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INVESTIGATION OF POSSIBLE PATIENT-TO-PATIENT TRANSMISSION OF HEPATITIS C IN A HOSPITAL

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In December 1993 the NSW Health Department was notified that two patients had presented with acute hepatitis C five and seven weeks after undergoing minor surgical procedures in the same operating session at a Sydney private hospital early that year. Neither case had other risk factors for acute hepatitis C. This article summarises progress to date in epidemiologic and laboratory investigations to determine how these cases (subsequently known as Case B and Case C) became infected. The investigations were carried out by the South Western Sydney Area Health Service (SWSAHS) and the Department's Public Health Division, with the consent and co-operation of the patients and the staff involved in their care.

EPIDEMIOLOGIC INVESTIGATION

Initially this comprised the following steps:

- confirmation of the diagnoses of acute hepatitis C;
- review of the patients' risk factors and medical history, including a search for stored blood or evidence of any previous testing for antibodies to hepatitis C virus (anti-HCV), in an attempt to pinpoint possible times of exposure;
- anti-HCV testing of hospital staff involved in the surgery session;
- anti-HCV testing of patients who had procedures in the same hospital's operating rooms on the same day as Cases B and C, the following day and the preceding day; and
- a review of infection control policies and practices at the hospital.

The diagnosis of acute hepatitis C in both Cases B and C was based on clinical symptoms (jaundice, lethargy, nausea and vomiting), abnormal liver function tests consistent with acute viral hepatitis, and the exclusion of other common causes of viral hepatitis. Case B was found to be anti-HCV positive 10 weeks after the surgical procedure, having been negative at the onset of the illness three weeks beforehand. Case C was anti-HCV positive on initial presentation seven weeks post-surgery.

Thirteen patients underwent surgical procedures in the same operating session as Cases B and C (the index session). The other 11 people were tested for anti-HCV, and three were found to be anti-HCV positive. They were designated Cases A, D and E.

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Anti-HCV testing was offered to 51 patients who were treated in other operating sessions on the same day as the index session, or on the day before or the day after. To date, 47 people have been tested for anti-HCV, and one person was found to be anti-HCV positive (designated Case F). Case F had a procedure on the afternoon before the index session.

Cases A, D, E and F were interviewed in relation to risk factors for hepatitis C and previous hepatitis C testing history. Relevant medical and occupational records were reviewed to identify possible risk factors.

None of the five cases who had procedures in the index session had had prior testing for anti-HCV, and no stored specimens from before the index session were available for testing on any of the cases.

LABORATORY INVESTIGATIONS

Serological testing was performed by the South Western Area Pathology Service (SWAPS) and local private pathology services. All anti-HCV positive patients were tested or retested by SWAPS using a second generation enzyme linked immunosorbent assay (ELISA)[a]. Samples found to be positive using this assay were reassayed by the NSW Blood Bank[b].

Anti-HCV positive sera were tested for the presence of hepatitis B surface antigen, hepatitis B core antibody and hepatitis B surface antibody[c].

Hepatitis C virus (HCV) RNA detection by reverse transcriptase polymerase chain reaction (PCR) assays was used to detect hepatitis C virus in aspectically collected blood samples from Cases B, C, D, E and F and on stored serum from Cases A and C. These tests were carried out by the Victorian Infectious Diseases Reference Laboratory (VIDRL)[d], Fairfield Hospital, Melbourne. All PCR results were confirmed by the VIDRL[e].

Genotyping of HCV RNA positive cases was also performed by the VIDRL[f]. Further comparison of the HCV RNA sequences is in progress.

FINDINGS

The theatre list for the index session

The order of the five cases who had surgery during the index session is shown in Table 1 where the results of reverse transcriptase(RT) PCR to detect HCV viraemia are also recorded.

a Sanofi Diagnostics Pasteur, France.
b Murex Diagnostics and Abbott Diagnostics, and further characterised using an immunoblot assay (Innogenetics, Belgium).
c Roche Diagnostic Systems.
d This assay used nested primers from the untranslated region (5' UTR) with two sequential rounds of amplification for greater sensitivity.
e The confirmatory test was Roche Diagnostics Systems HCV RNA detection test.
f Genotyping was done using a reverse phase hybridisation assay developed by Innogenetics that allows simple and rapid determination of HCV genotypes after PCR amplification[1].

TABLE 1

THEATRE LIST POSITIONS OF THE FIVE CASES WHO HAD SURGERY IN THE INDEX SESSION

Cases	Position on theatre list	HCV RT PCR Early 1993	HCV RT PCR Early 1994
A	5	not done	positive
B	6	not done	positive
C	8	positive	negative
D	9	not done	positive
E	10	not done	positive

Results of tests to detect hepatitis C virus in the cases (Table 1)

Case F, whose operation was on the previous day to the index session, was also HCV RT PCR positive.

Description of the cases

Case F

- Female in her 30s.
- Clinical history suggests that she acquired chronic hepatitis C before 1993.

Case A

- Male in his 40s.
- History of hepatitis B about 20 years ago. A review of his medical records revealed he had risk factors for hepatitis C.
- Serologic testing indicated past exposure to hepatitis A and hepatitis B.

Case B

- Female in her 20s.
- No history of having received blood or blood products, injecting drug use, tattooing, electrolysis or occupational exposures to blood or body fluids.
- She had only one sexual partner over five years, and he tested anti-HCV negative early in 1994.

Case C

- Female in her 60s.
- No history of having received blood or blood products since 1969 (she had normal immunoglobulin in 1965, a blood transfusion in 1965, and anti-D following delivery of her children). No history of injecting drug use, tattooing, electrolysis or occupational exposures to blood/body fluids.
- She has had one lifetime sexual partner who tested anti-HCV negative in 1994 and all current household contacts tested anti-HCV negative.

Case D

- Female in her 20s.
- No history of having received blood or blood products, injecting drug use, tattooing or electrolysis. She was employed as an assistant at two nursing homes for several years. Her duties involved assisting residents with their activities

of daily living. No documentation of needlestick injuries in the staff incident book or employment records maintained by the nursing homes.

- She has had one lifetime sexual partner who tested anti-HCV negative early in 1994.

Case E

- Male in his 30s.
- No history of having received blood or blood products, injecting drug use, tattooing or occupational exposures to blood or body fluids.
- He has had one sexual partner over 12 years, and she tested anti-HCV negative early in 1994.
- The procedure in the index session was his first invasive procedure.

Results of anti-HCV tests on surgical personnel and hospital staff

The surgeon, anaesthetist, surgical assistant, theatre and recovery nurses and the orderly associated with the index session were anti-HCV negative after the index session.

The hospital's infection control policies and practices

Review process

Policies and practices were reviewed by a team comprising a microbiologist, infectious disease clinicians, an infection control nurse, a theatre nurse educator, anaesthetist and Public Health Unit staff. In addition structured interviews on infection control were administered to relevant medical and theatre nursing staff and the infection control nurse of the hospital. The review focused on ward areas, operating rooms, the central sterile supply unit (CSSU) and the recovery area.

Pre-operative procedures

The cases were hospitalised in different ward areas. Premedications were given in different areas and at different times, and multi-dose vials were not used.

Surgical instrument sterilisation and usage

All cases had surgical procedures performed in the same operating room. Case A's procedure involved a steroid injection into a joint. Cases B, C, D and E had a variety of procedures. Because the nature of their procedures was different, it was extremely unlikely that any particular instrument(s) could have been used on all cases.

It was reported that all surgical instruments were washed manually, then autoclaved, either in a "flash" autoclave in the operating suite, or in a jacketed downward displacement autoclave in the CSSU. All autoclaves were reportedly checked weekly with spore tests, and daily with chemical indicators. There was a written service report indicating that a routine service was done on the autoclaves on the afternoon following the index session, and the contractor stated that they were all operational before and after the service.

The team was advised that a drill, which was used on Case F, would have been used on Case C and probably also on Case B, but not the other cases. This drill was also used on the person immediately following Case F, and this person tested anti-HCV negative. A saw used on Case F could have been used on Case B, but this saw was also used on the person immediately following Case F and on the first patient on the index session, both of whom tested anti-HCV negative.

Assuming the reported procedures were followed, the saw and drill would have been flash autoclaved when required for consecutive patients in a session and both instruments would have been through a jacketed autoclave cycle in CSSU the evening before the index session.

Anaesthetic procedures

All five cases had general anaesthesia, Laryngeal masks (LMs) were used on Cases A, B, C, D and E. It was reported that a packed sterile LM was used for each person. Size 3 LMs were used for females, and size 4 for males, so it would have been impossible for a single LM to be used on all cases. The reported practice of the anaesthetist was to insert the LM without a laryngoscope, after spraying the deflated LM cuff with a lignocaine 10% solution. He reported that he did not spray lignocaine directly into the patient's oropharynx.

He further reported that it was his practice to draw up intravenous anaesthetic agents in separate syringes if the injectate from one ampoule was to be used on two or more patients. Fentanyl and propofol were administered to all five cases. The drug register for fentanyl shows that none of the five cases shared a common fentanyl ampoule. Purchase records showed that propofol was bought only in 200mg ampoules at the time of the index session. The dose of propofol given to the cases ranged from 120mg to 220mg. Therefore, even if an ampoule was shared, it is extremely unlikely that it would have been used for more than three patients. The anaesthetist reported that he used an 18- or 19-gauge needle for drawing up the propofol in a 20ml syringe, and discarded the needle and syringe after drawing up or administering the dose for each patient.

Sucker heads and/or suction catheters were reportedly changed after each patient. Separate suction equipment was used in operating rooms and in the recovery area. Some patients, such as Cases A and C, were unlikely to have required suction in the recovery area because they were recorded as having been conscious on arrival. Others were unlikely to have required suction in the operating room because their LMs were recorded as having been still *in situ* when they arrived in the recovery area.

Anaesthetic circuits, consisting of inspiratory and expiratory tubing with a "Y"-piece at the "patient end", were reportedly changed at the end of every operating session. The anaesthetist stated that neither bacterial nor viral filters were used in the anaesthetic circuit.

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Hepatitis C investigation

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Genotyping results

Genotyping showed that Case F was infected with HCV genotype 3a and that Cases A, B, C, D and E were infected with HCV genotype 1a.

COMMENTARY

Hepatitis C is a recently discovered RNA virus and is the most common cause of non-A, non-B hepatitis. It is transmitted primarily by blood-to-blood contact, although viral RNA has been detected in other body fluids such as saliva, ascites fluid and semen[2,3]. Transmission of hepatitis C by blood products, by needle or equipment sharing during injecting drug use, and through occupational injury by "sharps" contaminated with infected blood[4], have been well documented. Sexual transmission of hepatitis C and vertical transmission from mother to baby occurs[5,6], but less frequently than for hepatitis B or HIV. Transmission of hepatitis C within households (possibly by sharing shaving equipment)[7] has been reported. In addition the potential for saliva to transmit hepatitis C has been postulated[2,8]. Acute hepatitis C is usually anicteric, and it was the coincidence of two clinically jaundiced patients among the four which drew attention to the incident.

Apart from possible nosocomial transmission of hepatitis C involving renal patients in dialysis units[9a, 9b], no other instance of patient-to-patient transmission of hepatitis C has been reported to our knowledge.

In this investigation healthcare worker-to-patient transmission was excluded. However patient-to-patient transmission remains the most plausible explanation for the cluster. Two potential source patients were identified: Case A, who underwent a procedure prior to Cases B, C, D and E in the index session; and Case F, who underwent a procedure in the afternoon before the index session. At the time of writing, one person who had a procedure on the day before the index session remains to be tested. Apart from haemostats, the instruments reportedly used on this patient were common only to Case D.

The review of the pattern of surgical instrument usage and sterilisation procedures indicated that Case F could not have been the source, a conclusion supported by the viral genotyping. This left Case A as the most likely source because:

- in the operating list, Case A preceded Cases B, C, D and E.
- his risk factor history suggested exposure to hepatitis C virus before the index day (probably in the 1970s or 1980s); and
- genotyping demonstrated that Cases A, B, C, D and E had the same genotypes (type 1a).

The possibility of the cluster being due to chance was explored. By chance alone, the probability that the four out of five sequential patients, who followed case A and who had no identifiable risk factor for infection, were anti-HCV positive was estimated to be 2.5×10^{-10} (assuming that the prevalence of hepatitis C was 0.4% in a population without risk factors for hepatitis C infection – a typical prevalence for NSW blood donors).[10]

Thus the cluster is most unlikely to have been due to chance. This is corroborated by the facts that two of the cases (Cases B and C) developed acute hepatitis C 5-7 weeks after the index session, no risk factors were identified to explain these cases' illnesses, and no hepatitis C risk factors were identified for Cases D and E.

While no clear mechanism of transmission of hepatitis C virus from Case A to Cases B, C, D and E was identified, the available evidence suggests that patient-to-patient transmission occurred during the surgical session early in 1993. The genotyping results give further support to this conclusion, but there have been only limited studies of HCV types in Australia and it appears that type 1 is commonly found[11]. Direct sequencing of HCV RNA from Cases A, B, C, D and E is in progress and may provide better evidence of the degree of the relatedness of the HCV strains.

The results of the investigation suggest that transmission did not occur via surgical instruments, multidose vials, reuse of laryngeal masks, laryngoscopes or suction equipment.

Below are two possible mechanisms which could account for transmission to Cases B, C, D and E. The second hypothesis is the more plausible of the two.

The first hypothesis involves inadvertent contamination and reuse of a needle or syringe used to draw up the intravenous anaesthetic agents propofol or fentanyl. Given that the propofol ampoules contain only 200mg each, inadvertent contamination of an ampoule might account for transmission to Case B but could not account for transmission to Cases C, D and E, unless there was contamination of the drawing-up needle or syringe and subsequent reuse. The anaesthetist and anaesthetic nurse assistants report that a sterile needle and syringe is used to draw up each dose, as is the routine practice.

The second hypothesis involves contamination of anaesthetic circuitry. Case A may have coughed at some stage during the procedure, introducing respiratory secretions into a reusable part of the anaesthetic circuitry (e.g. the "patient end" of the "Y"-piece). This would then have acted as a reservoir for the virus, which could have been transmitted to other patients as droplets via minor breaks in their oropharyngeal mucosa. By providing a clear airway, the laryngeal mask may have facilitated transmission. Significantly, the seventh patient on the theatre list, who has remained anti-HCV negative, had a short anaesthetic without the use of a LM. Both the duration of anaesthesia and airway management technique may have prevented transmission of the virus.

LMs commonly cause minor trauma to the pharyngeal mucosa, evidenced by bloodstained secretions on the laryngeal mask on removal. In a review of 25 cases in 1994 who had LMs inserted, 5 out of 25 (20%) had macroscopic evidence of blood on the LM.[12]

Transmission via bloodstained respiratory secretions is more plausible than transmission via saliva or non-bloodstained respiratory secretions, because salivary transmission of hepatitis C has not been documented. However, HCV RNA has been detected in saliva[2,3], experimental transmission of hepatitis C by saliva to a chimpanzee has been described[8], and transmission of hepatitis C by a human bite has been reported[13].

Conclusion

This investigation has drawn attention to possible patient-to-patient transmission of hepatitis C in a private hospital.

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Editorial Comment

The report by Chant et al of possible patient-to-patient transmission of hepatitis C in a private hospital operating session comes soon after the report of possible patient-to-patient transmission of HIV. The latter event has triggered a re-evaluation of infection control procedures, and widespread changes have already occurred in clinical practices for the disinfection and sterilisation of reusable equipment. The hepatitis C investigation underlines the importance of the reviews of infection control policies and practices which are currently in progress nationally and in NSW. Available evidence has led to a hypothesis that hepatitis C virus was transmitted through contamination of anaesthetic circuitry. The NSW Infection Control Policy for HIV, AIDS and associated conditions, published in 1992, states that "A filter for the anaesthetic circuit must be used to prevent cross-infection of the anaesthetic circuit." This precaution was primarily intended to prevent transmission of airborne pathogens such as *Mycobacterium tuberculosis*. Reportedly, filters were not used in the operating session in which hepatitis C transmission is hypothesised to have occurred, and it appears that they were not in general use at this time. As part of its review of infection control procedures, the NSW Health Department is assessing compliance with the Infection Control Policy, and associated implementation and enforcement issues.

Public Health Editorial Staff 1995

The editor of the Public Health Bulletin is Dr Michael Frommer, Director, Research and Development, NSW Health Department. Dr Lynne Madden is production manager.

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MALIGNANT MESOTHELIOMA AND ASBESTOS EXPOSURE IN NSW

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INTRODUCTION

Malignant mesothelioma is a cancer of the mesothelial cells which line body cavities and envelop organs such as the lung (pleura), heart (pericardium) and intestines (peritoneum). Exposure to asbestos or asbestiform fibres is the only known cause of the disease and accounts for 80-85 per cent of all cases[1]. It is one of the very few human cancers which is a hallmark of exposure to a single environmental agent.

Workplace exposure to asbestos, particularly to the mineral amphiboles (crocidolite and amosite) is the main risk factor for this disease[2]. In Australia this exposure is most likely to have occurred in the period 1944-1966 in the asbestos mining, manufacturing or processing industries, in shipbuilding or in the construction industries[3]. Concerns linger about the potentially hazardous effects of environmental or asbestos exposure. Some authors have predicted a "third wave of asbestos-related disease"[4] because of this environmental exposure.

In this article we examine the patterns of production and use of asbestos in NSW and trends in the incidence and distribution of malignant mesothelioma in NSW.

ASBESTOS PRODUCTION, CONSUMPTION AND EXPOSURE IN NSW

Asbestos fibre types can be ranked in terms of their propensity to induce mesothelioma: erionite (a zeolite mineral linked with very high rates of mesothelioma in rural Turkey), crocidolite (blue asbestos), tremolite, amosite (brown asbestos) and chrysotile (white asbestos)[5]. Chrysotile asbestos was by far the type used most commonly in Australian industry[6].

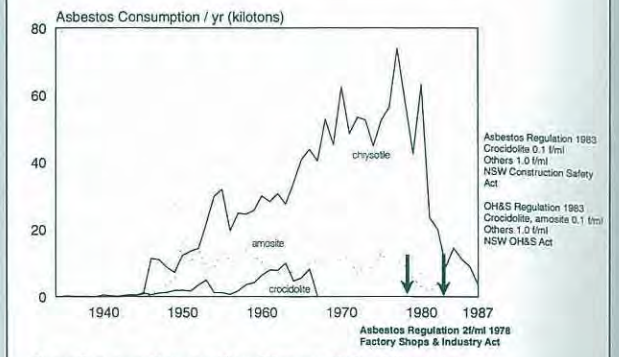
Among Australian States, NSW has been the leading consumer of all commercial types of crude asbestos fibre. Two asbestos mines, at Baryulgil and Barraba, produced significant amounts of chrysotile asbestos for the local market and for export. Added to this were imports of chrysotile, crocidolite and amosite for use in a wide range of industries. Data from the National Mesothelioma Surveillance Program reveal that of those people exposed to asbestos, 66 per cent had their first asbestos exposure in NSW[3].

Australian import, export and production figures for the years 1935-1980, by fibre type, were obtained from reports published by the Commonwealth Bureau of Mineral Resources, Geology and Geophysics[7,8]. The consumption of asbestos was calculated from these figures (Figure 3). Imports of processed asbestos products were excluded from this calculation. Also included in this figure are the important legislative changes which had an impact on workplace asbestos exposure over this period. The use of crocidolite asbestos declined rapidly after 1966, and imports were banned in 1968, but chrysotile and to a lesser extent amosite had peak exposures in the 1970s and declined only in the early 1980s.

There has been a sharp decline in usage in the building and construction industry but asbestos continues to be used in the automotive industry (for brake linings) and for industrial gaskets and seals.

FIGURE 3

ASBESTOS CONSUMPTION IN AUSTRALIA, BY FIBRE TYPE, 1947-87, AND DATES OF MAJOR LEGISLATED REFORMS IN ASBESTOS CONTROL IN NSW



Source: Commonwealth Bureau of Mineral Resources, Geology & Geophysics.

MALIGNANT MESOTHELIOMA IN NSW

We obtained data on mesothelioma occurrence in NSW from the Australian Mesothelioma Surveillance Program (the program) and the Australian Mesothelioma Register (the register). The rationale and methods used in these data collections are described in detail elsewhere[3]. Briefly, from 1980 to 1985 there was an intensive case finding and detailed data collection on notified mesothelioma cases. When the program ended, the register was established to continue monitoring of the incidence of mesothelioma and to collect less detailed information on each case. For this analysis, data on year of birth, sex, age at diagnosis, year of diagnosis and postcode were available. Data after 1991 are likely to be incomplete because of the lag time involved in notification.

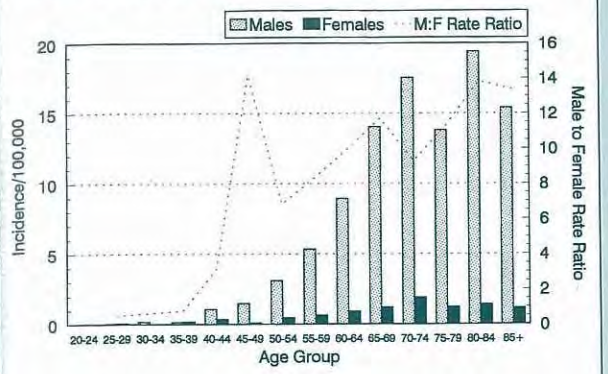
Data on mesothelioma incidence between 1947 and 1980 have been drawn from data published by Musk et al[9]. These data were obtained from members of the Royal College of Pathologists of Australasia. The NSW data from this paper have been appended to data from the program and the register to illustrate long-term trends in mesothelioma incidence. Standardised Incidence Rates (SIR) were calculated using the world population as a reference population. The age-adjusted trend for males and females between 1980 and 1990 was estimated using Poisson regression[a]. The criterion of significance for an increasing trend was that the confidence limits of the mesothelioma trend did not overlap the confidence limits for the trend in cancers at all sites[10].

All statistical analyses were conducted using SAS statistical package Version 6.08. Directly age-standardised rates were calculated using the method of Dobson et al[11]. SIR and their

a Poisson models were fit using proc genmod in SAS Windows Version. The model used was: log (incidence rate) = $\alpha_0 + \beta_1$ (age) + β_2 (year) Age entered as a grouped variable produced a better model fit. The average annual change in incidence rate was calculated from the maximum likelihood estimate of the parameter for year.

FIGURE 4

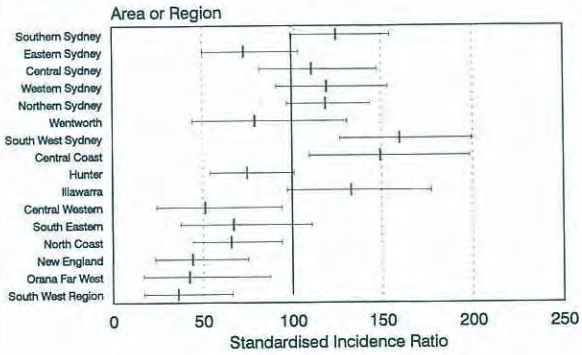
MESOTHELIOMA INCIDENCE IN NSW 1980-1992 BY AGE GROUP AND SEX



Source: Australian Mesothelioma Program and Register

FIGURE 5

MESOTHELIOMA IN MALES NSW 1980-1989 STANDARDISED INCIDENCE RATES BY AREA OR REGION



Source: Australian Mesothelioma Program and Register
Reference Population = NSW

95% confidence intervals[12] were calculated for each Area and Region, using the NSW population as a reference.

RESULTS

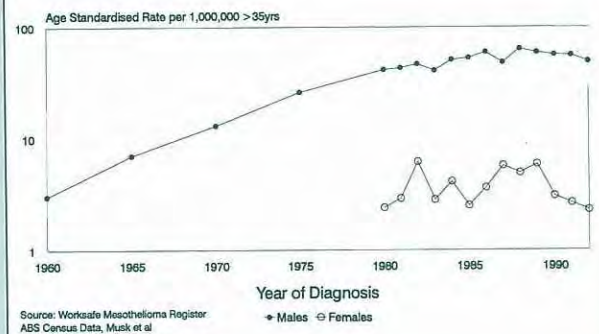
Between 1980 and 1992 there were 992 cases of mesothelioma in NSW. Males accounted for 88.3 per cent of cases. The mean age for males was 65.9 years and for females was 64.4 years (Figure 4). The male:female ratio rises rapidly with increasing age.

SIR for mesothelioma among males by Area and Region are shown in Figure 5. Both the Central Coast and South West Sydney Areas had higher than expected cases of mesothelioma. The SIR for the Central Coast was 1.56 (95% CI 1.15-2.05) and for South West Sydney was 1.60 (95% CI 1.27-2.00).

Over the past 15 years there has been a significant increase of 4.2 per cent a year (95% CI 2.7-5.9%) in the incidence of malignant mesothelioma in men. In women the rate of increase is 3.8 per cent (95% CI -0.005-8.3%), but this result is not statistically significant (Figure 6). Age-specific trends show the steepest increase is in men above 60 years of age (Figure 7).

FIGURE 6

MALIGNANT MESOTHELIOMA NSW 1960-1992 AGE-STANDARDISED INCIDENCE RATES



Source: Worksafe Mesothelioma Register
ABS Census Data, Musk et al

DISCUSSION

The rising incidence of malignant mesothelioma in NSW is the regional manifestation of what has been called "the most disastrous occupational epidemic in Australia's history". This disease claims more lives each year than any other work-related cause of death[13]. This review of trends in incidence data in NSW provides little evidence that the end of the epidemic is in sight. The 4 per cent annual increase in incidence is comparable to increases observed nationally[14]. The steep increase in incidence between 1975 and 1980 is due to the systematic under-estimation of mesothelioma incidence before the National Mesothelioma Surveillance Program. While the uncertainties of ascertainment of previous individual histories of occupational exposure and lung clearance rates of the fibres limit the ability to predict the final trajectory of this epidemic it is not likely that the incidence rate will begin to fall until 2010[13].

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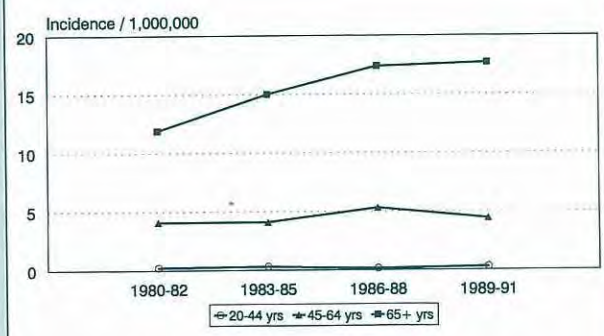
FIGURE 7

AGE-SPECIFIC TRENDS IN THE INCIDENCE OF MALIGNANT MESOTHELIOMA IN NSW 1980-1991



Source: Worksafe Mesothelioma Register ABS Census Data, Mask et al.

Malignant mesothelioma and asbestos

► Continued from page 53

In NSW, as elsewhere, the major impact of this epidemic has been on men who worked in high-risk industries such as asbestos mining or manufacture, shipbuilding or construction. The risk was highest in the period before legislated health and safety prohibitions on exposure and during the period of high asbestos consumption between 1940 and 1975.

The incidence rate for malignant mesothelioma in NSW is one of the highest in Australia. Only Western Australia and South Australia have higher rates and in Western Australia many cases can be attributed to exposure to crocidolite in the course of employment or residence at Wittenoom[15]. These findings accord with the previously mentioned observation that NSW was the State in which more than two-thirds of all mesothelioma cases reported nationally were first exposed to asbestos, and with the concentration of asbestos manufacturing and heavy industries in this State.

The distribution of reported cases in NSW shows, as expected, higher rates in urban than in rural areas and higher rates in industrial areas, with the Hunter region being an exception. High rates in the Gosford-Wyong region probably reflect the high number of elderly people in that area. However, the average time between first exposure to asbestos and the diagnosis of mesothelioma is 37 years[3] and this long latency period increases the chance of committing the ecological fallacy – wrongly attributing cause or place of first exposure to a locality.

In NSW, occupational exposure to asbestos is limited to the small numbers of workers in the now tightly regulated asbestos removal and brake lining and gasket manufacturing industries. The contemporary public health issue is whether the very low levels of asbestos found in both the urban environment and in the lungs of most urban dwellers[16] will be a cause of future cases of malignant mesothelioma. In the US and in Britain the absence of any rising trend in mesothelioma incidence in women is cited as evidence that ambient exposure to asbestos does not increase occurrence of mesothelioma. This inference cannot be made in NSW, where the incidence of mesothelioma in women is rising. This increase may be attributable to well-recognised occupational or para-occupational exposures or to non-occupational exposures. The fact that a significant proportion of women with mesothelioma (46 per cent) report previous exposure in both the program (1978-85) and the register (1986-92)[17] supports the former hypothesis, although the surveillance of mesothelioma trends in women, and their exposure histories, should continue.

There have been no general surveys of non-occupational exposure to asbestos in NSW, but overseas data indicate that exposures in the urban population not directly exposed to asbestos are likely to be in the < 0.0001-0.0005 fibres/ml range[1]. If an individual is exposed for a lifetime to asbestos levels of this magnitude, it has been estimated that he or she has less than a 1 in 100,000 chance of developing malignant mesothelioma[18]. These estimates suggest it is unlikely that exposure at these levels will contribute substantially to the occurrence of mesothelioma in coming years. The tragic inevitability of future cases of mesothelioma in NSW can therefore be largely attributed to exposures which occurred between 1940 and 1980.

ACKNOWLEDGMENTS

The authors thank Jim Leigh and Carlos Corvalan at Worksafe Australia for providing the mesothelioma data.

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NOTIFICATIONS

HAEMOPHILUS INFLUENZAE TYPE B (HIB)

Only 21 notifications for Hib disease were received for the first four months of 1994, for a rate of 1.02/100,000 population. This compares with a notification rate of 2.81/100,000 population for the same period in 1993.

Immunisation surveillance in the Northern Districts of NSW reveals coverage levels of 70 per cent. The Commonwealth Department of Human Services and Health is expected to continue to support the Hib "catch-up" program beyond June 1994.

PERTUSSIS (WHOOPING COUGH)

Notifications for pertussis peaked in epiweek 7. Two-thirds of notifications for 1994 were for the period before epiweek 8.

The notification rate for pertussis for the first four months of 1994 was 20.1/100,000 population. This compares with a rate of 7.9 for the same period in 1993.

Nineteen per cent of notifications were for children less than five years of age, and a further 40 per cent were for school-aged children. The mean age for notifications was 22.0 years (range one month to 86 years).

North Coast Public Health Unit (PHU) received 146 notifications at a rate of 104.1/100,000 population.

MEASLES

Notifications for measles peaked in epiweek 1. The notification rate for the first four months of 1994 was 11.7/100,000 population. This compares with a rate of 10.5 for the same period in 1993.

The North Coast Public Health Unit received 67 notifications at a rate of 47.8/100,000 population.

The mean age for notifications was 8.0 years (range four months to 41 years). Fifteen per cent of notifications were for neonates and infants (≤ one year of age). Fifty-nine per cent were for children over the age of five years, and 25 per cent were for people 12 years and older.

From October 1, 1994 it is anticipated that the schoolgirl rubella program will be replaced by a universal schoolchild measles-mumps-rubella program (not September 1 as reported in the April 1994 *NSW Public Health Bulletin*).

LEGIONNAIRES' DISEASE

Only one confirmed notification for Legionnaires' disease was received for April 1994. In previous years April has recorded the highest number of notifications for this condition. In anticipation of April being a higher risk month, the NSW Health Department issued a warning to building owners to ensure that maintenance standards of water-cooled air-conditioning systems were optimal.

Since 1992, 149 isolates of *Legionella* have been recorded on the Infectious Diseases Surveillance System; 80 per cent have been for *L pneumophila*, 17 per cent for *L longbeachae* and 3 per cent for *L micdadii*.

INFLUENZA SURVEILLANCE

NSW general practitioner sentinel influenza surveillance shows low levels of influenza activity in April (1.2 per cent of consultations), comparable to levels at the same time in 1993. During April reports were received from almost 100 doctors in both rural and metropolitan NSW through eight

FIGURE 8

HIB, NSW, <= FIVE YEARS OF AGE MARCH 1993-MAY 1994

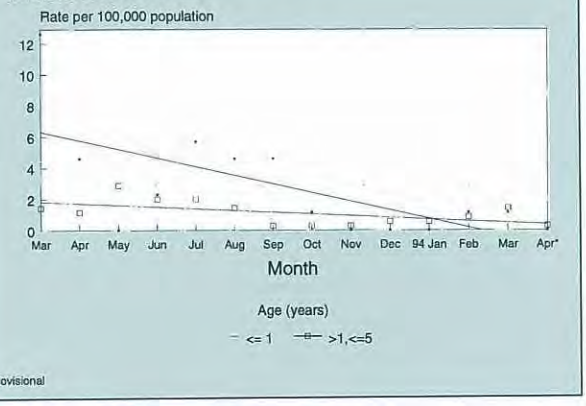
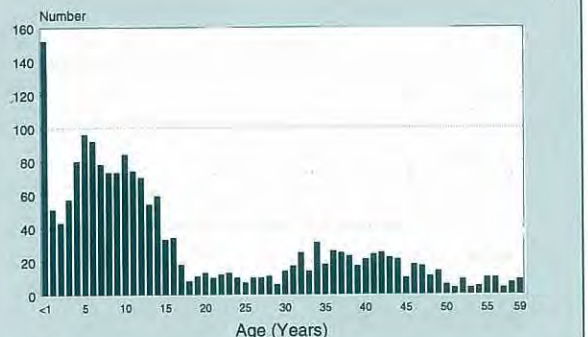


FIGURE 9

PERTUSSIS NOTIFICATIONS, NSW BY AGE, JANUARY 1993-APRIL 1994



PHUs. The highest level of influenza-like illness recorded so far this year by a PHU is 2.4 per cent of consultations by Western Sector. Data on school absentee rates have been received from 10 schools through four PHUs. No significant increase in absentee rates has been detected. To the end of April, laboratory surveillance had detected no isolations of influenza virus and a small number of positive serological tests, including both influenza A and B.

RESPIRATORY VIRUS SCREEN

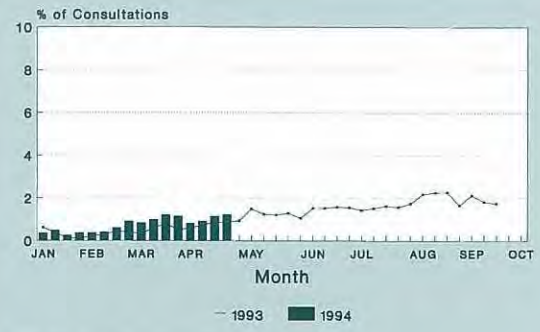
Each year it is important to identify which strains of influenza are circulating in the community and the extent of protection provided against them by the vaccine. Westmead ICPMR Virology Department has an important role as the NSW reference laboratory for influenza identification. Doctors are asked to submit samples to Westmead ICPMR from patients suspected of having influenza, particularly those ill early in the influenza season or index cases in outbreaks.

Collection requirements for viral culture

A throat swab specimen should be collected at the acute stage of the disease and sent with a minimum of delay. Specimen should be stored in a refrigerator and transported

FIGURE 10

"INFLUENZA-LIKE ILLNESS" NSW



(Source: NSW Sentinel GP Network)

to the laboratory on ice (but not frozen). When long-term storage is necessary, store at -70°C with dry ice and transport to the laboratory in that condition.

A cotton-tipped wooden or plastic swab can be used and transferred to viral transport medium (commercial medium is available). A dry swab should never be sent to the laboratory and Stuart's or other bacteriological medium is unsuitable.

All specimens must be submitted in leak-proof containers and all specimen containers should be enclosed in sealed plastic bags with the request form in the open section of the bag.

It is essential that all available information is on the request form, including date of onset of symptoms and other relevant clinical details.

Further information can be obtained by phoning Westmead ICPMR Virology Department on (02) 633 6230.

INFLUENZA CRITERIA (RACGP)

(A) Viral culture or serological evidence of influenza
or
(B) Influenza epidemic, plus four of the criteria in (C)
or
(C) Six of the following:
1. sudden onset (within 12 hours)
2. cough
3. rigors and chills
4. fever
5. prostration and weakness
6. myalgia, widespread aches and pains
7. no significant respiratory physical signs other than redness of nasal mucous membrane and throat
8. influenza in close contact

MEASLES IN ADULTS IN A HOME FOR THE INTELLECTUALLY DISABLED

Krishna Hort, Gay Rixon and Donald Holt
Northern Sydney Area Public Health Unit

The advent of vaccination has changed the epidemiology of measles. While most cases still occur in children in NSW, an increasing proportion of cases is occurring in adults. In 1993, 28 per cent of cases notified to the NSW Health Department were over 12 years[1].

This report describes two cases of measles reported in adults from a residential home for the intellectually disabled, and the actions taken by the Northern Sydney Area PHU to prevent further cases.

The index case, a female aged 36 years, developed measles, confirmed by serology, in January 1994, following a holiday visit with relatives. She had entered the residential home 18 months before, having spent most of her life in seclusion with her elderly mother. The second case, a female with Downs syndrome also aged 36 years, shared a room with the index case. She developed a measles rash 11 days after the onset of the rash in the index case; her diagnosis was also confirmed by serology. Neither case had any record of measles or measles vaccination.

There were 71 residents in the home, ranging in age from 18 to 71 years, who had been in contact with the two cases. Because of the possibility of spread to other susceptible people, the PHU (after discussion with the management of the home) decided to provide measles-mumps-rubella immunisation to residents under the age of 40 who did not have a history of measles or measles immunisation. Twenty-five residents were identified as under 40 years, of whom 13 fulfilled the criteria above, and all were vaccinated by the local doctor. No further cases of measles, or of reaction to the vaccination, were found on follow-up 18 days later.

This report illustrates the risk of adults developing measles in group residential settings and the measures that need to be considered in controlling an outbreak. Measles in adults over the age of 35 years is uncommon but it is possible that there were more susceptible people among our group of adults because of past seclusion and consequent lack of exposure to wild measles. The report also confirms that it is safe and sometimes appropriate to vaccinate susceptible adult contacts of measles, as recommended by the National Health and Medical Research Council[2] and the NSW Health Department[3].

Australian recommendations do not provide any guidelines about the upper age limit for vaccination of adult contacts. In the United States, where measles vaccine became available in 1963, people born before 1957 are generally considered immune to measles through contracting natural measles infection[4]. Measles vaccine was introduced in Australia in 1968, although coverage levels remained low for the first 10 years[5]. It is unlikely that people born in this country before 1968 would have been vaccinated, but most are likely to be immune from natural measles infection. However, there may be a small proportion of people born in the decade or so preceding the introduction of vaccination who did not have contact with natural measles, and therefore remain susceptible. Vaccination for such individuals in group residential settings should be considered in the event of a measles outbreak.

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TABLE 3

INFECTIOUS DISEASE NOTIFICATIONS FOR 1994 FOR NOTIFICATIONS RECEIVED BY APRIL 30, 1994 BY MONTH OF ONSET

Condition	Jan	Feb	Mar	Apr	Total
Adverse event after immunisation	4	4	–	4	12
AIDS	35	27	43	3	108
Arboviral infection	22	61	63	11	157
Foodborne illness (NOS)	16	5	4	34	59
Gastroenteritis (instit.)	1	11	9	–	21
Gonorrhoea	33	26	30	20	109
H influenzae epiglottitis	2	1	5	1	9
H influenzae meningitis	1	–	2	1	4
H influenzae septicaemia	1	1	1	–	3
H influenzae infection (NOS)	2	1	1	1	5
Hepatitis A – acute viral	49	46	43	10	148
Hepatitis B – acute viral	7	6	1	3	17
Hepatitis B – unspecified	306	289	336	82	1,013
Hepatitis C – acute viral	1	–	–	–	1
Hepatitis C – unspecified	557	695	601	176	2,029
Hepatitis D – unspecified	1	2	–	–	3
Hepatitis – acute viral (NOS)	1	1	–	–	2
HIV infection	24	39	39	13	115
Hydatid disease	–	1	1	–	2
Legionnaires' disease	3	4	4	1	12
Leptospirosis	1	2	2	–	5
Listeriosis	2	2	–	–	4
Malaria	5	9	7	–	21
Measles	147	64	28	5	244
Meningococcal meningitis	5	3	5	5	18
Meningococcal septicaemia	1	1	2	1	5
Meningococcal infection (NOS)	1	–	–	–	1
Mumps	1	–	–	–	1
Mycobacterial atypical	31	21	8	–	60
Mycobacterial tuberculosis	27	17	11	2	57
Mycobacterial infection (NOS)	7	13	10	4	34
Pertussis	169	127	98	22	416
Q fever	20	12	8	4	44
Rubella	8	6	4	–	18
Rubella – congenital	–	1	–	–	1
Salmonella (NOS)	59	69	77	34	239
Salmonella bovis morbificans	1	3	2	–	6
Salmonella typhimurium	47	51	46	8	152
Syphilis	84	83	95	25	287
Tetanus	–	–	–	1	1
Typhoid and paratyphoid	1	3	3	–	7
Total	1,683	1707	1,589	471	5,450

TABLE 4

SUMMARY OF NSW INFECTIOUS DISEASE NOTIFICATIONS APRIL 1994

Condition	Number of cases notified			
	Period		Cumulative	
	April 1993	April 1994	April 1993	April 1994
Adverse reaction	1	4	6	12
AIDS	29	3	143	108
Arboviral infection	64	11	533	157
Brucellosis	1	–	–	–
Cholera	–	–	–	–
Diphtheria	–	–	–	–
Foodborne illness (NOS)	17	34	49	59
Gastroenteritis (instit.)	–	–	39	21
Gonorrhoea	41	20	145	109
H influenzae epiglottitis	4	1	13	9
H influenzae B – meningitis	10	1	27	4
H influenzae B – septicaemia	3	–	11	3
H influenzae infection (NOS)	–	1	6	5
Hepatitis A	51	10	228	148
Hepatitis B	275	85	1,212	1,030
Hepatitis C	441	176	1,760	2,030
Hepatitis D	2	–	3	3
Hepatitis – acute viral (NOS)	1	–	2	2
HIV infection	46	13	202	115
Hydatid disease	–	–	–	2
Legionnaires' disease	14	1	33	12
Leprosy	–	–	–	–
Leptospirosis	1	–	8	5
Listeriosis	–	–	4	4
Malaria	9	–	67	21
Measles	29	5	213	244
Meningococcal meningitis	7	5	13	18
Meningococcal septicaemia	4	1	9	5
Meningococcal infection (NOS)	2	–	5	1
Mumps	–	–	–	1
Mycobacterial tuberculosis	31	2	136	57
Mycobacterial – atypical	32	–	142	60
Mycobacterial infection (NOS)	1	4	13	34
Pertussis	34	22	161	416
Plague	–	–	–	–
Poliomyelitis	–	–	–	–
Q fever	35	4	127	44
Rubella	29	–	149	19
Salmonella infection (NOS)	89	42	430	397
Syphilis	60	25	224	287
Tetanus	–	1	2	1
Typhoid and paratyphoid	4	–	18	7
Typhus	–	–	–	–
Viral haemorrhagic fevers	–	–	–	–
Yellow fever	–	–	–	–

TABLE 5

FOODBORNE INFECTIOUS DISEASE NOTIFICATIONS FOR NOTIFICATIONS RECEIVED BY APRIL 30, 1994 BY PUBLIC HEALTH UNIT

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	CWR	SWR	SER	U/K	Total
Foodborne illness (NOS)	1	10	7	10	13	7	3	4	1	–	–	–	2	–	–	1	–	59
Gastroenteritis (instit.)	12	1	–	3	3	–	–	1	–	1	–	–	–	–	–	–	–	21
Hepatitis A – acute viral	8	4	18	15	16	1	13	2	2	8	20	10	3	5	23	–	–	148
Listeriosis	–	–	1	–	–	–	–	–	1	1	–	–	1	–	–	–	–	4
Salmonella (NOS)	12	19	20	22	21	4	25	7	7	16	39	6	17	6	17	1	–	239
Salmonella bovis morbificans	–	1	1	1	1	–	1	–	–	1	–	–	–	–	–	–	–	6
Salmonella typhimurium	13	16	11	3	32	4	20	8	14	12	1	1	2	7	7	1	–	152
Typhoid and paratyphoid	3	1	2	–	–	1	–	–	–	–	–	–	–	–	–	–	–	7

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.

TABLE 6

INFECTIOUS DISEASE NOTIFICATIONS FOR 1994
FOR NOTIFICATIONS RECEIVED BY MARCH 30, 1994
BY PUBLIC HEALTH UNIT

Condition	CSA	SSA	ESA	SWS	WSA	WEN	NSA	CCA	ILL	HUN	NCR	NER	OFR	CWR	SWR	SER	U/K	Total
Adverse event after immunisation	–	–	–	2	3	2	–	1	–	–	1	–	–	–	2	1	–	12
AIDS	18	5	35	2	17	12	9	2	4	1	3	–	–	–	–	–	–	108
Arboviral infection	–	2	–	–	–	–	5	1	2	11	116	2	10	–	7	1	–	157
Gonorrhoea	8	7	49	4	6	1	5	3	2	4	2	3	9	1	3	2	–	109
H. influenzae epiglottitis	1	2	–	–	1	1	1	1	2	–	–	–	–	–	–	–	–	9
H. influenzae meningitis	–	–	–	1	1	–	1	–	–	–	–	–	–	1	–	–	–	4
H. influenzae septicaemia	–	–	–	–	1	–	–	–	–	–	2	–	–	–	–	–	–	3
H. influenzae infection (NOS)	–	–	–	–	1	–	1	2	–	–	1	–	–	–	–	–	–	5
Hepatitis B – acute viral	3	1	5	–	1	–	–	–	–	1	1	–	2	1	–	2	–	17
Hepatitis B – unspecified	136	123	104	257	148	6	143	14	18	27	18	1	4	5	9	–	–	1,013
Hepatitis C – acute viral	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1
Hepatitis C – unspecified	229	117	379	208	150	41	208	66	92	123	263	11	11	42	52	37	–	2,029
Hepatitis D – unspecified	–	–	–	–	–	–	1	–	–	–	2	–	–	–	–	–	–	3
Hepatitis, acute viral (NOS)	–	–	1	–	–	–	–	–	–	1	–	–	–	–	–	–	–	2
HIV infection	20	6	50	5	2	1	3	1	–	2	2	–	–	–	–	–	23	115
Hydatid disease	–	–	2	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Legionnaires' disease	1	1	1	1	2	–	3	–	2	–	–	–	–	1	–	–	–	12
Leptospirosis	1	–	–	–	–	–	–	–	–	2	1	–	–	–	1	–	–	5
Malaria	4	–	4	2	1	–	4	–	–	1	1	–	–	–	3	1	–	21
Measles	23	5	8	14	18	20	18	3	6	19	68	9	21	9	–	3	–	244
Meningococcal meningitis	1	2	2	2	3	1	–	2	–	2	1	–	–	1	1	–	–	18
Meningococcal septicaemia	–	1	–	1	–	–	–	1	–	1	1	–	–	–	–	–	–	5
Meningococcal infection (NOS)	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1
Mumps	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Mycobacterial atypical	15	4	17	–	1	–	15	–	–	2	3	–	–	1	2	–	–	60
Mycobacterial tuberculosis	5	12	5	8	13	2	1	1	3	4	2	–	–	–	1	–	–	57
Mycobacterial infection (NOS)	9	–	4	–	3	1	11	–	–	2	3	–	–	–	1	–	–	34
Pertussis	9	27	34	23	39	12	31	6	19	32	154	–	12	11	2	5	–	416
Q fever	–	–	–	–	–	–	–	–	–	8	10	5	19	–	2	–	–	44
Rubella	–	–	1	–	5	1	4	–	–	–	3	2	–	–	2	–	–	18
Rubella – congenital	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1
Syphilis	55	22	81	38	17	2	21	3	3	–	15	–	26	1	3	–	–	287
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1

TABLE 7

SURVEILLANCE OF NON-NOTIFIABLE SEXUALLY TRANSMITTED DISEASES
JANUARY-APRIL 1994
(Diagnoses from sexual health centres unless otherwise stated in footnote)

* First diagnosis; 1. 01/01/94-28/02/94; 2. 01/01/94-31/01/94; 3. No data yet received for 1994; 4. 01/01/94-31/03/94; 5. 01/01/94-30/04/94 6. No SHC in Region; 7. Laboratory and SHC data 01/01/94-31/03/94.

AHS Infection		CSA[1]	SSA[2]	ESA[1]	SWS[2]	WSA[3] + WEN	NSA[4]	CCA[5]	ILL[1]	HUN[3]	NCR[1]	NER[4]	OFR[3]	CWR[6]	SWR[7]	SER[3]
Chlamydia trachomatis	Male	–	–	10	1	–	–	–	2	–	–	3	–	–	–	
	Female	1	–	12	1	–	1	1	2	–	–	10	–	–	3	
	Total	**1**	**–**	**22**	**2**	**–**	**1**	**1**	**4**	**–**	**–**	**13**	**–**	**–**	**3**	**–**
Donovanosis	Male	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Female	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Total	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**
*Genital herpes	Male	2	1	57	–	–	6	5	–	–	1	1	–	–	–	
	Female	–	3	16	–	–	3	2	–	–	1	6	–	–	–	
	Total	**2**	**4**	**73**	**–**	**–**	**9**	**7**	**–**	**–**	**2**	**7**	**–**	**–**	**–**	**–**
*Genital warts	Male	7	6	161	19	–	11	16	11	–	6	3	–	–	1	
	Female	5	6	69	9	–	4	7	4	–	2	11	–	–	1	
	Total	**12**	**12**	**230**	**28**	**–**	**15**	**23**	**15**	**–**	**8**	**14**	**–**	**–**	**2**	**–**
Nongonococcal urethritis	Male	2	1	127	12	–	4	11	5	–	6	5	–	–	1	
	Female	–	–	–	–	–	2	–	–	–	–	–	–	–	2	
	Total	**2**	**1**	**127**	**12**	**–**	**6**	**11**	**5**	**–**	**6**	**5**	**–**	**–**	**3**	**–**
Lymphogranuloma venereum	Male	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Female	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
	Total	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**	**–**