



COMMUNICABLE DISEASES INTELLIGENCE

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CONTENTS

ARTICLES

Page

World Health Organization Tuberculosis Notification Update, 1994 164

Are we running out of antibiotics?
Beryl Wild 166

Bovine Spongiform Encephalopathy in the United Kingdom 169

Meningitis in Africa - the constant challenge of epidemics 171

OVERSEAS BRIEFS 172

COMMUNICABLE DISEASES SURVEILLANCE 172

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WORLD HEALTH ORGANIZATION TUBERCULOSIS NOTIFICATION UPDATE, 1994

Based on *Weekly Epidemiological Record* 1995;71:65-69

More than three million cases of tuberculosis were notified in 1994, but the World Health Organization (WHO) estimates that over eight million cases might have occurred in the world. This under-reporting reflects missing or incomplete information or poor performance of the recording and reporting systems of some national tuberculosis control programs. Three-quarters of the notified cases have been recorded in Asia and Africa. Underlying causes are poverty, urbanisation, malnutrition, and HIV.

Tuberculosis notifications

Notifications of tuberculosis cases for 1994, as reported to WHO by January 1996, were analysed. Most data were provided by WHO Member States through the WHO Regional and Country Offices or directly from National Tuberculosis Control Programmes.

In mid-1995, the WHO Global Tuberculosis Programme sent a data collection form to all Member States and other countries and areas to obtain information on the burden of tuberculosis in 1994. These forms were returned to the Global Programme for data analysis. Additional information was obtained from reports by other organisations involved in tuberculosis control, and from published literature. Prior to finalisation of the report, the data were sent to all WHO Regional Offices for verification and updating.

Out of 214 countries and areas, a total of 141 (66%) responded to the WHO inquiry for 1994 tuberculosis notification data. Information for another 17 countries and areas was obtained from other sources. These 158 countries and areas account for 90% of the global population. As not all countries provided WHO with data for 1994, the latest reports available between 1990 and 1994 have been used to generate a global picture of the disease (Table and Figure).

Table. Tuberculosis cases notified in the world for 1994, by WHO Region¹

WHO Region	Number of cases notified	Rate (per 100,000 population)
Africa	54,1360	96.8
Americas	264,221	34.9
Eastern Mediterranean	237,937	55.2
Europe	286,608	33.3
South-East Asia	1,298,999	94.4
Western Pacific	725,014	45.5
Global	3,354,139	60.1

1. Based on latest reports available.

The data shown should be interpreted with caution for a number of reasons:

- the information for the most recent years was sometimes missing or incomplete;
- for several countries the performance of the recording and reporting system of the National Tuberculosis Programme is poor;
- case definitions vary between countries; non-standard definitions are sometimes used for pulmonary and extrapulmonary tuberculosis, and not all countries distinguish between new tuberculosis cases and relapses.

Global tuberculosis situation

Figure 2 shows the latest available case notification rates (per 100,000 population) for all countries, categorised by low (<25), medium (25-100), and high (>100) levels of notification. Over 3.3 million cases of tuberculosis have been notified. This figure constitutes under-reporting, as it has been estimated that 7.5 million cases occurred in 1990 and 8.8 million will occur in 1995¹.

Regional tuberculosis situation

In the African, Eastern Mediterranean and European Regions, notifications are on the increase. In Africa, HIV, malnutrition, urbanisation and, to some extent, improvements in surveillance, may be the underlying factors. In Europe and the Eastern Mediterranean, large population movements, social upheaval, and in some countries HIV, have resulted in the re-emergence of a

Figure. Distribution of notified tuberculosis cases for 1994, by WHO Region

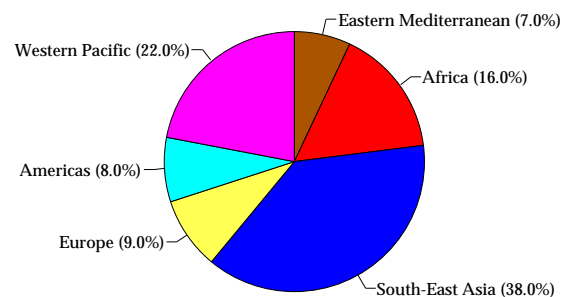
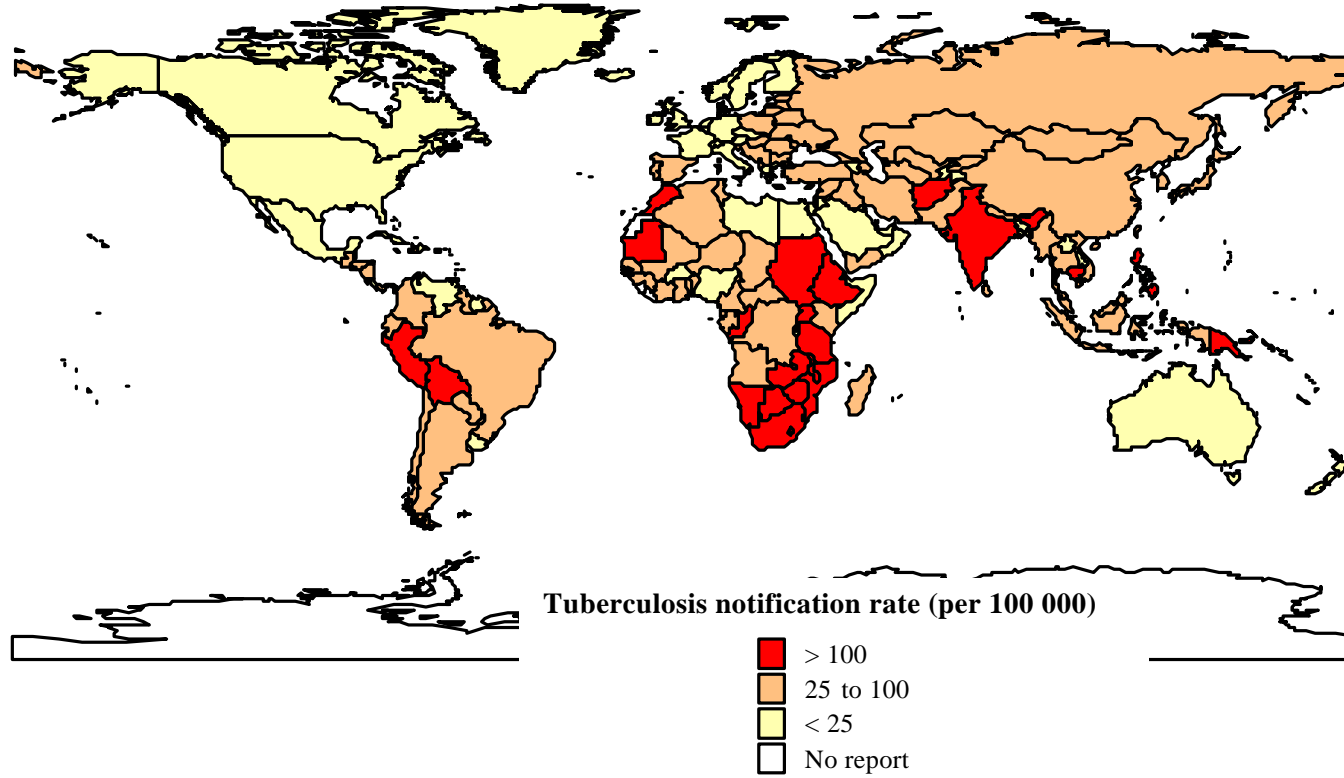


Figure 2 . Tuberculosis notification rates for 1994, by country



disease which until a few years ago was no longer considered a public health threat.

In the Regions of the Americas and the Western Pacific, case notification rates have remained relatively stable, indicating no significant improvements in tuberculosis control.

In the South-East Asia Region, a significant decrease in the case notification rates occurred during the past four years. This phenomenon is most likely an artefact, reflecting marked fluctuations in reporting activities of some large countries.

Tuberculosis Notification Update, February 1996 (ref. WHO/TUB/96.197), is available from the Programme Manager, Global Tuberculosis Programme, World Health Organization, CH-1211 Geneva 27, Switzerland, fax 41 22-791 41 99. It can also be ordered via email: raviglionem@who.ch, levym@who.ch, schmidts@who.ch.

Reference

1. Dolin PJ, Raviglione MC, Kochi A. Global tuberculosis incidence and mortality during 1990-2000. *Bull World Health Organ* 1994;72:213-220.

ARE WE RUNNING OUT OF ANTIBIOTICS?

Beryl Wild, King Edward Memorial Hospital for Women, Princess Margaret Hospital for Children, Perth, Western Australia
Adapted from *Microbiology Newsletter*, October 1995;10(2):2-5.

Resistance amongst bacteria predates antibiotic use in humans. Collections of bacteria established for taxonomic purposes before the clinical use of antibiotics contain organisms resistant to antibiotics. This is to be expected when one remembers that many soil and other bacteria excrete bactericidal substances, bacteriocins, into their environment to ensure their own survival amongst millions of competing organisms.

Since antibiotics have been in widespread clinical use, resistance has become commonplace. By 1950 infection with penicillin-resistant *Staphylococcus aureus* had become a widespread problem in hospital patients. This subsequently spread into the general community, so that now only about 10% of *S. aureus* isolates from community patients are susceptible to penicillin. Methicillin resistance is appearing in Western Australian isolates of *S. aureus*, ten years after methicillin resistance became widespread in the eastern States. Similarly, virtually all *Klebsiella* species isolated from the Western Australian population were susceptible to ampicillin in the 1950s, but are now all resistant. This resistance has spread into approximately 55% of all *Escherichia coli* and many of the other enterobacteriaceae.

Penicillin resistance is well known in *Neisseria gonorrhoeae* and has now been found in *Neisseria meningitidis* (none reported in Australia as yet). Penicillin resistance is becoming increasingly common in Australian isolates of *Streptococcus pneumoniae*, and invasive pneumococcal infections such as meningitis, which cannot be treated with penicillin, are being encountered. At present resistance to third-generation cephalosporins is rare and patients with pneumococcal meningitis should be treated with ceftriaxone or cefotaxime at least until susceptibility to penicillin has been confirmed in the laboratory.

How antibiotics work

Antibiotics in low concentrations are selectively more toxic to bacteria than they are to humans. In order to be effective, the organisms must be susceptible to an achievable concentration of the antibiotic. The most successful compounds appear to be those that interfere with the construction of the bacterial cell wall, the synthesis of protein on the bacterial ribosome or replication and transcription of bacterial DNA. Very few clinically useful agents act at the level of the cell membrane or by interfering with specific metabolic processes.

Mechanisms of antibiotic resistance

These include:

- (1) inactivation of the antibiotic before or after entering the bacterial cell. Examples include the many beta-lactamases produced by *S. aureus* and many Gram-negative enteric bacteria;
- (2) alteration of the cell surface to become less permeable to the antibiotic; and
- (3) modification of the antibacterial target structure so that it cannot bind to the antibiotic.

Bacteria achieve these apparently simple goals in a bewildering variety of ways, some (probably many) of which still await discovery. For example there are at best 44 different beta-lactamases produced by Gram-negative enteric bacteria; of these, about nine are quite common, and 27 are rare.

Bacterial resistance to a particular antibiotic may be 'constitutive', that is, no member of the species or genus is susceptible to the antibiotic in question because they lack the target required by the antibiotic; this is usually chromosomally specified.

Antibiotic resistance may also be acquired. This may be due to:

- (1) *Mutation in the bacterial chromosome* resulting in a gene product with reduced or absent ability to bind the antibiotic. Examples include high-level resistance to streptomycin in *M. tuberculosis*, penicillin-resistant *S. pneumoniae*, penicillin-resistant *N. gonorrhoeae*, and fluoroquinolone-resistant *E. coli*;
- (2) *Acquisition of resistance genes via plasmids*. Plasmids are self-replicating molecules of DNA which exist in the bacterial cell cytoplasm, and which usually produce a drug-inactivating or drug-modifying enzyme. Large plasmids often code for resistance to several antibiotics. Plasmids are most commonly transferred by conjugation between bacterial cells, and are common amongst Gram-negative enteric bacteria. In the laboratory, plasmids may also be transferred via bacteriophages, and also by transformation (uptake of naked DNA which has been released into the bacterial cell's environment), but it is not known how often this occurs in nature.
- (3) *Acquisition of resistance genes via transposons*. Transposons are discrete DNA sequences which transfer and rearrange their genetic material (encoding for resistance to a wide variety of antibiotics as well as many other metabolic properties) between bacterial chromosomes and/or plasmids. They are responsible for much of the development and spread of antibiotic resistance in hospitals. Once integrated into the host bacterium's chromosome, transposons are spread as stable genetic elements.

Transfer of resistance between disparate bacterial genera

When antibiotics are used, resistant bacteria are rapidly selected and become dominant amongst the patient's normal flora. This is particularly the case if the patient being treated is exposed to the bacterial flora of many other people, as occurs in hospital (this is fairly obvious with toddlers, but also occurs with adults.) Thus the best way of conserving antibiotics is not to use them. Unfortunately even if this were practical, once resistance to a new antibiotic has developed it can spread worldwide within two or three years.

Information on the types and modes of transfer of antibiotic resistance within and between bacterial species and genera is still expanding. Interesting examples include the identical erythromycin resistance genes found both in some *Streptococci* (Gram-positive) and also in some *Campylobacter* species (Gram-negative), and the acquisition by enterococci of aminoglycoside and beta-lactam resistance from *Staphylococci*. Although many resistance mechanisms remain unknown, clinical experience has shown that the mere use of antibiotics is a powerful selection factor for antibiotic-resistant organisms.

Responsible use of antibiotics

While we are aware of increasing antibiotic resistance in Australia and other countries with good controls on the supply of antibiotics, it is a much greater problem in countries such as Spain, where drugs can be freely purchased without a medical prescription. Thus controls must be in place to ensure that antibiotics are used only when necessary. Antibiotics are also used widely in agriculture to maximise meat and egg production, but current opinion is divided about the effect of this practice on the spread of antibiotic resistance amongst the human population. This would undoubtedly be a major influence if meat were eaten without cooking, but when it is cooked adequately (and stored separately from uncooked meat after cooking) most bacteria are killed.

What else can be done to preserve antibiotics for future use?

Advocated measures include:

Prevention of infection wherever possible. This includes improvement of living standards for people living in poor conditions, such as Australian Aborigines, and the universal use of the effective vaccines currently available.

Early and accurate clinical diagnosis. Laboratory diagnosis of the pathogen depends upon appropriate specimen collection. Adequate samples, preferably from ordinarily sterile sites, provide the best chance of identifying the true pathogen(s).

Rapid laboratory identification of antibiotic resistance is essential, and may be quite difficult for some types of bacteria.

Laboratory surveillance of antibiotic susceptibility patterns of local strains of bacteria is important to enable reliable antibiotic selection for individual cases before laboratory results are available, to document changes at both local and national levels, and to provide an early warning of cross infection problems.

Selective reporting of antibiotic susceptibility is recommended.

Early and appropriate treatment, including surgical drainage of pus and debridement of necrotic tissue is essential in order to reduce the infective load. Patients can still die despite being given appropriate antibiotics if surgery is unduly delayed.

Choice of antibiotic may vary with the type of patient, as well as the type of infection, thus

Narrow spectrum antibiotics should be given wherever possible.

Broad spectrum treatment may be required for immunocompromised patients more often than for previously healthy adults. In this context the compromised include patients at the extremes of age, pregnant women, those with chronic or severe underlying illness, as well as those with primary or acquired immunodeficiency.

Broad spectrum antibiotics may also be indicated in mixed or unknown infections rather than using combinations of drugs, or to avoid toxicity (e.g. aminoglycoside).

Antibiotic combinations may be indicated where -

- (1) the pathogen is unknown, but could be one or more of several bacteria;
- (2) infection is mixed;
- (3) antibiotic synergy is required, such as in the treatment of endocarditis;
- (4) drug resistance must be prevented, such as in mycobacterial infection;
- (5) reduced dosage of toxic agents can be achieved such as therapy of *Candida* infections with amphotericin and 5-fluorocytosine.

The site of infection is critical for it is here that adequate antibiotic levels must be achieved. Thus dosage, mode of administration, and choice of antibiotic often vary with the type and location of infection. Antibiotic levels at the site of infection must be at least equal to or greater than the minimal inhibitory concentration (MIC) of the organism. Thus intracellular organisms such as *Salmonella typhi* best respond to an antibiotic that penetrates cells (eg. ciprofloxacin).

Adjuvant therapy may affect antibiotic levels present at the site of infection: dexamethasone given for meningitis decreases penetration of vancomycin into the CSF, reducing its effectiveness in the treatment of penicillin resistant pneumococcal infection.

Host response to infection may necessitate different levels of dosage or choice of antibiotics. For example fever may recur in pneumococcal meningitis as the blood-brain barrier is restored, reducing the penetration of penicillin into the CSF; this can be corrected by doubling the dose of intravenous penicillin G.

Duration of treatment varies with the type of infection. For most conditions the optimum duration of antibiotic treatment has not been scientifically defined, often varies with host factors and therefore depends upon clinical judgement. For this reason frequent clinical review is necessary.

Antibiotics used for *surgical prophylaxis must 'cover' the usual normal flora for the operative site* and be given for the minimum time, which is usually a single dose. If surgery is prolonged a second dose may be given in order to maintain therapeutic tissue levels for up to six hours afterwards.

Infection control measures to identify and correct cross infection problems are integral to reducing spread of antibiotic resistant organisms. Apparently insignificant or unrecognised changes in hospital standards may lead to major opportunities for selection of 'new' pathogens; thus if pan-flusher sanitising equipment fails to reach adequate operating temperatures antibiotic resistant enterococci can survive and cause nosocomial infections.

Clinical pharmacists have an important role to play in identifying patients whose antibiotic regimens may require adjustment. Not only are the latest broad-spectrum antibiotics often very expensive, but if used indiscriminately and without justification, their useful life will be shortened. Automatic stop-orders and criteria for prescription of these drugs are likely to become more common in future.

Principles for conservation of antibiotics

The principles incorporated in the above guidelines were devised by Jawetz almost 50 years ago, but are often overlooked. In summary they comprise adherence to stringent policies to reserve the use of antibiotics for appropriate indications and duration of therapy, continuing nationwide and hospital surveillance for antibiotic resistance (for developing and modifying antibiotic policies as appropriate) and development of and adherence to appropriate infection control policies to reduce nosocomial spread of antibiotic-resistant organisms. These measures, together with rapid diagnostic tests to identify pathogens, have stood the test of time. They must be remembered, as today they offer us our best chance of extending the useful life of existing antibiotic agents.

Further reading: Chin G J, Marx J (eds). Resistance to Antibiotics. *Science* 1994;264:359-393.

BOVINE SPONGIFORM ENCEPHALOPATHY IN THE UNITED KINGDOM

From World Health Organization Fact Sheet 113, March 1996

Bovine Spongiform Encephalopathy (BSE) first came to the attention of the scientific community in November 1986 with the appearance in cattle of a newly-recognized form of neurological disease in the United Kingdom. Between November 1986 and May 1995 approximately 150,000 cases of this newly-recognized cattle disease were confirmed from approximately 33,500 herds of cattle in the UK. Epidemiological studies in the United Kingdom at that time suggested that the source of disease was cattle feed prepared from carcasses of cattle, and that changes in the process of preparing cattle feed introduced in 1981-1982 may have been a risk factor. Speculation as to the cause of the appearance of the disease in the food chain of cattle has ranged from spontaneous occurrence in cattle, the carcasses of which then entered the cattle food chain, to entry into the cattle food chain from the carcasses of sheep with a similar disease.

BSE is associated with a transmissible agent, the nature of which is not yet fully understood. The agent affects the brain and spinal cord of cattle and is characterised by sponge-like changes visible with an ordinary microscope. It is a highly stable agent, resisting heating to normal cooking temperatures and even higher temperatures such as those used for sterilisation, freezing, and drying. The disease is fatal for cattle within weeks to months of its onset.

By May 1995, BSE had been reported from 10 countries and areas outside the United Kingdom. In one group of countries - France, Portugal, Republic of Ireland and Switzerland - the disease occurred in native cattle, and this was thought to be in part related to importation of cattle feed from the UK. In another group - Falkland Islands (Las Malvinas), Oman Sultanate, Germany, Canada, Italy and Denmark - cases were only identified in cattle imported from the UK.

In July 1988 the UK banned the use of cattle carcasses in the preparation of cattle feed, and in 1989 the UK banned the use of brain and spinal cord - as well as tonsil, thymus, spleen and intestine - of cattle origin (known as Specified Bovine Offals or SBOs) in foods for human consumption. Cattle are continuously monitored for BSE in all affected countries, and BSE is decreasing in the UK.

BSE is one of several different forms of transmissible brain disease of animals. Others include scrapie, a disease common in sheep; a similar neurological disease in animals such as the mink, mule deer and elk; and, recently, neurological disease in household cats, the majority of which appear to have been in the United Kingdom.

Diseases in humans with sponge-like findings in brain under the microscope, and with severe and fatal neurological signs and symptoms, include kuru, a disease

which appears to be transmitted by human ritual handling of bodies and brains of the dead, and Creutzfeldt-Jakob disease (CJD). CJD occurs in a form associated with a hereditary predisposition (approximately 10% of cases), and in a more common, sporadic form that accounts for the remaining 90%. In recent years, it has been shown that CJD can be transmitted to humans by treatment with natural human growth hormone or grafting of tissues surrounding the human brain, and these means of transmission have now been controlled in the industrialised countries where these procedures were practised. Another similar human disease is Gerstmann-Straussler syndrome which appears to be familial.

After the identification of BSE, and as a regular activity to continue the study of the possible hazards of BSE for humans, the World Health Organization (WHO) held three meetings on the spongiform encephalopathies in 1991, 1993 and 1995, and one in collaboration with the Office of International Epizootics in 1994. The purpose of these meetings was to review the existing state of knowledge on spongiform encephalopathies including BSE, to evaluate possible means of transmission and to identify risk factors for infection. An express purpose of these meetings was to review the possible human public health implications of animal spongiform encephalopathies, with special emphasis on BSE.

The most recent WHO meeting compared the annual number of cases of CJD in France, Germany, Italy, Netherlands and the United Kingdom. This comparison showed that rates were similar in all these countries (approximately 1 per million), as was the age distribution and duration of illness prior to death. Cases reported from the United Kingdom were those which were identified through routine reporting and from an intensified surveillance system for CJD-like illness which had been set up in 1990.

Conclusions of this meeting were that the epidemiological evidence in Europe did not indicate a change in the incidence of CJD that could be attributed to BSE. If the measures taken in the United Kingdom regarding cattle feed and SBOs for human consumption, as well as other precautionary measures at farm, slaughter and meat processing levels were being strictly implemented, the risk of BSE transmission, and therefore of possible transmission of BSE to humans, would be minimised. The meeting recommended that WHO encourage research on BSE and its possible implications for human public health, and that WHO continue to provide guidance to countries in order to minimize the risk of transmission of BSE as described above, and of transmission of human diseases such as CJD through medical procedures.

During the past ten months, 10 humans in the United Kingdom have been identified with what appears to be a variant of CJD. The onset of the first case appears to have been as early as February 1994, and 8 of the 10 patients have died to date. These ten cases are all under the age of 42 years and some have had behavioural changes at the onset. All 10 cases have had a prolonged course of disease.

Results of patient interviews and medical history, genetic analysis and testing for other possible causes of this disease were reviewed by the United Kingdom Advisory Committee on Spongiform Encephalopathy which concluded that "although there is no direct evidence of a link on current data and in the absence of any credible alternative the most likely explanation at present is that these cases are linked to exposure to BSE before the introduction of the specified bovine offal ban in 1989". On 20 March 1996 the United Kingdom officially reported the cases and conclusions of this Committee in a press conference.

In the light of the new information on the 10 human cases of variant CJD reported by the United Kingdom on 20 March 1996, a WHO meeting of international experts in neurology, transmissible spongiform encephalopathies, epidemiology, veterinary science and public health is being organised at WHO Headquarters in Geneva on 2-3 April 1996 to review the present situation and to make further technical and public health recommendations as required. In particular, the meeting will identify those technical and scientific issues which must be addressed in developing best practices that will protect the consumer. WHO recommends that if similar disease is identified in any other countries, the national health authorities should be immediately notified. An updated fact sheet will be provided at the conclusion of the 2-3 April meeting.

Australia responds to BSE

The issue of a possible threat to public health in Australia from the importation or consumption of canned meat or other processed beef products originating in the United Kingdom is being reviewed as a matter of urgency by a high level task force established by the Federal Minister for Primary Industries and Energy, John Anderson, and the Federal Minister for Health and Family Services, Dr Michael Wooldridge.

The task force comprises the Chief Medical Advisor of the Department of Health and Family Services, the Chief Veterinary Officer from the Department of Primary Industries and Energy, and officials from the Australian Quarantine and Inspection Service and the National Food Authority.

The Federal Government has announced it will stop the importation to Australia of a small range of food products from Britain which may contain components of processed British beef and that existing stock is being from retail outlets.

Fresh and frozen beef has not been imported to Australia from Britain for a number of years nor has there been any importation of live cattle, cattle semen or cattle embryos from the United Kingdom since 1988.

Australia beef is safe to eat and there is no BSE in Australian cattle.

While the link between BSE and Creutzfeldt-Jacob Disease (CJD) has not been clearly established, and the risk of Australians contracting CJD from British beef products is almost negligible, precautionary measures are being implemented.

The Government decision was taken after considering updated advice from the BSE expert task force and consultations with key food manufacturing and retail industry groups.

A toll free telephone line is available so that members of the public can enquire about products which may be suspect and about the diseases BSE and CJD. The toll free number is 1800 02 06 13 and is open from 8.30am to 8.30pm every day.

MENINGITIS IN AFRICA - THE CONSTANT CHALLENGE OF EPIDEMICS

Based on a World Health Organization press release of 15 March 1996

The epidemics of meningococcal meningitis currently affecting several countries in Africa are not a new phenomenon, although the scale of the current outbreaks is worrying. Outbreaks and epidemics occur periodically throughout the world. In many countries of America, Asia and Europe, where the disease occurs sporadically, its frequency can increase suddenly and take the form of recurrent epidemics.

In sub-Saharan Africa since the 1980s, epidemics have become a constant concern for a considerable number of countries. The World Health Organization (WHO) estimates that epidemics of cerebrospinal meningitis are currently a public health hazard in the twenty following Member States: Benin, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Côte d'Ivoire, Ethiopia, Eritrea, Ghana, Mali, Mauritania, Niger, Nigeria, Rwanda, Senegal, Somalia, Sudan, Togo and United Republic of Tanzania. This means that there is a population of approximately 357 million Africans at risk.

The WHO Regional Office for Africa has just taken stock of the epidemiological situation in 18 high risk countries in the Region. As at 13 March 1996, using data available for 12 of those countries, the number of reported cases since January 1996 is 37,144, and the number of deaths is 5,348. The most affected countries at present are Nigeria (22,545 cases and 3,889 deaths), Burkina Faso (8,252 cases and 722 deaths), Niger (4,808 cases and 503 deaths), Mali (787 cases and 158 deaths), Benin (251 cases and 34 deaths), Chad (244 cases and 19 deaths) and the Central African Republic (152 cases and 22 deaths). In other countries, although no epidemic has been reported so far, the risk is real. The case fatality rate ranges from 7.8% to 20.1%, depending on the country.

The situation in Burkina Faso is particularly worrying, especially in five of the twenty-five affected provinces (out of a total of 30 provinces): those of Bam, Yatenga, San Matenga, Ouahigouya and Boulkiemde. In the capital, Ouagadougou, the hospital admits 100 new cases each day.

The WHO Regional Office for the Eastern Mediterranean, which is responsible for a number of countries on the African continent, has received reports of the first cases of meningitis in Sudan (28). The health authorities in that country are on the alert, as those cases have appeared earlier in the year than might have been expected.

Laboratory tests conducted in Norway on samples from Nigeria confirm that the current meningitis epidemic is serogroup A, as is usually the case in Africa.

Action in the field, coordinated by WHO in collaboration with UNICEF, Médecins Sans Frontières and other

organisations, must be aimed at bringing the situation in a country under control as quickly as possible. This can be achieved through rapid diagnosis and early treatment of cases, vaccination of the populations at risk, and by preventing propagation of the infection to neighbouring countries.

Prevention and control of epidemics of cerebrospinal meningitis are a major priority in the control of epidemic diseases. At times countries have to cope with several different types of epidemic at once. This is true at present in Nigeria which, in addition to meningitis, has a cholera epidemic in seven states, with 6,117 cumulated cases and 487 deaths since the beginning of the year. Nigeria also has epidemics of measles in eight states, with 3,462 cumulated cases and 302 deaths.

Tools and strategies exist that can reduce the devastating effects of meningitis epidemics. The best method of prevention is vaccination. There are also simple ways of treating patients, the main one being intramuscular oily chloramphenicol. Technical instructions for field staff have been developed.

Where, then, is the problem? There are three major challenges to national health services. The first is early detection of an epidemic. The second is prompt and proper treatment of cases of meningitis, to reduce the number of deaths to a minimum and limit transmission. Finally, mass vaccination of population groups at greatest risk in any zone where an epidemic is recognized will limit the number of cases.

Effective and rapid prevention and control of epidemics of cerebrospinal meningitis call for a high degree of preparedness on the part of health services, and maintenance of operational capacity to act. This means having the human resources, logistics and pharmaceutical supplies, such as vaccines and antibiotics, ready to go into action.

Sufficient stocks of serogroup A+C meningococcal vaccine must be available at all times, as well as intramuscular oily chloramphenicol. The ability of health teams to treat patients and to conduct mass vaccination campaigns at short notice must be improved.

WHO will organise wide-ranging consultations with the countries concerned and with national and international partners in order to set up a strategy and plan of action aimed at better forecasting and preparedness, at both national and intercountry levels. A broad alliance within the international community will be needed to strengthen existing mechanisms and resources so that the challenge of meningitis epidemics in sub-Saharan Africa can be met.

All WHO press releases, fact sheets and features can be obtained on Internet on the WHO home page <http://www.who.ch/>

OVERSEAS BRIEFS

In the past fortnight the following information has been provided by the World Health Organization (WHO), the Public Health Laboratory Service Communicable Diseases Surveillance Centre, London and the Scottish Centre for Infection and Environmental Health.

Meningitis in Africa

In a recent report from the WHO Office for the African Region the accumulated number of cases of meningitis had increased to 51,541 with 6,504 deaths (case fatality rate 12.6%). The increase can largely be attributed to an excess of 9,000 additional cases being reported from Nigeria. More than 4,000 of these were new cases reported in the past week.

Major increases were also reported in Burkina Faso, Mali, and Togo.

Cholera

The following countries have reported cholera cases which have occurred since the beginning of 1996: Af-

rica: Mali, Senegal, Somalia; Americas: Brazil, Colombia, Guatemala.

Rubella

England and Wales: For 1996 a total of 1,053 cases of rubella had been reported to the end of week 11¹. This compares to 107 for the same period in 1995. All regions have reported cases, and outbreaks have occurred in boarding schools, military establishments and universities.

Scotland: The outbreak of rubella continues with the four weekly total of 95 cases for weeks 5 to 8 1996 being the highest ever recorded². A total of three cases in pregnant women has been documented for 1996. Using patterns observed in recent years it is predicted that the current outbreak will generate between 600 and 2,000 laboratory confirmed cases, including between 10 and 32 infections in pregnancy.

1. *Communicable Disease Report* 1996;6:102.
2. *SCIEH Weekly Report* 1996;30:61.

COMMUNICABLE DISEASES SURVEILLANCE

National Notifiable Diseases Surveillance System, 3 to 16 March 1996

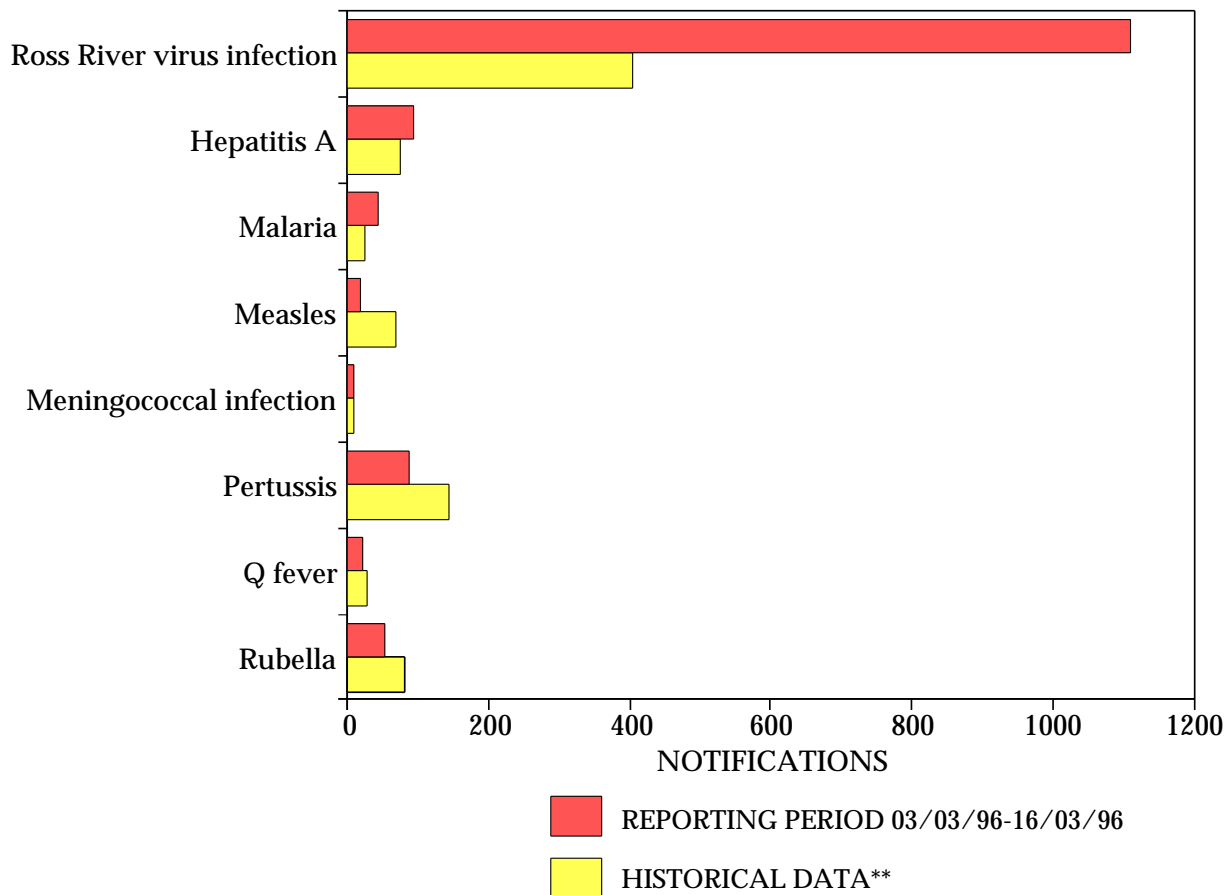
There were 2,816 notifications received for this two week period (Tables 1, 2 and 3, and Figure 1).

- There were 1,111 notifications of **Ross River virus infection**, 11% more than reported for the previous fortnight. The male:female ratio was 1.0:1.0. As for the previous three reporting periods, all age groups were affected, although 62% of cases were aged between 30 and 54 years. The greatest numbers of cases continue to be reported from Queensland (especially the southern and central coastal Statistical Divisions, and Darling Downs). In New South Wales the Northern and Richmond-Tweed Statistical Divisions are the most affected. In Western Australia the greatest numbers and rates are reported from the South West Statistical Division.
- Forty-eight cases of **Barmah Forest virus infection** were reported from Queensland and one case from South Australia. Thirty-two of the cases were in the age range 30-54 years.
- One case of **dengue** was reported from New South Wales in a young adult male.
- A case of **brucellosis** was reported from Queensland in a young adult male.
- Notifications of **campylobacteriosis** remain at a high level, 394 cases being reported in the current

fortnight. The male:female ratio was 1.3:1.0; all age groups were affected, with 21% of cases being aged less than five years.

- There were 245 notifications of **chlamydial infection** received, 44% of them being reported from Queensland. The male:female ratio was 1.0:2.0; 81% of the cases were aged between 15 and 29 years.
- One case of **cholera** was reported from New South Wales in a traveller from overseas.
- There were 140 notifications of **gonococcal infection** received; 103 cases were male and 37 cases were female; 57% of cases were aged between 15 and 29 years.
- One case of ***Haemophilus influenzae* type b infection** was reported from Victoria this period for a male.
- There were 94 cases of **hepatitis A** reported, 73% of them in males. The cases were from all but two of the five-year age groups from 0-4 years to 70-74 years; 44% of the cases were in males aged from 20 to 34 years. Two thirds of the cases were reported from the metropolitan Statistical Divisions of Sydney (29), Melbourne (19) and Brisbane (14).
- Nine cases of **hepatitis B (incident)** were reported; five were males and four were females. Their ages were from five age groups between from 10-14 years and 75-79 years.

- One case of **hydatid disease** was notified for a male from the Tasmanian Statistical Division of Mersey-Lyell.
 - Seven cases of **legionellosis** were reported. Five cases were in males and two in females. They were from age groups ranging from 45-49 years to 70-74 years. The cases were reported from five separate Statistical Divisions in four States.
 - Five cases of **leptospirosis** were reported. Their ages ranged from 22 to 47 years. All but one were males. Four cases were reported from three separate rural Statistical Divisions in Queensland. One case was reported from the Sydney Statistical Division.
 - Forty-four notifications of **malaria** were received; 30 were male and 14 were female. The ages of cases ranged from 3 years to 64 years. The cases were reported from fourteen separate Statistical Divisions in five States and Territories. Eighteen cases were reported from the Queensland Statistical Divisions of Northern and Far North.
 - Seventeen cases of **measles** were reported; nine cases were male and eight cases were female. Their ages ranged from less than one year to 42 years, seven cases being under five years of age.
 - There were 10 cases of **meningococcal infection** reported from five separate Statistical Divisions in three States. There were four males and six females. Their ages ranged from less than one year to 59 years. There was one apparent cluster of three cases reported from the same postcode area in Queensland during the current and previous two-week reporting periods.
 - There were 88 notifications of **pertussis**, a reduction on the number reported in each of the two previous fortnights; 36 cases were male and 51 cases were female, the sex of the remaining case being not reported. All age groups from 0-4 years to 75-79 years were represented. Four cases were aged less than one year, and a further six cases were less than five years of age. Eleven apparent clusters of two to eight cases each were reported from the same postcode area during the reporting period;
- the apparent clusters occurred in five separate States and Territories.
- Twenty notifications of **Q fever** were received. Eighteen cases were reported from nine separate rural Statistical Divisions in New South Wales and Queensland. One case was reported from each of the metropolitan Statistical Divisions of Melbourne and Sydney. All but one of the cases were males. All age groups but one between 5-9 years and 60-64 years were represented.
 - There were 53 cases of **rubella** reported; 33 cases were male and 20 cases were female. Recorded ages of cases were from all five-year age groups up to 50-54 years; 42% of the cases (22) were reported in males 15-24 years of age, and 23% (12) in women aged 15 to 44 years.
 - There were 200 cases of **salmonellosis** reported; 106 cases were male and 93 cases were female; the sex of the remaining case was not reported; 46% of the cases were aged less than five years.
 - Forty-three cases of **syphilis** were reported; 26 cases were male and 15 cases were female; the sex of the remaining two cases was not reported. All age groups from 15-19 years to 80-84 years were represented.
 - There were 25 cases of **tuberculosis** reported; 18 cases were male and seven were female. All age groups but one between 25-29 years and 80-84 years were represented. There was one case reported in a male child under 5 years.
 - Five cases of **typhoid** were reported; one case was male and four were female. The cases were reported from the Statistical Divisions of Canberra, Melbourne and Sydney.
 - Nine cases of **yersiniosis** were reported; Five cases were male, and four were female. Three cases were reported in children under five years of age, the remainder of the cases being aged between 10 and 54 years. One apparent cluster of two cases was reported from the same postcode area in Queensland.

Figure 1. Selected National Notifiable Diseases Surveillance System reports, and historical data¹

1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods: the corresponding periods of the last 3 years and the periods immediately preceding and following those.

Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 3 to 16 March 1996

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA ¹			
									This period 1996	This period 1995	Year to date 1996	Year to date 1995
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
<i>Haemophilus influenzae</i> b infection	0	0	0	0	0	0	1	0	1	5	16	19
Measles	2	7	1	2	0	0	4	1	17	81	117	533
Mumps	0	2	0	NN	0	0	0	0	2	4	25	27
Pertussis	0	39	2	23	18	4	0	2	88	179	646	1190
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0
Rubella	2	22	2	23	2	1	0	1	53	97	722	696
Tetanus	0	0	0	0	0	0	0	0	0	1	1	2

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

NN Not Notifiable.

Table 2. Notifications of other diseases¹ received by State and Territory health authorities in the period 3 to 16 March 1995

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA ²				
									This period 1996	This period 1995	Year to date 1996	Year to date 1995	
Arbovirus infection													
Ross River virus infection	0	127	6	755	1	-	12	210	1111	135	2991	548	
Dengue	0	1	0	0	0	-	0	0	1	3	13	6	
Barmah Forest virus infection	0	0	0	48	1	0	0	0	49	25	175	98	
NEC ^{3,4}	0	8	1	0	0	0	7	4	20	20	81	41	
Campylobacteriosis ⁵	10	-	19	74	34	18	156	83	394	473	2551	2382	
Chlamydial infection (NEC) ⁶	0	NN	25	107	0	10	66	37	245	274	1496	1394	
Donovanosis	0	NN	1	1	NN	0	0	0	2	8	15	24	
Gonococcal infection ⁷	1	28	37	33	0	0	18	23	140	116	747	651	
Hepatitis A	2	46	2	18	0	2	22	2	94	71	601	432	
Hepatitis B	5	0	0	3	0	0	1	0	9	11	57	76	
Hepatitis C incident	0	0	0	0	0	0	0	0	0	7	5	14	
Hepatitis C unspecified	9	0	0	91	0	15	0	34	149	361	1725	1920	
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	1	5	8	
Legionellosis	0	2	0	0	0	1	3	1	7	11	39	49	
Leptospirosis	0	1	0	4	0	0	0	0	5	3	50	34	
Listeriosis	0	0	0	0	0	0	0	0	0	4	12	21	
Malaria	2	10	0	28	1	0	3	0	44	14	168	116	
Meningococcal infection	0	4	0	3	0	0	0	3	10	13	59	73	
Ornithosis	0	NN	0	0	0	0	0	0	0	1	23	31	
Q fever	0	11	0	8	0	0	1	0	20	18	101	103	
Salmonellosis (NEC)	4	53	26	84	9	11	1	12	200	445	1584	1958	
Shigellosis ⁵	0	-	10	11	1	0	0	1	23	40	162	212	
Syphilis	0	21	14	6	0	1	0	1	43	79	274	419	
Tuberculosis	1	11	1	8	0	0	0	4	25	54	182	250	
Typhoid ⁸	1	3	0	0	0	0	1	0	5	3	27	24	
Yersiniosis (NEC) ⁵	0	-	0	8	1	0	0	0	9	11	70	106	

1. For HIV and AIDS, see Tables 5 and 6. For rarely notified diseases, see Table 3.
 2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
 3. Tas: includes Ross River virus and dengue.
 4. WA, NT and Vic: includes Barmah Forest virus.
 5. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
 6. WA: genital only.
 7. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
 8. NSW, Vic: includes paratyphoid.
- NN Not Notifiable.
NEC Not Elsewhere Classified.
- Elsewhere Classified.

Table 3. Notifications of rare¹ diseases received by State and Territory health authorities in the period 3 to 16 March 1996

DISEASES	Total this period	Reporting States or Territories	Year to date 1996
Botulism	0		0
Brucellosis	1	Qld	6
Chancroid	0		0
Cholera	1	NSW	3
Hydatid infection	1	Tas	9
Leprosy	0		2
Lymphogranuloma venereum	0		0
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1994.

Table 4. Australian Sentinel Practice Research Network, weeks 9 and 10, 1996

Condition	Week 9, to 3 March 1996		Week 10, to 10 March 1996	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	57	6.2	49	5.7
Rubella	3	0.3	3	0.3
Measles	0	0.0	0	0.0
Chickenpox	13	1.4	14	1.6
Pertussis	6	0.7	9	1.0
Gastroenteritis	122	13.4	119	13.8

Australian Sentinel Practice Research Network

Data for weeks 9 and 10 ending 3 and 10 March respectively are included in this issue of *CDI* (Table 4). The rate of reporting of influenza-like illness remains at a higher level than reported earlier in the year whilst that for measles remains low.

HIV and AIDS Surveillance

Methodological note

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a

combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly *Australian HIV Surveillance Report*, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 332 4648 Facsimile: (02) 332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for September 1995, as reported to December 1995, are included in this issue of *CDI* (Tables 5 and 6).

Table 5. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 30 September 1995, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
										This period 1995	This period 1994	Year to date 1995	Year to date 1994
HIV diagnoses	Female	0	1	0	1	0	0	1	0	3	1	64	58
	Male	2	36	0	10	0	0	10	3	61	78	585	643
	Sex not reported	0	0	0	0	0	0	0	0	0	1	8	9
	Total ¹	2	37	0	11	0	0	11	3	64	80	659	710
AIDS diagnoses	Female	0	0	0	0	0	0	1	0	1	5	22	29
	Male	0	17	0	7	2	0	8	2	36	88	415	618
	Total ¹	0	17	0	7	2	0	9	2	37	93	438	651
AIDS deaths	Female	0	1	0	0	0	0	0	0	1	6	28	29
	Male	0	16	0	2	2	0	6	1	27	47	418	514
	Total ¹	0	17	0	2	2	0	6	1	28	53	447	546

1. Persons whose sex was reported as transsexual are included in the totals.

Table 6. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 September 1995, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	15	543	3	94	44	4	163	69	935
	Male	163	9821	79	1529	553	70	3285	734	16234
	Sex not reported	0	2047	0	0	0	0	42	0	2089
	Total ¹	178	12418	82	1628	597	74	3498	805	19280
AIDS diagnoses	Female	4	127	0	26	17	2	47	17	240
	Male	69	3519	25	603	254	32	1265	258	6025
	Total ¹	73	3656	25	631	271	34	1319	276	6285
AIDS deaths	Female	2	94	0	19	13	2	28	9	167
	Male	49	2543	18	403	170	21	984	194	4382
	Total ¹	51	2643	18	424	183	23	1018	204	4564

1. Persons whose sex was reported as transsexual are included in the totals.

Surveillance of Serious Adverse Events Following Vaccination

The Serious Adverse Events Following Vaccination Surveillance Scheme is a national surveillance scheme which monitors the serious adverse events which occur rarely following vaccination. More details on the Scheme were published in *CDI* 1995:19;273-274.

Acceptance of a report does not imply a causal relationship between administration of the vaccine and the medical outcome, or that the report has been verified as to the accuracy of its contents.

It is estimated that 250,000 doses of vaccines are administered every month to Australian children under the age of 6 years.

Results for the reporting period 18 February to 16 March 1996

There were 15 reports of serious adverse events following vaccination for this reporting period. Reports were received from the Australian Capital Territory (2), New South Wales (5), Queensland (2), South Australia (4) and Victoria (2).

Of the 15 reports, 6 were cases of persistent screaming, 3 of hypotonic/hyporesponsive episodes, 3 of a temperature of 40.5°C or more, two of convulsions and one was another event temporally associated with vaccination (Table 7). The 'other' event was vomiting and fever following MMR vaccination.

Events associated with DTP vaccine alone or DTP in combination with other vaccines were associated with the first (8), second (2), third (2) and fourth (1) doses. Three children were hospitalised. All children had recovered at the time the initial report was sent in.

Table 7. Adverse events following vaccination for the following period

Event	Vaccines						Reporting States or Territories	Total reports for this period
	DTP	DTP/OPV	DTP/Hib	DTP/OPV/Hib	MMR	Hep B		
Persistent screaming	2	1	1	2			NSW, Qld, SA	6
Hypotonic/hyporesponsive episode	1			2			NSW, Qld, SA	3
Temperature 40.5°C or more			1	1	1		ACT, NSW, Vic	3
Convulsions		1		1			NSW, Vic	2
Other						1	ACT	1
TOTAL	3	2	2	6	1	1		15

Sterile Sites Surveillance (LabDOSS)

Data for this four weekly period have been provided by 9 laboratories. There were 402 reports of significant sepsis:

New South Wales: Prince of Wales, Sydney 35; Royal North Shore Hospital 43, John Hunter Hospital 76.

Tasmania: Royal Hobart Hospital 28; Northern Tasmania Pathology Service 8.

Northern Territory: Alice Springs Hospital 33.

Western Australia: Princess Margaret Hospital For Children 24, Sir Charles Gairdner Hospital 4.

South Australia: Institute of Medical and Veterinary Science, Adelaide 114.

Organisms reported 5 or more times from blood are detailed in Table 8. Other blood isolates not included in Table 8 were:

Gram-positive: 1 *Actinomyces naeslundii*, 1 *Aerococcus viridans*, 1 *Bacillus* species, 2 *Corynebacterium jeikeium*, 3 *Corynebacterium* species, 1 *Corynebacterium xerosis*, 2 *Enterococcus faecium*, 1 *Enterococcus* species, 1 *Listeria monocytogenes*, 1 *Streptococcus* Group C, 3 *Streptococcus* Group A, 3 *Streptococcus* Group G, 2 *Streptococcus sanguis* and 1 *Streptococcus 'milleri'*.

Gram-negative: 4 *Acinetobacter* species, 1 *Aeromonas hydrophilia*, 1 *Branhamella catarrhalis*, 2 *Campylobacter jejuni*, 2 *Citrobacter freundii*, 3 *Enterobacter* species, 1 *Gemella morbillorum*, 3 *Haemophilus influenzae*, 2 *Klebsiella oxytoca*, 1 *Klebsiella* species, 1 *Morganella morganii*, 3

Neisseria meningitidis, 3 *Proteus mirabilis*, 1 *Providencia* species, 3 *Salmonella* species, 1 *Salmonella typhi*, 1 *Serratia liquefaciens*, 3 *Serratia marcescens*, 2 *Serratia* species and 2 *Xanthomonas maltophilia*.

Anaerobes: 4 *Bacteroides fragilis*, 4 *Bacteroides* species, 4 *Bacteroides thetaiotaomicron*, 1 *Clostridium perfringens*, 1 *Clostridium* species and 3 *Propionibacterium* species.

Fungi: 2 *Candida* species

There were 159 blood isolates reported for patients over the age of 65 years (Figure 2).

There were 11 reports of meningitis and/or CSF isolates (Table 9).

Figure 2. LabDOSS reports of blood isolates, by age group

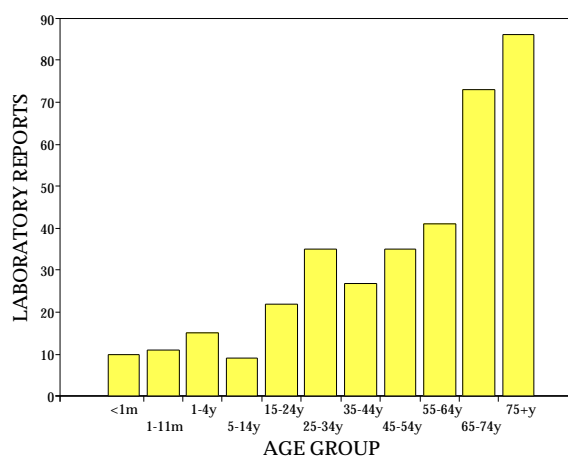


Table 8. LabDOSS reports of blood isolates, by organ-

Organism	Clinical information						Risk factors					Total ¹
	Bone/Joint	Lower respiratory	Endocarditis	Gastrointestinal	Urinary tract	Skin	Surgery	Immunosuppressed	IV line	Neonatal	Hospital acquired	
<i>Candida albicans</i>				2		1	2	3	1		2	6
<i>Enterobacter cloacae</i>							1	2			1	7
<i>Enterococcus faecalis</i>				3	1	2	1		1		2	7
<i>Escherichia coli</i>				19	32	2	2	17	1	1	7	79
<i>Klebsiella pneumoniae</i>				5	4	1	1	4	1		6	21
<i>Neisseria meningitidis</i>												6
<i>Pseudomonas aeruginosa</i>				2	1	2	2	9	1		7	19
<i>Staphylococcus aureus</i>	5	3	3	1	3	21	7	14	23	1	37	83 ²
<i>Staphylococcus coagulase negative</i>	1	1	1	2	2	2	3	6	1	3	11	25
<i>Staphylococcus epidermidis</i>	2	1		1		3		2	4	2	4	24
<i>Streptococcus</i> group B	1					1		1		2	1	6
<i>Streptococcus pneumoniae</i>		13		2	1			2			2	21
<i>Streptococcus</i> species			2	3	1	3		3			1	11

1. Only organisms with 5 or more reports are included in this table.

2. MRSA 10.

Figure 5. Adenovirus-not typed laboratory reports, 1995 to 1996, by month of specimen collection

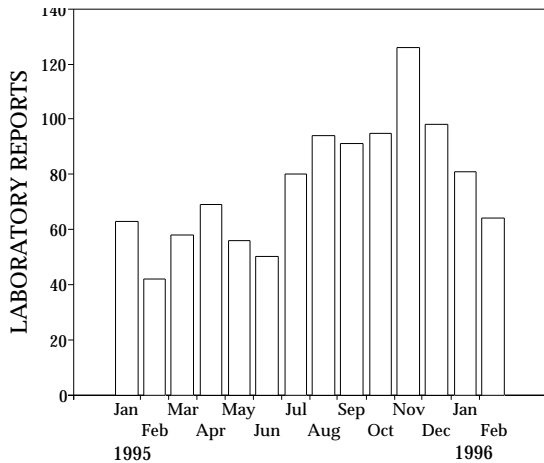
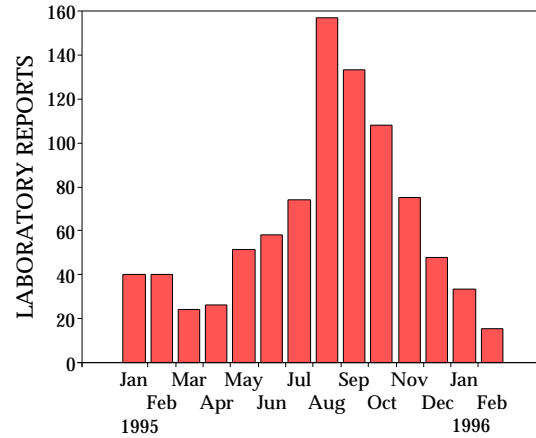


Figure 6. Parainfluenza type 3 laboratory reports, 1995 to 1996, by month of specimen collection



- **Adenovirus not typed** was reported for 78 patients this fortnight. Respiratory disease was reported for 31 patients, gastrointestinal disease 25 and eye disease 20. Diagnosis was by virus isolation (75) and antigen detection (3). Reports have continued to decline over recent months (Figure 5).
- **Parainfluenza virus type 3** was reported for 19 patients this reporting period. Diagnosis was by virus isolation (16) and single high titre (3). Reports have continued to decline since the peak in August (Figure 6). 1995 recorded the highest number of reports for any year of this scheme (Figure 7).
- **Bordetella pertussis** was reported for 10 patients this reporting period. Diagnosis was by antigen detection (6), single high titre (one), isolation (one), IgA detection (one) and method not stated (one). Included were 5 females and 5 males. Total **Bordetella** reports for February 1996 are lower than previous years (Figure 8).

Figure 7. Parainfluenza type 3 laboratory reports, 1982 to 1995, by year of specimen collection

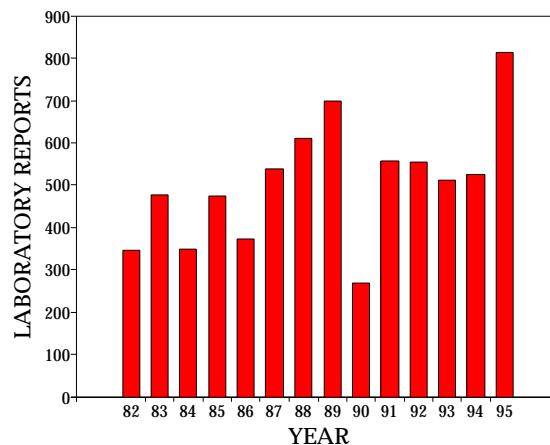


Figure 8. Bordetella pertussis and Bordetella species laboratory reports, 1994 to 1996, by month of specimen collection

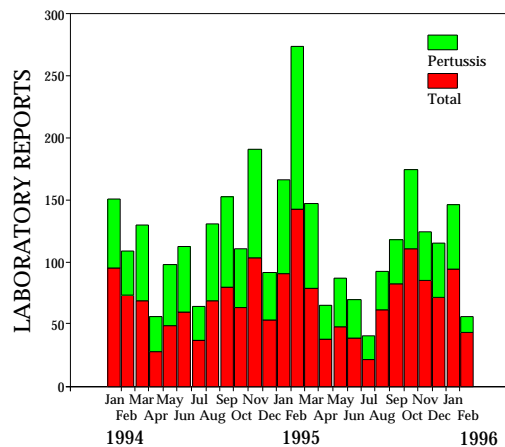


Table 10. Virology and serology laboratory reports by State or Territory¹ for the reporting period 7 to 20 March 1996, historical data², and total reports for the year

	State or Territory ¹								Total this fortnight	Historical data ²	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA											
Mumps virus			1						1	2.8	12
Rubella virus	1			12					5	17.0	203
HEPATITIS VIRUSES											
Hepatitis A virus			1	4			1	8	14	13.2	167
ARBOVIRUSES											
Ross River virus		49	11	344				146	550	94.2	1,536
Barmah Forest virus		2	3	14				5	24	11.0	89
ADENOVIRUSES											
Adenovirus type 41							2		2	0.0	2
Adenovirus not typed/pending		4		68			1	5	78	38.7	424
HERPES VIRUSES											
Herpes simplex virus type 1		8	4	161			1	47	221	179.0	1,746
Herpes simplex virus type 2		6	5	143			2	43	199	182.2	1,758
Herpes simplex not typed/pending	13								13	20.8	134
Cytomegalovirus	3	5	4	60			3	8	83	64.8	451
Varicella-zoster virus		3	2	21			1	2	29	40.0	384
Epstein-Barr virus	2	3	1	19			4	28	57	70.5	590
Herpes virus group - not typed								1	1	1.2	43
PICORNA VIRUS FAMILY											
Coxsackievirus A9							2		2	0.0	5
Echovirus type 14		1							1	0.0	23
Rhinovirus (all types)				16					16	29.5	159
Enterovirus not typed/pending	1			39				16	56	47.3	259
ORTHO/PARAMYXOVIRUSES											
Influenza A virus				1					1	7.5	60
Influenza B virus		1							1	3.7	22
Parainfluenza virus type 1				4					4	6.7	17
Parainfluenza virus type 2				1				1	2	2.0	14
Parainfluenza virus type 3	1	1		14				3	19	11.7	220
Respiratory syncytial virus		2		32				3	37	24.5	257
OTHER RNA VIRUSES											
HIV-1				1				4	5	1.0	43
Rotavirus		1							1	25.7	252
OTHER											
<i>Chlamydia trachomatis</i> not typed	3	1	20	40				25	89	102.2	912
<i>Chlamydia pneumoniae</i>				1					1	0.0	3
<i>Mycoplasma pneumoniae</i>	2		1	5			2	8	18	22.7	146
<i>Coxiella burnetii</i> (Q fever)		1						1	2	7.2	39
<i>Streptococcus</i> group A			6	6			2		14	11.7	147
<i>Bordetella pertussis</i>				2			6	2	10	43.0	123
<i>Bordetella</i> species				5					5	5.8	128
<i>Legionella longbeachae</i>								1	1	0.3	8
<i>Leptospira</i> species				1					1	1.2	11
<i>Treponema pallidum</i>			2				1	3	6	21.7	89
<i>Schistosoma</i> species								3	3	1.8	104
TOTAL	26	88	61	1014			28	368	1,585	1,112.3	10,580

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.

2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 11. Virology and serology laboratory reports by clinical information for the reporting period 7 to 20 March 1996

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
MEASLES, MUMPS, RUBELLA													
Mumps virus												1	1
Rubella virus								5				13	18
HEPATITIS VIRUSES													
Hepatitis A virus							8					6	14
ARBOVIRUSES													
Ross River virus								46		134		370	550
Barmah Forest virus								1		1		22	24
ADENOVIRUSES													
Adenovirus type 41						2							2
Adenovirus not typed/pending			1		31	23		1	20			2	78
HERPES VIRUSES													
Herpes simplex virus type 1					23			131	15		27	25	221
Herpes simplex virus type 2					1			93			67	38	199
Herpes simplex not typed/pending					1			2			7	3	13
Cytomegalovirus		1		3	22		1	1			1	54	83
Varicella-zoster virus								20				9	29
Epstein-Barr virus					14							43	57
Herpes virus group - not typed											1		1
PICORNA VIRUS FAMILY													
Coxsackievirus A9												2	2
Echovirus type 14						1							1
Rhinovirus (all types)					16								16
Enterovirus not typed/pending		2	2		25	10		3	1		1	12	56
ORTHO/PARAMYXOVIRUSES													
Influenza A virus					1								1
Influenza B virus					1								1
Parainfluenza virus type 1					4								4
Parainfluenza virus type 2					1							1	2
Parainfluenza virus type 3					14							5	19
Respiratory syncytial virus					37								37
OTHER RNA VIRUSES													
HIV-1												5	5
Rotavirus						1							1
OTHER													
<i>Chlamydia trachomatis</i> not typed									1		44	44	89
<i>Chlamydia pneumoniae</i>												1	1
<i>Mycoplasma pneumoniae</i>					12							6	18
<i>Coxiella burnetii</i> (Q fever)												2	2
<i>Streptococcus</i> group A										3		11	14
<i>Bordetella pertussis</i>					10								10
<i>Bordetella</i> species					1							4	5
<i>Legionella longbeachae</i>												1	1
<i>Leptospira</i> species												1	1
<i>Treponema pallidum</i>							1					5	6
<i>Schistosoma</i> species												3	3
TOTAL		3	3	3	214	37	10	303	37	138	148	689	1585

Table 12. Virology and serology laboratory reports by contributing laboratories for the reporting period 7 to 20 March 1996

STATE OR TERRITORY	LABORATORY	REPORTS
Australian Capital Territory	Woden Valley Hospital, Canberra	27
New South Wales	Royal North Shore Hospital, St Leonards	20
Queensland	Queensland Medical Laboratory, West End	692
	State Health Laboratory, Brisbane	415
Victoria	Monash Medical Centre, Melbourne	28
Western Australia	PathCentre Virology, Perth	187
	Western Diagnostic Pathology	216
TOTAL		1585