assess with qualitative tests, such as EIA. The intensity of colour change resulting from the presence of antibody is expressed as an optical density (OD) and interpreted as positive if it exceeds a predetermined "cut-off" set by the manufacturer (which may be an absolute value or relative to a negative control or background reading). Some laboratories report these values without appropriate calibration, which can give an erroneous impression of changing antibody levels.

IgG, which is formed following initial infection or immunisation, usually remains present for long periods, sometimes for life, and so is used to determine immune status or evidence of past (or current, if chronic) infection. For diagnosis of recent or acute infection, serum should be collected as soon as possible after the onset of symptoms, before IgG antibody is detectable or reaches its peak level. Another specimen collected 10-14 days later (sometimes longer) and tested in parallel with the first, showing seroconversion or a significant increase in the level of IgG, confirms a serological diagnosis of recent infection. Unfortunately, the first specimen is often not collected early enough and similar (stationary) levels of IgG are found between paired sera.

In general, IgM and IgA are detectable sooner than IgG, but remain present for a relatively short time—typically 4–6 weeks—after the onset of infection, and so are used as evidence of recent infection either a) early in the course of infection, before IgG is detectable or b) after IgG has reached a stationary level. Unfortunately, there is wide variation in the length of time for which IgM and IgA persist and occasionally they persist for years. Cross-reactions and false positive IgM results are not uncommon and IgM may reappear with reactivation of latent infections; for example, infection due to herpes viruses.

An alternative strategy for diagnosis of recent or initial infection, which is particularly important when assessing a suspected vertically transmissible infection (an infection transmitted from a mother to her baby) during pregnancy, is to measure IgG avidity. This is based on the fact that the longer the interval since infection first occurs, the more avid IgG becomes—that is, the more strongly it binds to its corresponding antigen. In practice a serum specimen is divided into two portions, one of which is treated with a concentrated urea solution, which separates antigen/antibody complexes in inverse proportion to the degree of IgG avidity. The two portions are tested in parallel (usually by EIA) and the readings showing the amount of antibody present are compared. The ratio between them (the avidity ratio) indicates whether the infection occurred a relatively long time ago (usually more than 3 months) if the ratio is high (the actual cut-off ratio varies for each antibody), or more recently if it is low. A low avidity does not always indicate recent infection (it occasionally remains low for many months), but a high avidity ratio has a high negative predictive value for recent infection.

FURTHER READING

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PART 2: USE OF DIAGNOSTIC TESTS IN PRACTICE

In general, the diagnosis of specific infectious diseases involves a combination of different tests and the level of certainty can be classified as confirmed or probable, depending on the type of infection and the clinical circumstances, as illustrated by the following examples.

MEASLES

Suspected acute measles infection is usually diagnosed by serum IgM, which is moderately reliable if a properly validated EIA is used. Ideally, IgG seroconversion would be confirmed (but takes longer). In some circumstances, a rapid diagnosis is made by IF or PCR on an upper respiratory specimen or PCR on blood or urine. Viral culture can be performed if molecular typing is required to identify the likely source of infection, such as imported. Immune status is assessed by a serum IgG assay.

TUBERCULOSIS (TB)

The Mantoux or tuberculin skin test (TST), a test of cell mediated immunity is used to diagnose TB infection. It has well-known limitations, including the fact that it may be falsely positive in people previously immunized with BCG. New tests that measure gamma interferon production by the patient's cells, in response to exposure to purified *Mycobacterium tuberculosis* antigens, *in vitro*, have the advantage that, unlike TST, they can distinguish between tuberculosis infection and previous BCG (Bacillus Calmette-Guerin) immunization. Diagnosis of TB disease in a patient with a consistent clinical picture is based on: microscopy of an appropriate specimen (usually sputum), using a stain for acid fast bacilli (which will detect mycobacteria, not specifically *M. tuberculosis*); and culture, which is relatively slow. PCR is specific and slightly less sensitive than culture, but relatively expensive and generally used only when there is urgency (for example, meningitis) or significant doubt about the diagnosis (for example, atypical presentation in a high risk patient).

PERTUSSIS

PCR on a throat swab or nasopharyngeal aspirate or swab is now the diagnostic test of choice early in the course of illness (in the first 1–2 weeks) because it is more sensitive and reliable than culture. Various serological tests are used. Seroconversion is rarely demonstrated because of the subacute course of illness. Later in the course of illness, and in older children and adults, specific IgA (above an arbitrary cut-off) is now the most commonly used (and the only commercially available) method. However, there is limited validation of its predictive value.

HUMAN IMMUNODEFICIENCY VIRUS (HIV) INFECTION

IgG antibody assays (usually by EIA) are used for screening, case detection and diagnosis of chronic HIV infection. A positive result is confirmed by repeat EIA and Western blot (a specialised serum antibody test). Acute HIV infection, if symptomatic, can be diagnosed by demonstrating seroconversion. Quantitative PCR is performed to measure viral load (which is a measure of infectivity and the need for treatment) and to monitor the efficacy of treatment, once begun.

GLOSSARY OF TERMS

CELL MEDIATED IMMUNITY (CMI)

CMI is the host response to infection, which involves cells (such as lymphocytes and macrophages) and local soluble mediators (such as cytokines), which together combine to kill or inactivate invading organisms, usually with varying levels of assistance from specific antibodies (which are responsible for humoral immunity). CMI can be detected simply by an intradermal inoculation of antigen, which, in someone with past exposure who has had a CMI response, will stimulate a delayed type hypersensitivity reaction (a localized, raised, red lump after 48 hours). Different types of pathogen stimulate a predominantly CMI response (for example intracellular bacteria, such as *M. tuberculosis*) or a predominantly humoral (antibody) immune response (for example, many viruses) but both types of response are usually involved to varying degrees.

DEOXYRIBONUCLEIC ACID/RIBONUCLEIC ACID (DNA/RNA)

The genetic material of bacteria (as well as that of other organisms, including mammals) and many viruses is amplified in nucleic acid amplification tests (NAAT). Some viruses contain RNA not DNA, which can be detected in NAAT by first converting it to DNA with the enzyme reverse transcriptase.

ENZYME IMMUNOASSAY (EIA)

EIA is the most commonly used antibody assay. A specific antigen, which is a component of a micro-organism, is combined *in vitro* (usually in the wells of a plastic microtitre plate) with test serum. A specific antibody, if present, will attach to the antigen to form an antigen/antibody complex, which is detected (after removal of any non-

specific antibody) by an antihuman antibody combined or conjugated with an enzyme. After removal of excess conjugate, remaining enzyme (indicating the presence of specific antibody) is detected by addition of a substrate, which changes color in the presence of the enzyme. EIA can be configured to detect different classes of antibody individual – IgG, IgA or IgM.

IMMUNOGLOBULIN (IG), SUCH AS IGG, IGA, IGM

Immunoglobulins or antibodies are formed, specifically, in response to foreign antigens, including microbial pathogens (and vaccines). Once programmed, the cells that produce specific antibodies can be rapidly recruited to produce more, very rapidly, if the host is again exposed to the same antigen. A specific antibody "neutralises" the organism or antigen, making it more easily engulfed by phagocytic cells (such as macrophages), which kill or inactivate them. IgM is formed first (after 5 to 10 days), after initial exposure, but usually persists for only a few weeks to months. IgG is formed a little later (after 7 to 21 days) and generally persists for life, although the level may fall in the absence of re-exposure. IgA is formed predominantly at mucosal surfaces, but is also detectable soon after exposure, usually for a short period, in serum.

IMMUNOFLUORESCENCE (IF)

The principle of IF is similar to that of EIA except that the antigen is an object that can be seen with a light microscope (for example, bacteria or a virus-infected cell monolayer) and is fixed to a microscope slide. Test serum is added and, after washing, the presence of antigen-antibody complexes is detected by adding an antihuman antibody conjugated with a fluorescent dye, the presence of which (indicating specific antibody) is detected with an ultraviolet microscope.

IMPORTED INFECTION

An infection that is acquired overseas.

MEDIA

Bacteria are cultured on artificial media, which provide nutrients for their growth. Media may be solid (nutrients are added to agar – a jelly-like substance derived from sea-weed, which is solid at room temperature) or liquid (nutrient broth). Agar plates are typically enriched by addition (to basic nutrient preparations) of horse or sheep blood, which are lysed by hemolytic bacteria and so assist identification. To facilitate the identification or isolation of a target pathogen, one of the following is added:

- specific nutrients that enhance the growth of fastidious bacteria
- selective agents that inhibit the growth of unwanted bacteria in favour of target bacteria, or
- chemical reagents that change colour in the presence of certain bacterial enzymes.

For example, antibiotics are added to more easily detect the presence of antibiotic resistant bacteria.

PREDICTIVE VALUES—NEGATIVE AND POSITIVE (NPV/PPV)

Negative and positive predictive values are quantitative estimates of the likelihood that a negative or positive test result truly is negative or positive, respectively. The values depend on the sensitivity and specificity of a particular diagnostic test and the prevalence of the condition in the population studied. (Sensitivity refers to the percentage of cases of a disease, such as an infection, that are detected by a diagnostic test, and specificity refers to the proportion of subjects not affected by a condition in whom a diagnostic test is negative.) Given a fixed proportion of tests that give false positive results, the positive predictive value (proportion of all positive results that are true positive) will be higher if the prevalence of the condition/infection in the population is high. For example, in patients with a clinical illness consistent with, say, acute toxoplasmosis, the PPV of a positive toxoplasma IgM test will be higher than in an asymptomatic subject such as a pregnant woman in whom toxoplasma IgM is tested as part of routine antenatal screening.

VERTICALLY TRANSMISSIBLE INFECTION

This is an infection that is transmitted from a mother to her fetus or infant by a mechanism that depends on the unique mother-infant relationship. Infection may occur: during pregnancy (across the placenta at any time during pregnancy or by spread from the mother's cervix into the amniotic fluid—called ascending infection—usually late in pregnancy); during delivery (for example by exposure of the infant to the mother's blood or genital secretions); or after birth (for example by breast feeding).

MALARIA

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Malaria is one of the most important vector borne diseases in the world. Approximately 1 million deaths per year are attributable to malaria with the majority of these being in sub-Saharan Africa.[1]

Malaria is transmitted to humans via the bite of an infected female *Anopheles* mosquito. There are four major species of malaria parasites that cause disease in humans. These belong to the genus *Plasmodium*—and of these, *Plasmodium falciparum* causes the most severe disease.

Inside the mosquito, the parasites multiply and develop further until they reach the stage when they can infect humans. Inside a human, the parasite undergoes a complex life cycle that ultimately destroys red blood cells which often leads to anaemia. A patient infected with *P. falciparum* and left untreated can develop severe malaria affecting vital organs such as the brain, lungs and kidneys, and can result in significant morbidity and subsequent mortality. Infected humans can transmit the parasite to mosquitos that feed on their blood.

Mosquito control efforts have eradicated malaria from many temperate zones including Australia, but it remains endemic to Central and South America, Africa, eastern Europe, Asia and parts of the south-western Pacific.

Children and pregnant women are the most vulnerable to malaria. It is estimated that 1 in 5 childhood deaths in Africa are attributable to the disease.[1] Chronic anaemia resulting from malaria can adversely affect a child's growth and development. In endemic regions of Africa the *P. falciparum* infects pregnant women resulting in severe anaemia for the mother and low birth weight and subsequent poor survival for the infant. Approximately 10, 000 maternal deaths and 8-14% of all low birth weight babies born in Africa each year are due to malaria. [1]

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

DIAGNOSIS

The signs and symptoms of acute human malaria include fever, myalgia, malaise, headache, nausea, tachycardia, tachypnoea, cough and vomiting. Given the non-specific presentation, clinical diagnosis is difficult. Consequently, the primary method of diagnosis involves the examination of thick and thin blood films for the presence of malaria parasites. Other methods of diagnosis include serology and Polymerase Chain Reaction (PCR) but both of these have their disadvantages. More recently, a number of Rapid Immunochromatography Tests have become commercially available. Tests that identify the presence of malaria histidine rich protein II are most sensitive to the diagnosis of *P. falciparum.* Tests for other species of malaria currently lack sensitivity and are thus more prone to producing false negatives. These tests may therefore be useful as a back up for inexperienced laboratories, but they cannot replace microscopy as the most acceptable diagnostic test for malaria.

VECTORS

There are more than 400 species of *Anopheles* mosquito, of which about 70 can be malaria vectors. In general, those species that are abundant and are primarily attracted to humans are the most efficient vectors.

Those mosquitos with a longer life span and that are endophilic (enter dwellings) and endophagic (feed indoors) such as the African *An. gambiae,* are generally better able to transmit the disease.

Malaria used to be endemic to regions in northern Australia and transmission has occurred in southern regions. While the infection has been eradicated, the *Anopheles* vectors are still present throughout the country. However with modern health services it is considered unlikely that malaria would become re-established in Australia, even in the previously endemic areas.

Strategies to control vectors elsewhere, range from spraying insecticides within houses to pyrethroid impregnated bednets and treated curtains. Personal protection for travellers can be afforded by bednets and topical insect repellents, as well as appropriate chemoprophylaxis.

Worldwide the eradication of malaria has been hampered by the development of both drug resistance and insecticide resistance (due to both physiological and behavioural changes) and infrastructure constraints. Research into vaccines continues but the existence and affordability of a cure remains uncertain.

REFERENCE

1. World Health Organisation. Health Topics: Malaria. At: www.who.int/topics/malaria/en/, accessed January 2006.

COMMUNICABLE DISEASES REPORT, NEW SOUTH WALES, FOR JANUARY AND FEBRUARY 2006

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

TRENDS

Tables 1 and 2 and Figure 1 show reports of communicable diseases received through to the end of February 2006 in NSW.

ROSS RIVER VIRUS INFECTION ON THE RISE

Ross River virus (RRV) is spread by mosquitoes that feed on infected animals and people. The virus then multiplies within the mosquito and is passed to other animals or people when the mosquito feeds again. Infections tend to peak in the summer and autumn months. The virus is not spread directly from one person to another.

Many people who are infected with the virus will never develop symptoms. Some people will have flu-like symptoms that include fever, chills, headache and aches and pains in their muscles and joints. Some joints can become swollen, and joint stiffness may be particularly noticeable in the morning. Sometimes a rash occurs on the body, arms or legs. The rash usually disappears after seven to 10 days. There is no specific treatment for Ross River virus infection.

In recent weeks NSW has experienced a rise in reports of RRV infection. There were 210 cases with a reported onset in February and 190 in January 2006, compared with 103 in December, 67 in November and 17 in October 2005 (see Figure 2). This compares with only 34 reported cases in January 2005. Cases have been largely reported from rural areas.

Prevention depends on avoiding being bitten by mosquitoes, especially in the summer and autumn months when infections peak. Advice includes:

- avoid being outside unprotected before dawn and after dusk
- when outside wear loose fitting, light coloured clothing that covers your arms and legs
- use an insect repellent that contains the chemical DEET (N, N-diethyl-m-toluamide) or picaridin
- fit fly screens to all windows, doors and chimneys and keep them in good repair
- use a knockdown insecticide (following the advice of the manufacturer) in bedrooms approximately half an hour before going to bed.

LEGIONNAIRES' DISEASE CLUSTER

Sydney West Public Health Unit staff reported a cluster of five cases of Legionnaires' disease with reported onset in January 2006, including four in the Blacktown area. The five cases were all men aged between 50 and 78 years. *Legionella pneumophila* serogroup 1 was cultured in sputum from one case. The others had positive urinary antigen tests. All cases were interviewed about exposures during their incubation periods (2–10 days before onset).

The investigation found that three of the five men had visited the Blacktown shopping area during their incubation periods. Common exposures in the Prospect and Seven Hills areas were also reported by two men. South Western Public Health Unit staff, assisted by Blacktown Council, reviewed the maintenance of cooling towers in these areas to ensure compliance with the *NSW Public Health Act 1991* (for details of these requirements, see: www.health.nsw.gov.au/public-health/ehb/general/microbial/microbial.html). In addition, the public health unit staff contacted Emergency Departments and general practitioners in the area to advise them to report other possible cases. There is no evidence of ongoing risk.

A CASE OF CUTANEOUS ANTHRAX

In January, Greater Southern Public Health Unit staff reported a case of cutaneous anthrax in a man from the state's west. The man lived on a property in the 'anthrax belt' that runs down the west of NSW. Anthrax has survived for many decades as spores in the soil in this part of the country, and animals are occasionally diagnosed with anthrax, so farmers routinely vaccinate their animals to prevent disease. The man reported minor trauma to his arm around December 2005 while working on farm machinery. It was at this site that pimples developed and then eschars, which are black ulcers that are typical of anthrax. In late January 2006 anthrax was cultured from the patient's eschars. The patient was treated with penicillin and recovered. For more information see www.health.nsw.gov.au/infect/diseases.html and scroll down to 'Anthrax'.

ENTERIC DISEASE

Some types of ***Escherichia coli*** bacteria can produce toxins (Shiga toxins). These bacteria are called **Shiga toxigenic *E. coli* (STEC)** or **verotoxigenic producing *E. coli* (VTEC)**. Various strains of VTEC (for example *E. coli* O111 and *E. coli* O157) have been responsible for outbreaks of serious human disease, including bloody diarrhoea that can sometimes be complicated by haemolytic uraemic syndrome (HUS), a severe condition characterised by kidney failure, bleeding and anaemia. VTEC infection may be acquired by eating contaminated food; commonly undercooked burgers, unwashed salad vegetables, and unpasteurised milk and milk products.[1] Usually only one or two cases of VTEC or HUS are reported each year in NSW.

From October 2005 to early February 2006, NSW Health received 12 notifications of cases of VTEC infection. Staff at one laboratory reported changing the laboratory's stool specimen screening method in 2005, which may account for some of the increase in notifications. The ages of the patients ranged from three to 85 years, and five were female.

Six reports of HUS were received in the same period in NSW. Consequently public health units were asked to undertake active surveillance for HUS cases by contacting renal physicians in their Areas to identify any unreported cases of HUS. As a result three additional cases of HUS were identified. Of all nine HUS cases, three were female, and the patients' ages ranged from one to 64 years. Three of the 9 HUS cases had associated VTEC infections, making a total of 15 VTEC cases for the period October 2005 to early February 2006.

All patients who had had HUS and VTEC were interviewed to identify any common exposures. None were identified.

Clinicians should report cases of HUS to their local public health unit on diagnosis, and test these cases for evidence of VTEC infection. To avoid infection it is important to:

- cook minced meat thoroughly, at least until any juices run clear
- wash salad vegetables thoroughly before eating
- avoid unpasteurised products.

VTEC may also be spread though contact with infected animals, especially cattle.

Reference

1. Heymann DL (ed). *Control of Communicable Diseases Manual 18th Ed.* 2004: American Public Health Association, Washington.

CRYPTOSPORIDIOSIS

Reports of cryptosporidiosis remained elevated throughout summer. The high numbers were probably the result of an ongoing outbreak, and changes to procedures in some laboratories that have resulted in an increase in the routine testing of stools.

The outbreak seemed to begin in rural areas in spring 2005, when some cases were linked to contact with infected farm animals, or contact with other cases. Over summer, more than 50 per cent of cases reported swimming in pools during their incubation periods (in the previous two to 12 days), suggesting that swimming remains an important risk factor.

Public health units routinely investigate cases of cryptosporidiosis reported by laboratories, and where a cluster of cases linked to a pool is identified, public health unit staff contact the pool operators to ensure that the pool is maintained according to NSW Health guidelines (see: www.health.nsw.gov.au/public-health/ehb/water/recreational.html). People with diarrhoea should not enter swimming pools for at least a week after complete recovery.

LISTERIOSIS

Listeriosis is a rare but serious illness caused by eating food contaminated with the bacteria *Listeria monocytogenes.* The bacteria are common in soil and the environment. Most people do not get sick after exposure to *Listeria*, but some people, including pregnant women, unborn and newborn babies, older people and people with compromised immune systems, are at increased risk of illness.

In February, NSW Health was notified of a 34-year-old pregnant woman with listeriosis. The woman presented to hospital with a fever and back pain. She reported no knowledge of listeriosis and had been consuming foods considered to be of high risk throughout her pregnancy. Both mother and baby recovered.

The risk of exposure to *Listeria* can be reduced by a few food safety principles: wash your hands before preparing food, keep raw and cooked foods separate, store perishable foods in the fridge and consume as soon as possible, cook meats thoroughly and wash fruit and vegetables before eating.

Foods that are considered to be high risk and that should be avoided if you are at risk of listeriosis are:

- pre-packed salads and fruit
- pre-cooked diced chicken (as used on sandwiches)
- delicatessen sliced meats
- raw seafood
- soft cheeses (such as brie and camembert)
- sprouted seeds
- mushrooms.

Listeria survives at low temperatures so it is advisable to avoid foods that have been stored in the fridge for long periods of time.

For more information, see www.health.nsw.gov.au/public-health/cdscu/facts/pdf/listeriosisfs.pdf.

FIGURE 1

REPORTS OF SELECTED COMMUNICABLE DISEASES, NSW, JAN 2000 TO FEB 2006, BY MONTH OF ONSET

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

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

BFV = Barmah Forest virus infections, RRV = Ross River virus infections

Lab conf = laboratory confirmed

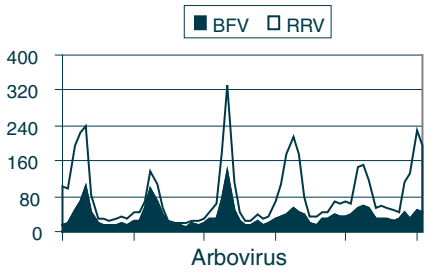
Men Gp C and Gp B = meningococcal disease due to serogroup C and serogroup B infection, other/unk = other or unknown serogroups.

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

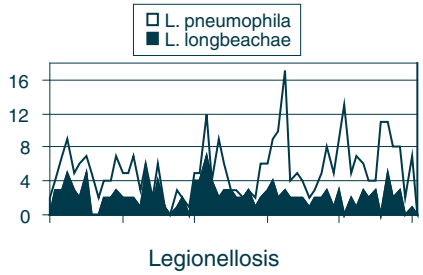
NB: Outbreaks are more likely to be reported by nursing homes and hospitals than by other institutions

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

Dec 05–Feb 06	
Male	50%
<5	1%
5–24	10%
25–64	81%
65+	8%
Rural	91%



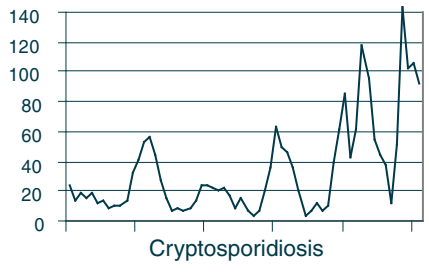
Arbovirus



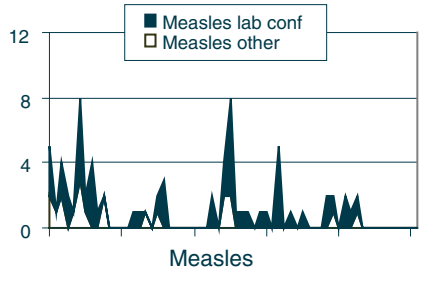
Legionellosis

Dec 05–Feb 06	
Male	80%
<5	0%
5–24	0%
25–64	60%
65+	40%
Rural	0%

Dec 05–Feb 06	
Male	46%
<5	42%
5–24	34%
25–64	23%
65+	1%
Rural	59%



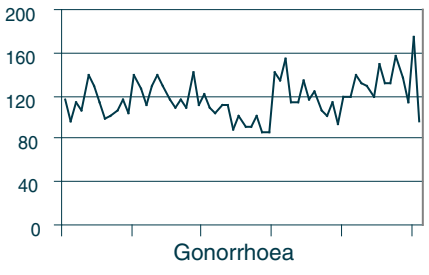
Cryptosporidiosis



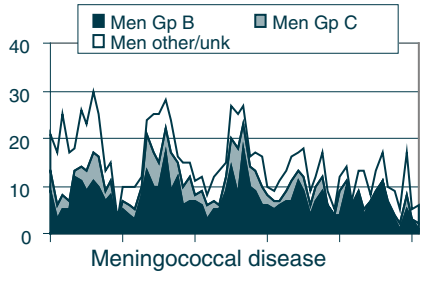
Measles

Dec 05–Feb 06	
Male	0%
<5	0%
5–24	0%
25–64	0%
65+	0%
Rural	0%

Dec 05–Feb 06	
Male	87%
<5	1%
5–24	27%
25–64	71%
65+	1%
Rural	13%



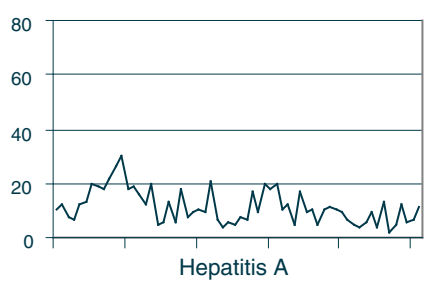
Gonorrhoea



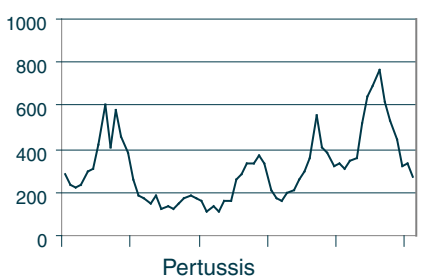
Meningococcal disease

Dec 05–Feb 06	
Male	79%
<5	39%
5–24	39%
25–64	22%
65+	0%
Rural	32%

Dec 05–Feb 06	
Male	54%
<5	4%
5–24	63%
25–64	25%
65+	8%
Rural	21%



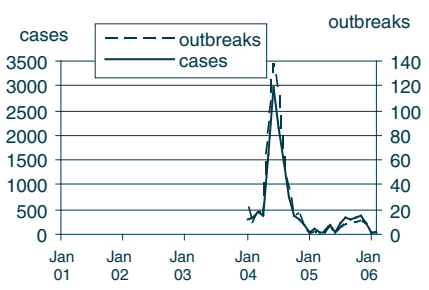
Hepatitis A



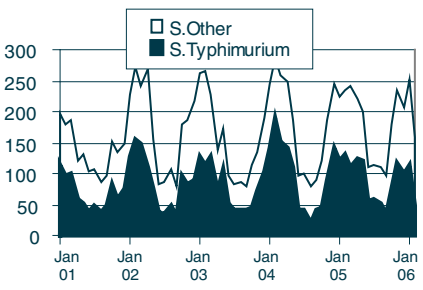
Pertussis

Dec 05–Feb 06	
Male	40%
<5	2%
5–24	11%
25–64	71%
65+	16%
Rural	33%

Dec 05–Feb 06	
All outbreaks	12
Nursing homes	4
Hospitals	7
Child care	1
Schools	0
Other	0



Gastroenteritis outbreaks in institutions



Salmonella infections

Dec 05–Feb 06	
Male	49%
<5	26%
5–24	28%
25–64	35%
65+	11%
Rural	43%

TABLE 1 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN JANUARY 2006 BY AREA HEALTH SERVICES

	Area Health Service (2006)																			Total	
	Greater Southern		Greater Western			Hunter / New England		North Coast		Northern Syd / Central Coast		South Eastern Syd / Illawarra		Sydney South West		Sydney West					
Condition	**GMA**	**SA**	**FWA**	**MAC**	**MWA**	**HUN**	**NEA**	**MNC**	**NRA**	**CCA**	**NSA**	**ILL**	**SES**	**CSA**	**SWS**	**WEN**	**WSA**	**JHS**	**for Jan+**	**To date+**
Blood-borne and sexually transmitted§																				
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	40	26	-	9	20	107	27	42	46	43	103	52	210	141	82	37	85	4	1088	1088
Gonorrhoea*	2	-	-	1	2	5	1	2	2	-	15	3	63	46	8	1	12	-	163	163
Hepatitis B–acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hepatitis B–other*	2	3	-	3	1	5	4	2	2	7	35	4	37	57	63	5	34	2	267	267
Hepatitis C–acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hepatitis C–other*	12	20	1	7	18	38	9	20	25	35	27	30	62	61	101	24	54	29	585	585
Hepatitis D–unspecified*	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	1	1
Syphilis	-	-	-	-	2	-	1	1	-	1	10	2	9	23	11	3	4	-	67	67
Vector-borne																				
Barmah Forest virus*	8	1	-	-	1	9	1	9	10	4	1	4	1	2	-	-	-	-	51	51
Ross River virus*	46	3	5	21	28	11	20	11	3	4	2	11	5	1	1	2	2	-	177	177
Arboviral infection (other)*	1	-	-	-	-	1	-	1	1	-	1	-	1	-	1	-	1	-	8	8
Malaria*	-	-	-	-	-	-	-	1	2	-	1	1	1	1	2	-	1	-	12	12
Zoonoses																				
Anthrax*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Leptospirosis*	1	-	-	-	-	1	4	-	1	-	-	-	-	-	-	-	-	-	7	7
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis*	2	-	-	-	-	1	-	1	-	-	-	-	-	-	-	1	-	-	6	6
Q fever*	-	2	-	4	1	2	4	1	-	-	-	-	1	-	1	-	-	-	16	16
Respiratory and other																				
Blood lead level*	1	1	-	-	-	4	-	1	-	-	-	1	1	1	10	2	-	-	22	22
Influenza*	-	-	-	-	-	2	-	-	1	-	6	2	63	2	2	1	23	-	102***	102
Invasive pneumococcal infection*	1	1	-	-	3	5	-	4	-	5	6	1	2	2	3	-	4	-	38	38
Legionella longbeachae infection*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Legionella pneumophila infection*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	2	2
Legionnaires' disease (other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	1
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	1	1
Meningococcal infection (invasive)*	-	-	-	1	-	-	1	1	1	-	-	-	1	1	1	-	1	-	8	8
Tuberculosis	1	1	-	-	-	1	-	-	-	1	6	1	4	6	-	1	10	-	32	32
Vaccine-preventable																				
Adverse event after immunisation (AEFI)**	1	-	-	-	2	-	1	-	-	-	1	-	-	-	-	1	-	-	6	6
H. Influenzae b infection (invasive)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Measles	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mumps*	-	-	-	1	-	-	-	-	-	-	-	-	3	3	-	-	2	-	9	9
Pertussis	29	11	-	4	8	49	2	15	10	12	51	23	79	38	36	12	44	-	423	423
Rubella*	-	-	-	-	-	-	1	-	-	-	-	-	-	-	1	-	-	-	2	2
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Enteric																				
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cryptosporidiosis*	5	3	-	4	8	6	6	11	10	1	9	8	1	8	11	8	6	-	105	105
Giardiasis*	3	2	-	1	2	12	5	8	2	5	26	9	34	9	10	4	10	-	142	142
Haemolytic uraemic syndrome	1	-	-	-	-	-	1	-	1	-	-	-	-	-	-	-	1	-	4	4
Hepatitis A*	-	-	-	-	-	1	-	-	-	1	2	-	1	1	1	-	1	-	8	8
Hepatitis E*	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
Listeriosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Salmonellosis*	3	10	1	4	7	9	10	20	32	10	30	7	33	19	32	17	27	-	273	273
Shigellosis*	-	-	-	-	-	-	-	-	1	-	1	-	3	1	-	-	-	-	6	6
Typhoid*	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	-	-	-	2	2
Verotoxin producing *E. coli**	1	-	-	-	-	3	-	-	-	-	-	-	-	-	-	-	1	-	5	5
Miscellaneous																				
Creutzfeldt-Jakob disease	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Meningococcal conjunctivitis	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

* lab-confirmed cases only + includes cases with unknown postcode § HIV and AIDS data are reported separately, quarterly in the *NSW Public Health Bulletin.*

** AEFIs notified by the school vaccination teams during the National Meningococcal C Program are not included in these figures. These notifications are reviewed regularly by a panel of experts and the results will be published quarterly in the *NSW Public Health Bulletin.* N.B: From 1st Jan 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester & Greater Taree LGAs; Sydney West also comprises Greater Lithgow LGA

GMA = Greater Murray Area
SA = Southern Area
FWA = Far West Area
MAC = Macquarie Area
MWA = Mid Western Area
HUN = Hunter Area
NEA = New England Area
MNC = North Coast Area
NRA = Northern Rivers Area
CCA = Central Coast Area
NSA = Northern Sydney Area
ILL = Illawarra Area
SES = South Eastern Sydney Area
CSA = Central Sydney Area
SWS = South Western Sydney Area
WEN = Wentworth Area
WSA = Western Sydney Area
JHS = Justice Health Service

TABLE 2 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN FEBRUARY 2006 BY AREA HEALTH SERVICES

	Area Health Service (2006)																			Total	
	Greater Southern		Greater Western			Hunter / New England		North Coast		Northern Syd / Central Coast		South Eastern Syd / Illawarra		Sydney South West		Sydney West					
Condition	**GMA**	**SA**	**FWA**	**MAC**	**MWA**	**HUN**	**NEA**	**MNC**	**NRA**	**CCA**	**NSA**	**ILL**	**SES**	**CSA**	**SWS**	**WEN**	**WSA**	**JHS**		**for Feb+**	**To date+**
Blood-borne and sexually transmitted§																					
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Chlamydia (genital)*	43	24	11	24	32	120	32	31	37	43	93	45	162	97	61	33	85	3		979	2081
Gonorrhoea*	-	-	-	4	-	3	-	1	4	2	14	2	65	34	12	4	16	-		167	341
Hepatitis B–acute viral*	1	-	-	-	-	1	-	-	-	-	1	1	-	-	-	-	-	-		4	9
Hepatitis B–other*	3	1	4	1	-	8	1	4	2	3	32	8	22	30	43	9	46	1		220	503
Hepatitis C–acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2		2	3
Hepatitis C–other*	12	6	7	7	30	32	16	20	29	25	29	31	60	52	83	32	44	9		527	1117
Hepatitis D–unspecified*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	1
Syphilis	-	1	6	1	3	3	1	-	2	1	6	3	29	32	12	4	5	-		101	162
Vector-borne																					
Barmah Forest virus*	6	-	-	1	-	9	-	12	10	6	1	5	3	-	-	1	-	-		54	106
Ross River virus*	48	10	12	17	18	15	16	17	20	3	5	19	2	-	5	2	1	-		210	388
Arboviral infection (other)*	-	-	-	-	-	-	-	-	-	-	3	-	1	-	-	1	1	-		6	15
Malaria*	-	-	-	-	-	1	-	-	1	-	-	1	1	-	-	-	5	-		10	22
Zoonoses																					
Anthrax*	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		1	1
Brucellosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Leptospirosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	6
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Psittacosis*	-	-	1	-	-	5	-	-	1	-	-	-	-	-	-	-	1	-		8	16
Q fever*	-	1	-	2	2	-	4	2	2	-	-	1	-	-	1	-	-	-		15	35
Respiratory and other																					
Blood lead level*	-	-	-	-	-	3	-	-	-	-	2	3	1	3	10	-	-	-		22	44
Influenza*	-	2	-	-	-	-	-	-	1	-	3	1	5	-	-	-	-	-		12	114
Invasive pneumococcal infection*	1	1	-	1	1	2	-	2	-	1	-	3	6	2	4	-	1	-		26	65
Legionella longbeachae infection*	-	1	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-		2	3
Legionella pneumophila infection*	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	3	-		4	6
Legionnaires' disease (other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	1
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	1
Meningococcal infection (invasive)*	-	-	-	-	-	-	-	-	-	-	1	-	2	-	3	-	-	-		6	14
Tuberculosis	1	1	-	-	-	1	-	-	-	2	4	-	1	4	4	-	4	-		32	65
Vaccine-preventable																					
Adverse event after immunisation (AEFI)**	-	-	2	-	1	-	-	-	1	-	-	-	-	-	-	-	1	-		5	12
H. Influenzae b infection (invasive)*	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-		1	1
Measles	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Mumps*	1	-	-	-	-	-	-	-	1	-	1	-	-	2	1	-	-	-		6	15
Pertussis	12	5	5	8	3	16	2	11	4	16	52	10	63	31	41	16	56	-		352	775
Rubella*	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-		1	3
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Enteric																					
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-
Cryptosporidiosis*	7	3	4	2	2	12	4	9	17	1	18	5	15	10	8	2	9	-		128	235
Giardiasis*	9	5	-	6	5	11	4	4	3	8	33	7	21	12	13	10	14	-		165	309
Haemolytic uraemic syndrome	-	-	-	-	1	-	-	-	-	-	1	-	3	-	-	-	-	-		5	9
Hepatitis A*	-	-	1	-	-	1	-	-	-	-	-	-	5	2	5	-	-	-		14	22
Hepatitis E*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-		1	2
Listeriosis*	-	-	-	-	1	-	-	-	-	-	-	-	1	-	-	1	-	-		3	3
Salmonellosis*	4	2	3	11	7	12	18	11	24	6	25	16	34	16	23	7	17	-		236	510
Shigellosis*	1	-	-	1	1	-	-	1	-	-	2	-	1	3	-	1	-	-		11	17
Typhoid*	-	-	-	-	-	-	-	-	1	-	1	-	-	-	-	-	1	-		3	4
Verotoxin producing *E. coli**	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		1	6
Miscellaneous																					
Creutzfeldt-Jakob disease	-	1	-	-	-	-	-	-	-	-	1	-	-	-	1	-	-	-		3	3
Meningococcal conjunctivitis	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-

* lab-confirmed cases only + includes cases with unknown postcode § HIV and AIDS data are reported separately, quarterly in the *NSW Public Health Bulletin.*

** AEFIs notified by the school vaccination teams during the National Meningococcal C Program are not included in these figures. These notifications are reviewed regularly by a panel of experts and the results will be published quarterly in the *NSW Public Health Bulletin.* N.B: From 1st Jan 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester & Greater Taree LGAs; Sydney West also comprises Greater Lithgow LGA

GMA = Greater Murray Area
SA = Southern Area
FWA = Far West Area
MAC = Macquarie Area
MWA = Mid Western Area
HUN = Hunter Area
NEA = New England Area
MNC = North Coast Area
NRA = Northern Rivers Area
CCA = Central Coast Area
NSA = Northern Sydney Area
ILL = Illawarra Area
SES = South Eastern Sydney Area
CSA = Central Sydney Area
SWS = South Western Sydney Area
WEN = Wentworth Area
WSA = Western Sydney Area
JHS = Justice Health Service