NEW SOUTH WALES
Public Health Bulletin

Volume 5
Number 2
February, 1994

NSW HEALTH DEPARTMENT

ISSN 1034 7674

COUNTING THE COSTS: A REVIEW OF TWO TUBERCULOSIS MASS CONTACT SCREENING INVESTIGATIONS

Cait Lonie, Michael Levy, Michael Frommer

INTRODUCTION

Methods to control the spread of tuberculosis include effective treatment of active cases and screening for infection[1]. Mass screening of the community with annual chest x-rays was a major component of the Australian Tuberculosis Campaign between 1948 and 1976[2]. This campaign was ceased in 1979 following marked decreases in the prevalence of tuberculosis.

As the positive predictive value of the screening tests is greater in groups with a higher prevalence of disease[3], screening for tuberculosis in Australia is now targeted at high-risk groups to increase the benefits. The contacts of active cases are one such group, especially where there is an elevated risk of disease transmission, e.g. the closest contacts of active cases. However, sometimes screening of contacts with a lower risk may be undertaken, e.g. screening contacts at worksites. This article reviews the costs and benefits of contact screening which was carried out at two worksites.

METHODS OF THE CONTACT INVESTIGATIONS

The index cases will be called Case A and Case B, and their worksites Site A and Site B respectively.

Case A was highly infectious with evidence of transmission to family contacts, who required chemoprophylaxis. By contrast, Case B was only mildly to moderately infectious, and there was no evidence of transmission to close contacts. In both investigations the index case had worked on a number of shifts and in different places at the site.

While it was impossible to determine the degree of workers' contact with the index cases, all workers who had been potentially exposed to the index cases (by virtue of having been employed at either site) were screened. This included workers who had subsequently left (retired, resigned or taken a transfer) between the time of exposure and the surveys.

Workers from Site A and Site B were offered screening at the workplace, and those who had left either site were contacted by letter requesting that they attend their local chest clinic for screening. The screening at Site A took place within weeks of the diagnosis of the index case, while at Site B there was a 10-month delay. To protect the index case's identity, all employees at both worksites were included in the screening, and no attempt was made to assess the risk of exposure among the potential contacts in relation to their work location.

Of the 273 workers employed at Site A at the time of the survey, 243 were screened at the workplace with a mobile chest x-ray and

Continued on page 14 ▶

Contents

Correspondence

Please address all correspondence and potential contributions to:

The Editor,
NSW Public Health Bulletin,
Public Health Division,
NSW Health Department
Locked Bag No 961,
North Sydney NSW 2059
Telephone: (02) 391 9218
Facsimile: (02) 391 9232

TABLE 1

COMPARISON OF SCREENING AT TWO WORK SITES

	Site A	Site B
Date of diagnosis of index case	September 1991	April 1991
Date of screen at work site	September 1991	February 1992
Degree of index case infectivity+	High	Low-moderate
Transmission to close contacts	Yes	No
Total number of people screened from workplace	273	441
Number of retirees/transferees for screening	Included above	105
Mean age (years)	40.7	37.3
Number (%) born overseas	167 (62%)	283 (64%)
Number (%) born in South-East Asia	80 (30%)	176 (40%)
Screening tests used	Mantoux test Chest x-ray	Chest x-ray
Total number with abnormalities	30 (11%)	23 (5%)
Number of abnormalities on chest x-ray alone	22 (8%)	23 (5%)
Tuberculous abnormalities detected		
Transmitted cases	0	0
Active cases	0	1
Inactive cases	3	1
Conditions detected requiring other referral	4	1
Definite abnormalities on x-ray, but did not attend for follow-up	*	4/23 (17%)
Technical fault, but did not attend for follow-up	*	9/23 (39%)
Number requiring ongoing review in chest clinics	6	6
Costs	$6,697	$4,783**

+ Degree of infectivity is based on evidence of transmission to others, length of illness and direct smear positivity.

* Not available.

** Does not include the cost of following up transferees and retirees.

Tuberculosis screening

► *Continued from page 13*

Mantoux test. The remaining 30 workers were absent at the screening times, and they attended the local Chest Clinic. A total of 441 Site B workers was screened at the workplace with a mobile chest x-ray (but not a Mantoux test), and 105 workers who had left site B were invited to attend local chest clinics.

Any workers with abnormalities consistent with tuberculosis infection requiring further investigation were referred to a local Chest Clinic. The indications for referral and further investigation depended on the clinical interpretation of the test results and clinical policies in each Chest Clinic. People with abnormalities not consistent with tuberculous infection were referred to their general practitioner for further investigation and treatment.

Data from each investigation were collected from Chest Clinics and entered onto an EPI_INFO database for analysis. Chest Clinic staff, including nurses, physicians and technical assistants, were interviewed.

Costs were approximated from Medicare schedules[4], from NSW Health Department figures[5] and calculated from approximate costs supplied by Chest Clinic staff.

RESULTS FROM THE INVESTIGATIONS

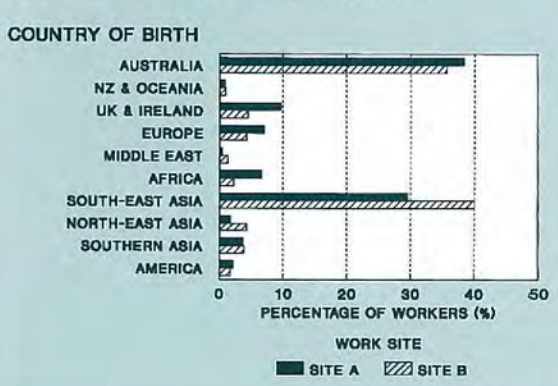
The conduct and results of the investigations are summarised in Table 1. The workers were predominantly male, with a high proportion of workers born overseas, particularly South-East Asia (see Figure 1).

Site A

Most workers at Site A were screened with a chest x-ray

FIGURE 1

COUNTRY OF BIRTH IN EMPLOYEES SCREENED FOR TUBERCULOSIS, SITE A COMPARED TO SITE B



and a Mantoux test. Two hundred and twenty-nine contacts (84 per cent) had Mantoux tests, the results of which were read for 226. One hundred and one (44 per cent) of these were negative (<5mm). Of the 115 contacts with positive Mantoux results 106 (92 per cent) had a history of BCG vaccination, two had not had a BCG, and in seven the BCG status was not known. Of these last two groups, eight had normal x-rays so they required ongoing review and one had an abnormal chest x-ray which required a clinic visit and ongoing review at Chest Clinics.

Of the chest x-rays, abnormalities were detected in 22 (8 per cent) of the contacts. Three of these were considered to be normal on second reading. The other 19 required a larger film. These contacts were discharged as normal (six) or with inactive TB (three), or referred to their general practitioner

with incidental findings (four), and six required ongoing review.

Site B

Workers at Site B were screened with a chest x-ray only. Twenty-three (5 per cent) of the x-rays of staff at Site B were read as abnormal and were referred to the Chest Clinic closest to their place of residence. These contacts either failed to attend for follow-up (four), were discharged as normal (12), required ongoing review (four), diagnosed with an incidental finding (one) or diagnosed with active TB (one). This last contact was a 27-year-old Vietnamese Chinese woman who had a positive Mantoux test and a chest x-ray consistent with tuberculosis. Although she denied other contacts with tuberculosis, her physician believed the disease was probably not a result of transmission from the index case because of the degree of contact with the index case and her ethnic background.

There were 23 contacts at Site B who had technical faults with their chest x-rays and were asked to attend their local chest clinic for a repeat chest x-ray. These contacts either failed to attend for follow-up (nine), were discharged as normal (seven), had inactive TB (one), required ongoing review (one) or were lost to follow-up (five).

COSTS OF THE SCREENING INVESTIGATIONS

The direct costs of the investigations include the cost of the mobile van, staff time at Chest Clinics and at the site, and the tests required. The indirect costs include the loss of the workers' time, and the opportunity costs for the resources used: Chest Clinic Nurses, radiographers, consultants and the mobile van. Intangible costs would include the anxiety caused by false positive abnormalities found on the screen, or by the identification of asymptomatic underlying or unrelated disease.

An estimate of the direct costs is presented in Table 2. The cost at Site A was approximately $6,700 ($24.53 for each person screened), and for Site B $4,800 ($8.76 per person). These figures are underestimates because indirect or intangible costs are not included.

Costs at Site A were higher as a nurse was required for three days to give and read the Mantoux tests. Costs at Site B may be lower than expected because of the large number of people who did not attend for further investigation after abnormalities or faults with their x-rays.

DISCUSSION

This review highlights many of the problems encountered during mass screening for tuberculosis infection. Both the Mantoux test and the chest x-ray lose sensitivity and specificity for tuberculosis infection when the prevalence of the infection in the population being screened decreases. This is reflected in an increase in the number of people with false positives that require further investigation and ongoing review for periods up to one year. This may amount to a considerable intangible cost for those affected.

A large percentage of contacts at each site were lost to follow-up. Many of these were contacts who were not in close touch with the index case, and because of the nature of their work could not return to have their Mantoux test read. Once the decision to screen contacts is made, it is essential that they are followed up, otherwise the reasons for screening these contacts must be questioned.

In this review, the contacts born in Asia were three times more likely to have a positive Mantoux test than workers born in Australia ($X2=47.7$, $p<0.001$), and three of the contacts (75 per cent) with inactive tuberculosis came from Asian countries. At worksites where there is a high proportion of workers born overseas, this will result in a high rate of false positives (i.e. Mantoux positives due to previous exposure or BCG vaccination). Unless this can be effectively managed in the protocols used to screen overseas-born contacts, the costs for screening are increased.

TABLE 2

COST ESTIMATES FOR THE SCREENING OF THE TWO WORK SITES

	Site A	Site B
On-site costs	$1,443	$1,802
Personnel costs	$2,070	$ 947
First follow-up at clinic	$2,798	$1,712
Second follow-up at clinic	$ 386	$ 322
TOTAL	$6,697	$4,783

Although screening for tuberculosis has traditionally been used for the control of tuberculosis, it is most effective when it is targeted at high-risk populations. Screening of contacts with less exposure may be ineffective and very costly. The NSW Health Department is addressing these issues by encouraging a risk assessment approach for contacts, so the highest risk contacts are screened first, and only if there is evidence of transmission in this group would screening be conducted in any lower risk contacts.

At the time of this review there were no standardised Health Department policies on mass contact screening. These principles are outlined in a strategy for the control of tuberculosis in NSW[6]. This is an important element in addressing the continuing problem of tuberculosis infection in our community.

---

1. American Thoracic Society, Medical Section of the American Lung Association. Control of Tuberculosis. *Am Rev Resp Dis* 1983; 128:336-342.
2. Porter RM, Boag TC. The Australian Tuberculosis Campaign 1948-1976. Melbourne: Menzies Foundation, 1991.
3. Henekens CH, Buring JE. Epidemiology in Medicine. Boston/Toronto: Little, Brown and Company, 1987.
4. Medicare Benefits Schedule. Department of Health, Housing and Community Services, Canberra, 1991.
5. Lagaida R, Hindle D. A Casemix Classification for Hospital-Based Ambulatory Services. NSW Health Department, July 1992.
6. Westley-Wise T, Levy M, Lonie C, McAnulty J, Winks M, Stewart G. Controlling Tuberculosis in NSW. NSW Health Department, March 1993.

DRINKING WATER QUALITY – A HEALTH PERSPECTIVE

John W Archer
Author, On The Water Front (1991)

One major problem confronting scientists interested in possible links between health and drinking water has always been the difficulty of proof. It is not easy to demonstrate whether a connection exists between common disorders like gastroenteritis and water supplies. A Victorian Department of Water Resources report points out that many Australian epidemics of waterborne diseases such as hepatitis, gastroenteritis and amoebic dysentery have not been documented or recorded because they "were not scientifically proven to be linked to drinking water".

The report continues: "... to prove an association of drinking or bathing or washing food in contaminated water with sickness requires an expensive and comprehensive investigation which can therefore rarely be carried out"[1].

This problem is not new. Although many believed there might be some connection, it was not until late in the 19th century that microscope studies began to reveal the existence of deadly bacteria in some water supplies. However, authorities did not see any cause for alarm. London water engineer Ralph Dodd wrote in 1805: "Thames water being kept in wooden vessels, after a few months, often becomes putrid . . . and produces a disagreeable smell. But even when drunk in this state, it never produces sickness; therefore it is evident no harm or ill occurs to persons whose resolution, notwithstanding its offensive smell, induces them to drink it."[2]

Thirty years later thousands of Londoners died in the first of several cholera epidemics which recurred until the 1860s when people began to connect the disease with drinking water. Even so, given the existing levels of pollution, it did not appear much could be done to make it safe.

In 1904 Sir Alexander Houston discovered that the addition of chlorine to water could destroy the typhoid bacillus. Excited scientists soon found that disinfecting water with chlorine also eliminated cholera, dysentery and several other waterborne diseases which often resulted in epidemics. Most water treatment systems since then have been designed to disinfect water with chlorine to achieve an acceptable level of bacteriological safety.

To check whether the disinfection process was working properly water scientists and microbiologists evolved a series of tests using organisms known as coliforms. *Coliforms* and *Escherichia coli* (E. coli) are part of a group of largely benign bacteria whose presence in water may be an indication that the water may be contaminated. They are the normal inhabitants of the gut of birds, humans and other warm-blooded animals, and are not capable of surviving in water for long periods. Coliforms are used as indicators because if they are detected in water, their presence indicates that excrement has polluted the water, and that other more serious pathogens may also be present.

E. coli is a member of the coliform group which is predominant in fresh faeces, so its presence in water indicates recent faecal contamination. Both coliforms and E. coli are counted by water laboratories as part of regular tests to ascertain the microbiological quality of water supplies. It is on the basis of these examinations that our water is pronounced safe. However bacteria may not be the only threat.

A major Canadian study recently revealed an association between gastrointestinal symptoms and tap water consumption, and casts serious doubts about the methods used to evaluate drinking water quality. The study, commissioned by the University of Quebec, was conducted on 600 families. Half those surveyed drank water which had been treated by the City of Montreal's modern treatment plant while the balance consumed the same water after further home treatment with a reverse osmosis purifier.

The drinking water met all the standards required to assure its bacteriological safety as well as all the other normally accepted water quality guidelines. It was pronounced to be "perfectly safe and of high quality". The same words are frequently used to describe Australian tap water supplies. It is important to note that this water supply was prepared from sewage-contaminated surface waters.

The Canadian study demonstrates that, in spite of meeting these standards, the water was "found to be associated with a significant level of gastrointestinal illnesses". The group who purified their tap water at home had 30 per cent fewer gastrointestinal disorders during the 18-month study.

The authors warn that water treatment systems based on disinfection for bacteria are ineffective against many parasites and viruses and consequently the use of coliforms to indicate water quality may result in "a false sense of security"[3].

The presence of *Cryptosporidium parvum* is causing particular concern to water authorities in Australia and the USA. For example, it is now estimated that 380,000 people were affected by the epidemic of cryptosporidiosis which occurred in Milwaukee, Wisconsin, in April 1993. City officials believe the parasite, which is not killed by chlorination, may have contaminated the municipal water supply after passing through the existing treatment plant.

In July 1993 in Sydney there was considerable public interest when the media discovered an environmental impact study which revealed that *Cryptosporidium* had been found in all the Sydney Water Board's storages and at several locations in the pipeline system "at levels similar to those found in the UK and the USA".

The water was pronounced safe to drink because the number of parasites found was "extremely low". However the World Health Organisation and other authorities believe there is not yet enough information about what constitutes an infective dose. As one British scientist points out: "This is a crucial issue for the water industry. Currently nationally recommended methods do not provide an indication of the infective potential of oocysts. Presently the prudent assumption is made that any oocyst is capable of causing infection."[4]

A month later the problem re-emerged when 10 districts around Melbourne were warned to boil their water because of the levels of *Cryptosporidium* in the Tarago reservoir.

With new Australian technology recently developed for the detection of viruses and parasites in water, it appears there's a lot more to be learned about the connection between water and community health.

One problem with suggesting routine analysis for *Cryptosporidium* and giardia is that they are expensive and very time-consuming. The National Health and Medical Research Council does not recommend routine analysis for *Cryptosporidium* or giardia.

---

1. Dept Water Resources, Vic. *Microbiological Drinking Water Quality*, 1985 p5.
2. Weightman, Gavin. *London River* 1990 p129.
3. Payment, P, Franco, E, Siemiatycki, J. Absence of relationship between health effects due to tap water consumption and drinking water quality parameters. *Wat Sci Tech* 1993. Vol 27 No 3-4, pp 137-143.
4. Benton, C, Ives, KJ, Miller, DG, West, PA. *Cryptosporidum in Water*. 1991 p6.

EDITORIAL COMMENTS

The Health Department, with other health authorities and water supply agencies, has recognised the potential importance of protozoan parasites in drinking water supplies and their possible role in previously unexplained outbreaks of gastroenteritis.

Mr Archer raises the issue whether *Cryptosporidium* should be monitored routinely. The key issue, however, is the role of indicators and the way this information is used in managing water supplies. Knowledge of the environmental conditions in the drinking water catchments is critical. The Department strongly supports the principle of multiple barriers based first on raw water of the highest possible quality. Indicators then are used for quality control and, where necessary, additional indicators can be used to test for probable contamination based on knowledge of the catchments. It is not necessary to test for all the individual contaminants of concern.

The Canadian water supply referred to by Mr Archer was known to be contaminated with human sewage. This raises a number of questions:

- Were the treatment processes provided appropriate given this source of contamination?
- Did the indicators used provide assurance of process control and, therefore, water quality?
- What were the environmental and recreational impacts to the waterways and the consequences for all users of the water?

These questions need a broad, strategic answer and cannot be avoided by a different monitoring regime. In assessing the implications of the new water plants the Water Board has made such a holistic commitment to managing Sydney's water supply. As well as upgrading the water treatment processes, the Water Board has a concurrence role over development in the "inner catchments"; it is promoting catchment management of privately owned land and is eliminating or upgrading sewage effluent disposal within the catchments.

Based on information provided by the Water Board the Department does not believe the levels of *Cryptosporidium* found in Sydney's water supply in June 1993 represented a public health threat. Mr Archer is quite right, however, when he states that we really do not know the infective dose for *Cryptosporidium*. It is considered to be quite low – maybe 10 oocysts or fewer, and possibly as low as 1 oocyst per person. Certainly the dose will vary for individuals and is most critical for immunologically impaired groups. The absence of a specific action level for *Cryptosporidium* in the most recent (draft) NHMRC Guidelines for Drinking Water is a reflection of this uncertainty. An animal model has recently been developed which may provide more information on the behaviour of the organism.

The Health Department is developing a more clearly defined audit role for drinking water quality as a result of the proposed corporatisation of the Water Board. Any agreement will address all issues of water quality, including raw water supplies, quality assurance/quality control and the most appropriate indicators for routine and event monitoring.

PUBLIC HEALTH ABSTRACTS

Professor James S. Lawson, Professor and Head of the School of Health Service Management at the University of NSW, has prepared the following public health items from the literature.

HIGH RATES OF DISABILITY IN IMMATURE INFANTS

Neo-natal intensive care has been described as 'perhaps the most successful of all medical technologies'. In terms of improving the chance of survival this may be so, but the rate of disabilities among the survivors is high as has been shown by a comprehensive survey involving nearly 100,000 infants in the United Kingdom. About 3.5 of 1,000 of these births were before 29 weeks of gestation. Half the babies survived to be discharged from the nursery. At four years, 93 per cent of the premature infants were still alive. Only 35 per cent of those four-year-olds were within normal limits. Around 29 per cent had mild disability, 13 per cent a moderate disability and 23 per cent were severely disabled. The severe disablements included cerebral palsy, blindness, severe hearing loss and intellectual handicap. A number of babies had multiple disabilities. An important finding was that the incidence of disability increases with declining gestational age of the babies.

Johnson A, Townshend P, Yudkin P et al. Functional abilities at age 4 years of children born before 29 weeks of gestation. *Br Med J*, 1993; 306:1715-1718.

NEVER DISMISS WHAT A PATIENT TELLS YOU

When a 90-year-old was admitted to hospital he asked the staff to let his mother know. The doctors thought he must be senile. In fact, his mother, at 113 years, is the oldest woman in Britain.

Editorial. *Br Med J*, 1993; 307:48-49.

SEX, PREGNANCY, HORMONES AND MELANOMA

Many questions remain unanswered about the relationship between melanoma (the most rapidly increasing Australian cancer) and the hormonal environment. Several conclusions can be made within the current state of knowledge. First, there is no evidence that the use of oestrogens, either as oral contraceptives or hormone replacement therapy, has a role in the aetiology of melanoma. Second, women have a survival advantage over men that could be due to the inhibitory effect of normal oestrogens in the growth of melanoma. Third, prescribed oestrogens do not promote progression of the disease in patients with melanoma, therefore women who have been treated for melanoma can safely use hormonal supplements. Last, pregnancy seems to carry no adverse effect on survival after treatment for melanoma. (However, patients with thick melanomic lesions are advised to delay pregnancy for two to three years as this is when they are at the greatest risk of relapse).

Jatoi I and Gore ME. Sex, pregnancy, hormones and melanoma. *Br Med J*, 1993; 307:2-3.

BREAST-FEEDING REDUCES RISK OF BREAST CANCER

A large British study has confirmed that breast-feeding is associated with a statistically significant decreased risk of breast cancer. The risk of breast cancer falls with increased duration of breast-feeding, and with the number of babies breast-fed. However, breast-feeding each baby for longer than three months confers no additional benefits.

United Kingdom National Case-Control Study Group. Breast feeding and risk of breast cancer in young women. *Br Med J*, 1993; 307:17-20.

INFECTIOUS DISEASES

TABLE 3

INFECTIOUS DISEASE NOTIFICATIONS FOR 1994 BY PUBLIC HEALTH UNIT, RECEIVED BY 27.1.94

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	SWR	SER	U/K	Total
AIDS	–	–	–	–	1	–	–	–	–	–	1	–	–	–	–	–	2
Arboviral Infection	–	–	–	–	–	–	–	–	–	–	3	1	–	–	–	–	4
Gonorrhoea	–	–	2	–	2	–	–	1	–	–	–	3	2	–	–	–	10
H. influenzae epiglottitis	–	1	–	–	–	–	1	–	–	–	–	–	–	–	–	–	2
H. influenzae infection (NOS)	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1
H. influenzae meningitis	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	1
H. influenzae septicaemia	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1
Hepatitis A – acute viral	–	–	4	–	–	–	1	–	–	2	1	4	–	–	–	–	12
Hepatitis B – acute viral	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1
Hepatitis B – unspecified	10	5	4	1	4	–	12	–	1	3	–	1	–	1	–	–	42
Hepatitis C – acute viral	–	–	–	–	–	–	–	–	–	–	–	–	–	1		–	1
Hepatitis C – unspecified	5	4	21	–	4	1	11	3	–	9	13	4	–	8		1–	84
Hepatitis – acute viral (NOS)	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
HIV infection	4	–	8	2	–	–	–	–	–	–	–	–	–	–	–	8	22
Leptospirosis	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1
Malaria	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Measles	5	1	4	1	9	3	4	1	1	2	29	3	9	–	1	–	73
Meningococcal meningitis	–	1	–	–	1	1	–	–	–	–	–	–	–	–	–	–	3
Meningococcal septicaemia	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1
Mycobacterial tuberculosis	–	1	–	–	3	–	–	–	–	1	–	–	–	–	–	–	5
Pertussis	1	2	–	–	1	–	3	–	1	–	20	–	–	–	1	–	29
Q fever	–	–	–	–	–	–	–	–	–	1	1	1	–	–	–	–	3
Rubella	–	–	–	–	2	–	1	–	–	–	–	–	–	–	–	–	3
Salmonella (NOS)	–	4		–	3	1	1	1	–	4	–	2	3	2	–	–	21
Salmonella bovis morbificans	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Salmonella typhimurium	–	–	4	–	3	–	1	–	–	–	–	–	–	–	–	–	8
Syphilis	–	–	4	–	1	–	3	1	–	–	2	–	2	–	–	–	13

TABLE 4

SELECTED INFECTIOUS DISEASE NOTIFICATIONS BY PUBLIC HEALTH UNIT, RECEIVED BY 27.1.94

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	SWR	SER	Total
H. Influenzae epiglottitis	–	1	–	–	–	–	1	–	–	–	–	–	–	–	–	2
H. Influenzae meningitis	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1
H. Influenzae septicaemia	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	1
H. Influenzae infection (NOS)	–	1	–	–	–	–	1	–	–	–	1	–	–	–	–	1
Measles	5	1	4	1	9	3	4	1	1	2	29	3	9	–	1	73
Pertussis	1	2	–	–	1	–	3	–	1	–	20	–	–	–	1	29
Rubella	–	–	–	–	2	–	1	–	–	–	–	–	–	–	–	3

TABLE 5

FOODBORNE INFECTIOUS DISEASE NOTIFICATIONS BY PUBLIC HEALTH UNIT, RECEIVED BY 27.1.94

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	SWR	SER	Total
Salmonella (NOS)	–	4	–	–	3	1	1	1	–	4	–	2	3	2	–	21
Salmonella bovis morbificans	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1
Salmonella typhimurium	–	–	4	–	3	–	1	–	–	–	0	–	–	–	–	8

ABBREVIATIONS USED IN THIS BULLETIN:

CSA Central Sydney Health Area, SSA Southern Sydney Health Area, ESA Eastern Sydney Health Area, SWS South Western Sydney Health Area, WSA Western Sydney Health Area, WEN Wentworth Health Area, NSA Northern Sydney Health Area, CCA Central Coast Health Area, ILL Illawarra Health Area, HUN Hunter Health Area, NCR North Coast Health Region, NER New England Health Region, OFR Orana and Far West Health Region, CWR Central West Health Region, SWR South West Health Region, SER South East Health Region, OTH Interstate/Overseas, U/K Unknown, NOS Not Otherwise Stated.

Please note that the data contained in this Bulletin are provisional and subject to change because of late reports or changes in case classification. Data are tabulated where possible by area of residence and by the disease onset date and not simply the date of notification or receipt of such notification.

FIGURE 2

HAEMOPHILUS INFLUENZAE TYPE B, NSW FEBRUARY 1993–JANUARY 1994

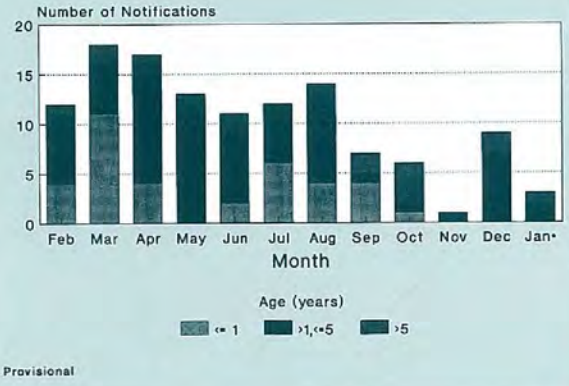


TABLE 6

INFECTIOUS DISEASE NOTIFICATIONS BY SELECTED MONTH OF ONSET NOVEMBER 1993 – JANUARY 1994

Condition	Month			
	Jan 94	Nov 93	Dec 93	Total
Adverse event after immunisation	1	–	1	2
AIDS	5	28	33	66
Arboviral infection	10	16	11	37
Cholera	–	–	1	1
Foodborne illness (NOS)	1	13	8	22
Gastroenteritis (instit.)	1	73	24	98
Gonorrhoea	16	33	41	90
H influenzae epiglottitis	2	–	3	5
H influenzae infection (NOS)	1	–	1	2
H influenzae meningitis	1	1	3	5
H influenzae septicaemia	1	–	1	2
Hepatitis A – acute viral	20	40	33	93
Hepatitis B – acute viral	–	11	5	16
Hepatitis B – unspecified	86	408	294	788
Hepatitis C – acute viral	1	5	–	6
Hepatitis C – unspecified	174	770	607	1551
Hepatitis D – unspecified	1	–	1	2
Hepatitis, acute viral (NOS)	1	–	–	1
Hydatid disease	–	2	–	2
Legionnaires' disease	1	9	5	15
Leptospirosis	1	1	1	3
Listeriosis	–	1	1	2
Malaria	3	7	5	15
Measles	99	589	314	1002
Meningococcal meningitis	3	12	9	24
Meningococcal septicaemia	1	5	4	10
Mumps	–	3	3	6
Mycobacterial – atypical	–	27	7	34
Mycobacterial infection (NOS)	1	16	9	26
Mycobacterial tuberculosis	8	23	15	46
Pertussis	48	308	184	540
Q fever	6	28	22	56
Rubella	3	121	25	149
Salmonella (NOS)	32	89	39	160
Salmonella bovis morbificans	1	1	3	5
Salmonella typhimurium	11	11	18	40
Syphilis	26	93	70	189
Tuberculosis – non active	–	6	5	11
Typhoid and paratyphoid	–	1	2	3
Total	566	2751	1808	5125

NOTIFICATIONS

HAEMOPHILUS INFLUENZAE TYPE B

The immunisation program against *Haemophilus influenzae* type B, introduced in July 1993, is already demonstrating excellent effect on the epidemiology of this infection. No notifications for *Haemophilus influenzae* type B have been received for the past three months for infants. Only three notifications were received for January, for a rate of 0.6/100,000 population. This compares with a notification rate of 2.0/100,000 population for January 1993.

PERTUSSIS (WHOOPING COUGH)

The pertussis notification rate for the State for January was 4.1/100,000 population. This compares with a rate of 22.2 for 1993. North Coast Public Health Unit (PHU) received 14 notifications at a rate of 44.2/100,000 population.

Fifteen per cent of notifications were for children under five years of age. A further 55 per cent of notifications were for school-aged children.

MEASLES

The notification rate for the State is 9.2/100,000 population. This compares with a rate of 38.5 for 1993. The North Coast PHU received 21 notifications at a rate of 66.3/100,000 population.

Measles notifications in Western Sydney peaked in epiweek 44 of 1993. The infant measles immunisation schedule in the outbreak-affected areas (Blacktown and Penrith) reverts to 12 months of age on March 1.

The mean age for notifications was 7.0 years (range six months to 19 years). Eleven per cent of notifications were for neonates and infants. Fifty-six per cent of notifications were for children over the age of five years, while 16 per cent were for people 12 years and older.

From July 1 this year the schoolgirl rubella program is expected to be replaced by a universal schoolchild measles-mumps-rubella program.

Continued on page 20 ▶

TABLE 7

NOTIFICATIONS OF NON-NOTIFIABLE SEXUALLY TRANSMITTED DISEASES JANUARY-DECEMBER 1993
(Diagnoses from sexual health centres unless otherwise stated in footnote)

* First diagnosis; 1. 01/01/93-31/12/93; 2. 01/01/93-30/11/93; 3. 01/01/93-30/06/93; 4. 01/01/93-31/08/93; 5. No SHC in Region; 6. Laboratory and SHC data 01/01/93-30/11/93; 7. No SHC in Region. Data from GP network 01/01/93-31/10/93.

AHS Infection		CSA[1]	SSA[2]	ESA[1]	SWS[1]	WSA[2] + WEN	NSA[2]	CCA[2]	ILL[3]	HUN[4]	NCR[2]	NER[1]	OFR[4]	CWR[5]	SWR[6]	SER[7]
Chlamydia trachomatis	Male	3	4	96	7	29	3	–	8	11	2	4	13	–	12	
	Female	1	5	75	10	22	1	1	4	32	2	15	13	–	27	
	Total	**4**	**9**	**171**	**17**	**51**	**4**	**1**	**12**	**43**	**4**	**19**	**26**	**–**	**39**	**4**
Donovanosis	Male	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Female	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Total	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**
*Genital herpes	Male	10	13	351	3	42	12	7	7	21	5	3	3	–	3	
	Female	10	12	214	2	24	3	9	8	24	6	9	5	–	17	
	Total	**20**	**25**	**565**	**5**	**66**	**15**	**16**	**15**	**45**	**11**	**12**	**8**	**–**	**20**	**3**
*Genital warts	Male	55	86	727	127	211	33	31	62	93	41	16	20	–	2	
	Female	29	66	303	49	85	19	14	25	37	23	25	15	–	1	
	Total	**84**	**152**	**1030**	**176**	**296**	**52**	**45**	**87**	**130**	**64**	**41**	**35**	**–**	**3**	**15**
Nongonococcal urethritis	Male	11	15	748	32	357	14	15	52	69	20	6	13	–	1	
	Female	2	–	–	3	3	4	5	–	–	4	–	1	–	–	
	Total	**13**	**15**	**748**	**35**	**360**	**18**	**20**	**52**	**69**	**24**	**6**	**14**	**–**	**1**	**–**
Lymphogranuloma venereum	Male	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Female	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Total	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**

► Continued from page 19

FOODBORNE ILLNESS

Notifications of salmonella were received from 10 PHUs in the January reporting period. A total of 30 (5.1/100,000 population) notifications was received in this period. In the same period last year 16 (2.7/100,000 population) notifications were received. The highest rate of notifications of 6 (1.0/100,000 population) occurred in the Western Sydney and Wentworth Areas. It should be noted that these data are only provisional, as the final number of notifications for January 1993 was 123 (20.8/100,000 population).

The Microbiological Diagnostic Unit, University of Melbourne, where *Salmonella typhimurium* phage typing is done in association with the National Salmonella Surveillance Scheme, notified Epidemiology Branch of an unusual cluster of 13 cases of *Salmonella typhimurium* phage type 9 with isolation dates between January 2 and January 27. Only two notifications of *S. typhimurium* phage type 9 were received in January 1993. No geographic relationship is evident except for three cases from a small community in the Illawarra Area.

Two apparently related cases of listeriosis — one isolated on January 27 in an 80-year-old female and the other on January 30 in a 31-year-old male — are being investigated by Eastern Sydney PHU. Both cases are immuno-compromised individuals.

IMPROPERLY STORED SPIT ROAST: THE CAUSE OF A FOODBORNE OUTBREAK

I Beer, MJ Ferson, Eastern Sydney Public Health Unit

The host of an adult's birthday party contacted the Public Health Unit to report suspected food poisoning among a number of guests. Results of the investigation into the source of illness, including epidemiological analysis, microbial examination of foodstuffs and inspection of food handling methods, are outlined in the following report.

Methods

A guest list and menu were obtained and a questionnaire seeking demographic, food and illness information was prepared. Those who attended were contacted and interviewed by telephone. A case was defined as any person who developed gastrointestinal symptoms within 48 hours of the party. The surveys were analysed using EpiInfo software. Food samples were collected for routine examination by the NSW Health Department's Division of Analytical Laboratories.

Results

Food preparation

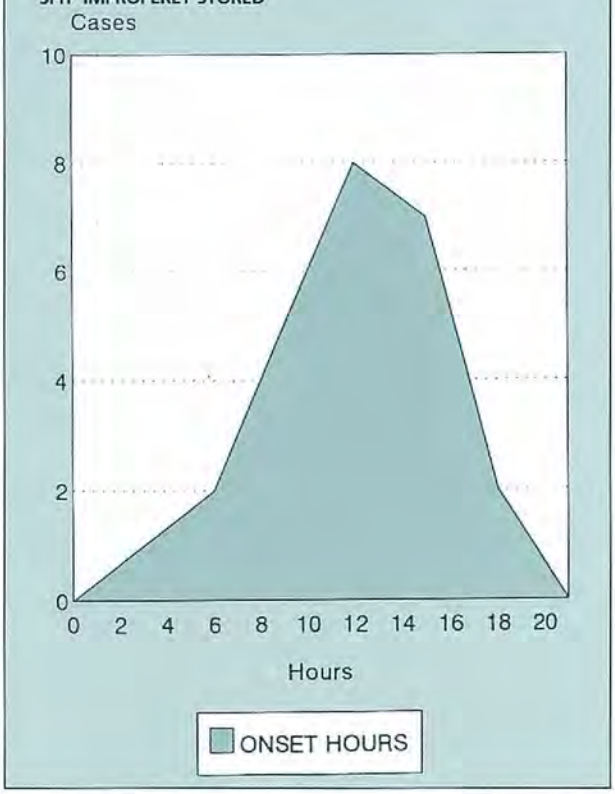
Most of the foods served at the party were prepared by family members. The spit roasts, consisting of a large cut of beef and legs of pork, were cooked and reheated at the function by a chef employed by a commercial caterer. Left-over meats were submitted for microbial analysis.

Inspection of the caterer's premises revealed that the preparation areas were confined and dirty as a result of renovations. In particular the thermometer used to check the temperature of the meat had a build up of grease covering the probe.

The beef had been cooked at very low heat (80°C) for approximately 18 hours, by which time the core temperature had reached 60°C. The legs of pork had been cooked at a high temperature for about four hours until the core temperature reached 60°C. Both meats had been allowed to cool at room temperature after cooking and prior to reheating at the party, for nine hours in the case of the beef and three hours for the pork.

FIGURE 3

EPIDEMIC CURVE, FOODBORNE OUTBREAK ASSOCIATED WITH SPIT IMPROPERLY STORED



Epidemiological analysis

Interviews were conducted with 41 (55 per cent) of the 75 guests. Of the 25 cases, all suffered diarrhoea, 76 per cent abdominal cramps and 32 per cent nausea. Nobody complained of nausea. The mean incubation period calculated from the hour the meal started until the onset of first symptom was 12±4.0(SD) hours, range 5-19 hours (Figure 3). The mean duration of symptoms was 24±15 hours (range 3-56).

Food-specific odds ratios and corresponding P-values were calculated. The only foods significantly associated with illness were the roast pork and roast beef (P=0.006). The importance of these two foods could not be separated as all guests ate either both roasts or neither.

Microbial examination

Stool specimens were collected from two guests still recovering from the illness. They were cultured according to routine procedures for *Salmonella*, *Shigella* and *Campylobacter*, and found to be negative for these pathogens.

Left-over meat, consisting of large portions of beef and pork still on the bone, were cultured separately for *Clostridium perfringens*, *Salmonella* and *Escherichia coli*. *C perfringens* was found in all samples at levels ranging from 4.0×10^6 to 3.3×10^7 per gram. *E coli* was found at high levels in four samples ranging from 9.3×10^2 to $>1.0 \times 10^6$ per gram.

When *C perfringens* was found in the meats, the clinical laboratory was contacted to determine whether it was possible to look for the bacterium in the stool specimens. However, as these clostridia are normally present in the bowel of healthy persons they are not sought in faecal specimens.

Discussion

The short incubation period, duration of symptoms and predominance of diarrhoea amongst cases are consistent with *C perfringens* type A food poisoning[1,2]. No other pathogens were detected in either the stools or food samples. Concentrations of *C perfringens* were in excess of the 10^6 per gram required to produce enough toxin to cause food poisoning in otherwise healthy individuals[2]. The epidemiological results implicated the pork and beef as the most likely foods to have caused illness.

Most outbreaks of *C perfringens* are associated with improperly cooked, stored or reheated meat products. In these outbreaks it is common to find that the meat has been stored for long periods of time at ambient temperatures. In this case, the roast pork and beef were not cooked to a core temperature of at least 75°C required to kill vegetative cells[2,3], and had been kept at room temperature for several hours. Spores would have survived the cooking process, then germinated and multiplied rapidly during the cooling and reheating of the spits.

Conclusions

In this outbreak of *C perfringens* food poisoning, proper food preparation and storage techniques were not observed and temperature controls were grossly lacking.

Food safety measures need to consider the ability of *C perfringens* to multiply at temperatures of up to 50°C and to form spores. Since multiplication does not occur at refrigerator temperatures, virtually all cases of food poisoning are caused by failure to properly refrigerate cooked foods, especially those stored in large portions.

Spores present in raw meat can survive cooking and resume cell growth when the meat cools. The enterotoxin produced by *C perfringens* type A is destroyed if heated to 60°C for ten minutes[2,3]. Cooked meat should be maintained at a temperature above 60°C or below 5°C during storage. Reheated meats should reach a core temperature of 75°C immediately before serving to destroy vegetative cells.

---

1. Roach RL, Sienko DG. *Clostridium perfringens* outbreak associated with minestrone soup. *Am J Epidemiol* 1992;136:1288-1291.
2. Lund BM. Foodborne disease due to *Bacillus* and *Clostridium* species. *Lancet* 1990; 336:982-986.
3. Labbe RG. Bacteria associated with foodborne diseases, *Clostridium perfringens*. *Food Technol* Apr 1988:195-196.

TYPHOID FEVER ACQUIRED AT HOME

Mark J Ferson and Lorraine C Young,
Eastern Sydney Public Health Unit

Case one

The Victorian Health Department informed the Eastern Sydney PHU on December 6 of a case of typhoid in a young child living in Melbourne. Two weeks before the onset of illness, the child had spent a week with family in Sydney. Neither the index case nor his contacts had recently travelled overseas. Stool specimens were collected from all the child's family and household contacts in Melbourne. The child's mother became ill with typhoid 18 days after the onset of illness in her child. The PHU followed up the four family members living in Eastern Sydney. *Salmonella typhi* was isolated from three of four specimens from the 53-year-old grandfather living in Sydney. He had spent most of his life in Southeast Asia, and although he had no history of typhoid fever, one of his sons had had typhoid fever as a child, 16 years ago. The grandfather was about to have surgery for gall stones, but this was deferred pending antibiotic treatment of his typhoid carrier state.

Case two

The Prince of Wales Children's Hospital notified the PHU on December 17 of the growth of *Salmonella typhi* from the blood of a three-year-old girl with an eight-day history of anorexia, vomiting, watery diarrhoea and high fever. Neither the child nor other family members had recently travelled overseas. The PHU arranged collection of stool specimens from the household, which consisted of parents, two siblings, a grandmother and an uncle. The organism was isolated from two specimens obtained from the 68-year-old grandmother, who was born in Greece and remembers having typhoid fever as a child.

Discussion

Between January 1991 and December 1993, 31 cases of enteric fever were notified to Eastern Sydney PHU. These consisted of 22 (71 per cent) cases of typhoid, 6 (19 per cent) of paratyphoid A and 3 (10 per cent) of paratyphoid B. There were 19 (61 per cent) males and 12 females; ages ranged from 3-68 years, and 25 (81 per cent) were individuals of 15 years and over.

The vast majority of cases had travelled overseas (25), generally to destinations in Asia or South America, or had been born overseas (3). Two cases, including Case two, had most likely acquired the infection from a household contact. In one case the source of infection was not determined.

The cases described highlight the importance of obtaining faecal specimens from all household contacts when the source of the infection in the index cases is not readily apparent.

IMMUNISATION RATES IN THE ILLAWARRA

Illawarra School Health Service and the Illawarra Public Health Unit

Few measures in public health are as effective as childhood immunisation programs. However the success of these programs depends on high immunisation coverage, and assessing this coverage can be difficult.

Obtaining accurate estimates of the immunisation status of children in NSW is complicated by the fact that there is no standardised immunisation surveillance system[1]. Childhood immunisation services are provided by general practitioners (GPs), local government and community health services. The Australian Bureau of Statistics (1983)[2] found that about 60-70 per cent of immunisations are conducted by GPs and the remainder by community programs. Collecting and collating reliable data on immunisation coverage from all these sources has obvious problems.

One method of assessing childhood immunisation coverage is by accessing the immunisation information collected by the School Health Services. In the Illawarra this information is routinely collected on the immunisation status of all school entry children. The School Health Service is also responsible for implementing the Year Seven schoolgirl rubella program and the Adult Diphtheria Tetanus (ADT) and Sabin boosters for Year 10 students.

Continued on page 22 ▶

► Continued from page 21

The high school data are very reliable and easily accessed. The limitation of the school entry information is that it relies on parental recall, as parents are not required to provide documented evidence of immunisation status, and it does not provide any information on age-appropriate immunisation. This will change after 1994 with the introduction of the Public Health (Amendment) Act 1992. The Act will require parents of children starting school in the kindergarten class to provide documentation of immunisation status in the form of an immunisation certificate and of children enrolling in child care centres to provide documentation of age-appropriate immunisation. The introduction of this legislation should provide more reliable data.

Results

School entry immunisation

The school entry data include all primary schools in the Illawarra Area, which extends from Helensburgh in the north to Gerringong in the south. This is a total of 53 primary schools with a kindergarten enrolment of 3,043. Of this number 2,543 (83.6 per cent) were fully immunised, 456 (15 per cent) partly immunised and only 18 (0.5 per cent) not immunised at all. There were 26 children of the total enrolment of 3,043 who did not return their screening cards to the school. Table 8 shows the immunisation status of the Illawarra's school entry children for 1992.

The school health screening card requests a yes/no/not sure answer as to whether the child had three immunisations as a baby, a booster at 18 months, a booster at or before school entry and a measles/mumps vaccination. Table 9 lists the immunisation, the number of school entry children who were reported to be fully immunised for that immunisation in 1992 and shows the National Health and Medical Research Council (NHMRC) targets to the year 2000[3].

TABLE 8

1992 IMMUNISATION STATUS OF ILLAWARRA'S SCHOOL ENTRY CHILDREN

Immunisation status	Number	
Fully immunised	2,543	83.6%
Partially immunised	456	15.0%
Unimmunised	18	0.5%
No record	26	0.8%
Total	3,043	100%

TABLE 9

IMMUNISATION CATEGORY, NUMBER OF CHILDREN IMMUNISED AND NHMRC TARGETS TO THE YEAR 2000

Immunisation	Number immunised	NHMRC targets 1994	1996	2000
Three immunisations as a baby	3,008 (99.7%)	90%	95%	99%
18-month booster	2,977 (98.6%)	90%	95%	99%
Preschool booster	2,858 (94.7%)	90%	95%	99%
Measles/mumps	2,709 (89.7%)	90%	95%	99%

TABLE 10

ILLAWARRA SCHOOLGIRL RUBELLA IMMUNISATION PROGRAM 1993

Response	Number and percentage of girls	
Immunised by SHS*	1,329	80%
Immunised by GP	176	10%
Total immunised	1,568	90%

* SHS = School Health Service

TABLE 11

ILLAWARRA HIGH SCHOOL ADT/SABIN BOOSTER IMMUNISATION PROGRAM FOR 1993

Response	Number and percentage of students	
Immunised by SHS*	2,331	74%
Immunised by GP	195	6%
Total immunised	2,526	80%

* SHS = School Health Service

SCHOOLGIRL RUBELLA AND THE ADT/SABIN BOOSTER PROGRAM

The Illawarra School Health Service coordinates the Year Seven schoolgirl rubella and the ADT/Sabin booster program in all high schools — both government and non-government — in the Illawarra Area. There are 23 high schools with a total enrolment of 1,678 Year Seven girls and 24 high schools with a total enrolment of 3,156 Year 10 students. Tables 10 and 11 show the response to information and consent forms sent home with Year Seven girls for the rubella program and Year 10 students for the ADT/Sabin booster program. Students absent on the day of immunisation are offered immunisation the next year.

Conclusion

The results obtained from the School Health Service indicate good immunisation coverage of children aged 5-15 years in the Illawarra, with 99.7 per cent of all school entry children having received all their baby (two months, four months, six months) immunisations, 98.6 per cent having received their 18-month triple antigen, 94.7 per cent their preschool booster and 89.7 per cent their measles/mumps. Ninety per cent of Year Seven girls were vaccinated for rubella and 80 per cent of Year 10 students received their ADT/Sabin boosters.

These results compare very well with the 1989-1990 National Health Survey conducted by the Australian Bureau of Statistics which found that only 53 per cent of children less than six years old had received full age-appropriate immunisation[4]. The Illawarra's immunisation rates are on target with the recommendation from the NHMRC's National Immunisation Strategy, which has set national goals for immunisation coverage to the year 2000. These include a 90 per cent coverage by 1994, 95 per cent coverage by 1996 and near universal coverage by 2000 of all children of school entry age for diphtheria, tetanus, pertussis, polio, measles/mumps/rubella and adolescent measles/mumps/rubella.

The introduction in NSW of school entry legislation for 1994 should increase the Illawarra's immunisation rate

of measles/mumps/rubella from 89.7 per cent to 95 per cent, allowing it to achieve the NHMRC's national goals.

1. Carey M. A Review of Childhood Immunisation in New South Wales. NSW Health Department, Epidemiology and Health Services Evaluation Branch. 1991.
2. Australian Bureau of Statistics. Children's immunisation survey New South Wales, November 1983. Sydney: Australian Bureau of Statistics. Catalogue No 4352.1. 1985.
3. National Health and Medical Research Council Communicable Diseases Committee. Report by the Panel on a National Immunisation Strategy. April 1993
4. Australian Bureau of Statistics. 1989-1990 National Health Survey. Children's immunisation, Australia Catalogue No 4379.0, 1992

Q FEVER: SOUTH COAST DISTRICT

Warren Matthews, Peter Hlavacek, Greg Sam
South Eastern Public Health Unit

The South Eastern Public Health Unit investigated a cluster of 11 cases of Q fever on the NSW South Coast. All cases were diagnosed on clinical and epidemiological grounds and 10 were serologically confirmed.

All cases were at a party attended by 23 people on a beef cattle property on June 19, 1993. During the party the index case (the owner of the property) assisted in the birth of a calf. The calving was witnessed by most of those at the party. Strong winds prevailed at the time of the calving. There was no indication that the cow was diseased, and the birth was uncomplicated.

The index case developed symptoms consistent with Q Fever 12 days after the birthing. The acute phase of the illness lasted 10 days and was characterised by fever, headaches, generalised myalgia, malaise, depression and an enlarged liver. Within the ensuing 35 days a further 10 cases exhibited symptoms consistent with Q fever.

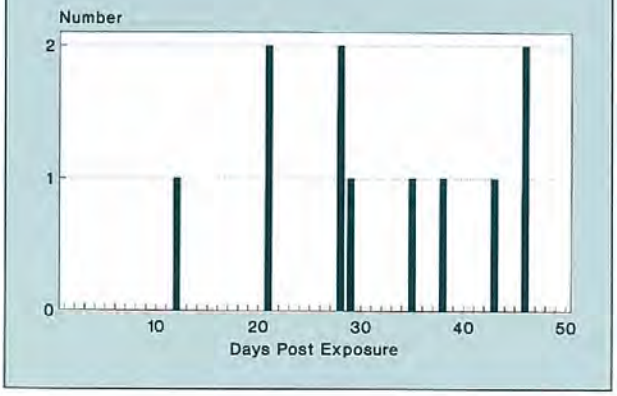
Discussion

The index case was most probably infected during the delivery of the calf on June 19, 1993. The cases who witnessed the calving were most probably infected through direct inhalation of contaminated aerosols post-partum. The cases who had not been at the calving were probably indirectly infected as no other direct exposure was reported.

A 37-year-old woman and her 22-month-old breastfed child were at the calving. The mother developed symptoms after nine days and the child some 21 days after her mother. This interval raises the possibility of secondary transmission via contaminated breast milk.

FIGURE 4

Q FEVER SOUTH COAST



Active surveillance of the local area identified three further cases unrelated to this event, however all had visited the property and developed symptoms within the 35-day period.

The Department of Agriculture's veterinary service states that the incidence of *Coxiella burnetii* in cattle and sheep is common and usually asymptomatic. Testing of beef cattle is not routinely performed. Testing for Q fever in the cow and calf involved in this episode is being undertaken.

While large outbreaks of Q fever are uncommon, a survey of literature revealed episodes similar to this outbreak. Kosatsky (1984) reports 12 people developing Q fever after contact with a cat which had just given birth.

Sporadic cases of Q fever in occupational settings have resulted in improved awareness among employees, employers and health professionals. Routine immunisation has been recommended to abattoir staff and individuals with clearly defined occupational risk.

Kosatsky T. Household outbreak of Q fever pneumonia related to a parturient cat. Lancet 1984; ii:1447-1449.
Benenson Abram S. Control of Communicable Diseases in Man. Fifteenth Edition, 1990.
Miles T. Q fever Vaccination Programme for the Hunter Area. NSW Health Department. January 1992.

INVESTIGATION OF AN OUTBREAK OF HEPATITIS A LINKED TO A RESTAURANT

Sami Gounder, Gay Rixon and Helen Longbottom

In September 1993 the Northern Sydney Area Public Health Unit, with the assistance of the Western/ Wentworth Public Health Unit, investigated an outbreak of hepatitis A among a group of people who had eaten at the same restaurant.

A private laboratory noted a geographic cluster of four cases of hepatitis A. Subsequent investigation by the PHU, with the co-operation of general practitioners, revealed the cases were linked to a local restaurant. Active surveillance identified four other cases of hepatitis A also linked to the restaurant.

Review of the incubation periods of all cases showed it was possible they were linked to a single index case. Analysis of the food histories of the cases implicated a specific food preparation process.

Members of the PHU inspected the restaurant and interviewed the owner. There was no history of illness among restaurant staff and this was confirmed by a review of employment records. Serology on some restaurant staff did not show evidence of recent hepatitis A infection. A number of deficiencies was noted in the kitchen design and the food preparation and handling techniques. These were reported to the owner and the recommended changes have been implemented.

Although we were unable to identify the index case, our investigation showed a definite link between the cases and the restaurant. Since the investigation and the implementation of our recommendations there have been no further cases of hepatitis A linked to this restaurant. The investigation highlights the effectiveness of the notification system; the collaboration that is developing between the PHUs and private laboratories; and the difficulties in identifying the source of foodborne outbreaks.

NEWS AND COMMENT

UNLEADED PETROL AND BENZENE

A Clever Country – The Health Benefits of Removing Lead from Petrol[1] in the *Bulletin* was a sensible article as far as it went. It quotes the NHMRC as advocating the increased use of unleaded petrol and accelerated reduction of lead in petrol. Further, it reports that the Lead in Petrol Working Group is considering an education campaign be instituted to encourage owners of pre-1986 cars to switch to using unleaded petrol (for those models which can do so).

It is disappointing that Stephen Corbett and Christine Cowie of the Environmental Health Section responsible for the article appear to be unaware of reports from Europe that the use of unleaded petrol without a catalytic converter constitutes an increased risk of leukaemia. The culprit is said to be carcinogenic benzene which is discharged in the exhaust if no catalytic converter is used. In addition, the additive MTBE in unleaded petrol is reported to have been found in the environment as a result of leaking underground petrol station tanks.

The National Society for Clean Air in Britain is reported now to feel that 'to represent unleaded fuel as greener than leaded is misleading'. Professor Roger Perry, of the Imperial College, London, is quoted as saying: "If I had the option of being exposed to low levels of lead or very low levels of benzene, I would go for the lead any time."

Donald Scott-Orr
Department of Health, London

AUTHORS' RESPONSE

In response to our article[1] Dr Scott-Orr correctly raises concerns about the possible health hazards of fuel octane enhancers such as benzene or other aromatic hydrocarbons which have been used as an alternative to tetra ethyl lead since the early 1980s. In doing so he echoes the concerns which have been raised in Italy and in some other European countries that the carcinogenic effects of benzene in the environment may outweigh any gains achieved by reducing environmental lead contamination.

This is unlikely to be a problem in Australia. Unleaded petrol used in Australia has a specified Research Octane Number (RON) of 91, while in Europe the RON of unleaded petrol is 95. In Europe this higher octane rating is achieved in part by increasing the amount of aromatics, including benzene, in the fuel. The important point to make is that the transition to the use of unleaded fuel in Australia will not be accompanied by an increase in the amount of aromatic compounds added to the fuel mix.

If on health grounds current levels of benzene were found to be hazardous then these policies would need revision. However, the low levels of benzene found in ambient air in Australian cities suggests that benzene will not be a major problem here.

Policy at both Federal and State levels is to lower the amount of lead added to petrol by about 25 per cent in all States and to reduce RON from 97 to 96, with a view to further reduction in 12 months. If there continues to be a high demand for high octane fuel the introduction of octane enhancers will have to be considered. In the meantime, industry and government have initiated research into the:

- capacity of industry to reduce lead levels while maintaining satisfactory octane and environmental performance;
- capacity of the leaded fleet to operate satisfactorily on reduced octane fuel; and
- feasibility, costs and benefits of using alternative fuel octane enhancers such as Methyl Manganese Tricarbonyl (MMT) and Methyl Tertiary Butyl Ether (MTBE).

1. Corbett S, Cowie C. A Clever Country – The Health Benefits of Removing Lead from Petrol. NSW Public Health Bulletin November 1993.

MENTAL HEALTH DIRECTORY AND WALL CHART

In 1993 the Mental Health Branch published the first edition of the *Directory of Mental Health Services in NSW*. It was the first comprehensive listing of mental health services and facilities to be published and distributed by the NSW Health Department.

On the basis of the high level of responses, comments and alterations to entries which followed the launch and distribution of the directory, a second edition was printed almost immediately and distributed in January 1994. In addition to public mental health services, the second edition has a comprehensive listing of non-government and multicultural services which are relevant to mental health.

It was felt that professionals such as GPs, police and Department of Community Services officers would also benefit from having readily accessible information on the services available in their Area/District, rather than a directory for the whole State. Accordingly, we are printing wall charts for each Area/District in NSW which will be distributed free of charge to such groups and other interested groups/professionals for whom this information would be valuable.

The charts are easy to read and a convenient size for offices and waiting rooms. They should be available from this month.

PROFESSIONAL DEVELOPMENT

Public health is a rapidly developing profession, with many workforce issues ranging from training to employment conditions being debated. To encourage this debate and communication on these issues, the editor of the *Bulletin* invites submission of short articles, letters and commentary. These will be included in this column under the title of Professional Development. Contributions from public health bodies and universities which run public health courses are welcomed.

1994 PUBLIC HEALTH OFFICER PLACEMENTS

Public Health Officers have been assigned the following placements:

PHO	Feb 1994-July 1994	Aug 1994-Jan 1995
Third Year Officers		
Glen Close	Chronic Diseases*	Chronic Diseases*
Susan Furber	Eastern Sydney Area PHU (Part time)	Eastern Sydney Area PHU (Part time)
Cait Lonie	Injury*	Health Service Development and Planning Branch
Justine Waters	Health Outcomes*	Health Outcomes*
Second Year Officers		
Magnolia Cardona	Western Sector PHU	Western Sector/Orana and Far West PHUs
Jennifer Chipps	Environmental health*	Environmental health*
Leena Gupta	Health Service Evaluation*	Health Service Evaluation*
Bernie Towler	Orana and Far West PHU	AIDS Bureau
First Year Officers		
James Blogg	Health Service Development and Planning Branch	Health Service Development and Planning Branch
Suzanne Blogg	Health Promotion	Health Promotion
Hugh Burke	Broken Hill	Broken Hill
Tony Butler	Eastern Sydney Area PHU	Hunter PHU
Valerie Delpech	Infectious Diseases*	Maternal and Child Health*
Gerard Fitzsimmons	Western Sector PHU	Southern Sydney Area PHU
Veth Guevarra	Maternal and Child Health*	Infectious Diseases*
Stephen Hooppell	AIDS Bureau	Northern Sydney Area/ Central Coast Area PHUs
Jeannine Liddle	Northern Sydney Area/ Central Coast Area PHUs	Western Sector PHU

* Epidemiology and Health Services Evaluation Branch

SUPERVISORS' WORKSHOP

The first workshop for supervisors of Public Health Officers was held on February 15, 1994. Its overall objective was to discuss issues of supervision in the context of adult learning, culminating in a list of "best practice" points for supervisors. Associate Professor Jackie Lublin, Director of the Centre for Teaching and Learning in the University of Sydney, was the workshop facilitator and 24 PHO supervisors attended. A full article on the workshop will appear in a forthcoming issue of the *Bulletin*.