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VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME:

In this period (24 May to 6 June 1990) 1202 reports were processed.

There were 13 reports of Q fever (10 males, 2 females and 1 not stated). One was described as a 17-year-old meatworker from Kingaroy. Exposure details have been supplied for a 25-year-old male reported with Q fever in the last period. He was described as a grazier/stationhand from Toowoomba.

The increased activity exhibited by para influenza type 1 and respiratory syncytial virus reported in CDI 90/10 is continuing.

Echovirus type 11 was isolated from the CSF of a 16-year-old female.

Coxsackievirus B was isolated from the CSF of a 57-year-old male.

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OVERSEAS BRIEFS

1. DENGUE IN FIJI

The epidemic of dengue in Fiji continued into April. For the two weeks ending the 14 April 1990 a further 64 cases were reported. This brings the total number of cases reported for the calendar year to 1325.

2. INFLUENZA IN FIJI

The Republic of Fiji reports that for the two weeks ending the 14 April 1990 there were 3 064 cases of influenza reported. The total for the calendar year now stands at 33 671.

3. INFLUENZA IN AUSTRIA

The influenza A epidemic which started in early February waned at the end of march and early April. At the same time influenza B activity became more important and was detected in all parts of the country.

4. CHOLERA IN THE FEDERATED STATES OF MICRONESIA

The Department of Human Resources in the Federated States of Micronesia has confirmed an outbreak of cholera in Truk state. All of the 17 cases confirmed thus far (to 13 June 1990) have been from the islands situated in Moen lagoon.

All people arriving in Truk are being notified of the outbreak. They are being advised not to eat raw shellfish, to drink only boiled water and to maintain high standards of personal hygiene.

AUSTRALIAN HIV SURVEILLANCE REPORT: 18 MAY 1990

The national Centre in HIV Epidemiology and Clincal Research reports that as at 20 April 1990, a total of 1805 cases of AIDS had been reported in Australia. As mentioned in CDI 90/8, routine reports from the Centre have been condensed to two tables, as below.

For the most recent reporting period, 24 March to 20 April (weeks 13 - 16), 45 new cases of AIDS were reported in Australia.

Table 1: New cases of AIDS and deaths from AIDS for the period 24 March to 20 April (weeks 13 - 16), and cumulative cases and deaths to 20 April 1990, by sex and State in which initial diagnosis was made.

State/ Territory	1990 Weeks 13 - 16				1982 - 1990 Cumulative to 20 Apr 90			
	Cases		Deaths		Cases		Deaths	
	M	F	M	F	M	F	M	F
NSW	40	0	21	0	1133	33	716	23
VIC	2	0	0	0	375	9	185	4
QLD	1	0	1	0	125	5	74	4
WA	0	0	0	0	79	6	38	2
SA	1	0	1	0	59	2	31	1
NT	0	0	0	0	2	0	1	0
TAS	0	0	0	0	8	1	3	1
ACT	1	0	0	0	24	0	12	0
Total	45	0	23	0	1805	56	1060	35

Table 2: Notifications of persons newly diagnosed as HIV antibody positive, and cumulative since the introduction of HIV antibody testing, by sex and State/Territory of notification.

State/ Territory	1990 Weeks 13 - 16			1985 - 1990 Cumulative to 20 Apr 90			
	M	F	TOTAL	M	F	NK	TOTAL
NSW	-	-	N/A #	5,250	293	2,766	8,309 +
VIC	12	0	12	2,016	12	276	2,304
QLD	12	2	14	856	30	0	886
WA	6	0	6	489	26	0	515
SA	17 *	1 *	18 *	329	24	34	387
NT	1	0	1	7	0	43	50
TAS	0	0	0	46	2	0	48
ACT	0	0	0	7	0	97	104
Total	48	3	51	9,000	387	3,216	12,603

NK Sex not known

Notifications not available

+ Cumulative to 30 June 1989; see 23 March 1990 Report for further details

* Weeks 9 - 16

LABORATORY SAFETY GUIDELINES THAT TAKE ACCOUNT OF HIV AND OTHER BLOOD-BORNE AGENTS

AUSTRALIAN NATIONAL COUNCIL ON AIDS BULLETIN NO.3 JANUARY 1990

INTRODUCTION

These guidelines advise clinical laboratory staff of the standard safety practices to be used when handling biological specimens and reagents that may contain blood, blood products or body fluids of human origin.

The risk of laboratory acquired infection with human immunodeficiency virus (HIV) is very low.

In the most recent review of HIV infection risk to health care workers (HCW), surveillance projects in the US and UK reported that of 1676 HCW suffering needlestick, non-intact skin or mucous membrane exposure to blood from HIV positive patients, five had seroconverted.[1] In addition, fourteen HCW not in surveillance projects (and no other identifiable risk factors) had seroconverted following similar exposure to HIV positive blood.[1] Given that in the US alone, 6.8 million persons are employed in health services, the occupational risk is thus very low and certainly much less than that of hepatitis B virus (HBV) to a non-immune person.[2] Nevertheless, the consequences of HIV infection can be grave.

These guidelines embody the principle that all blood, blood products, body fluids and associated reagents are regarded as infected with HIV and/or other blood-borne pathogens, and that laboratory staff adhere rigorously to protective measures which minimise exposure to these agents.

These guidelines have been written for staff in clinical laboratories and should be read in conjuction with other documents on standard safety practices in laboratories.[2-5] Staff occupied in the propagation or research of HIV or other blood-borne pathogens are referred to other relevant guidelines.[7]

LABORATORY HAZARDS

HIV has been isolated from blood, semen, vaginal secretions, saliva, tears, breast milk, cerebrospinal fluid, amniotic fluid, and urine and is likely to be isolated from other body fluids, secretions and excretions.

<u>To date, blood is the only body fluid implicated in the transmission of HIV in the health care setting.</u>

Nevertheless, until further epidemiological studies are available, these guidelines apply to blood and all body fluids whether visibly contaminated with blood or not. HIV and other blood-borne pathogens may be transmitted in the laboratory directly by:

(a) percutaneous inoculation eg. needlestick, and
(b) contamination of non-intact skin or mucosal surfaces eg. splashing or touching eyes with contaminated hands. Environmentally mediated transmission of HIV although theoretically possible has not been documented. Aerosols (as distinct from droplets) have not as yet been shown to be a mode of transmission of HIV or HBV.

PROTECTIVE MEASURES

The procedures outlined in early laboratory safety manuals were based on the infectivity of aetiological agents.[3-8] In 1984 the Centres for Disease Control (CDC) in the US modified this approach to include procedures grouped on the transmission mode and for the first time included measures protecting against skin exposure.[5] In 1987 CDC published comprehensive guidelines which advocated procedures to protect staff whilst handling blood or body fluids.[9] The guidelines were updated further in 1988.[10] The CDC guidelines were not aetiological-agent based but were written to "universally" protect against all blood-borne pathogens. The premise that blood and body fluids from all patients are potentially infective, was termed "Universal Blood and Body Fluid Precautions". It is unfortunate that in the subsequent shortening of the phrase to "Universal Precautions", confusion has arisen in that the recommendations were extrapolated by some laboratories to include all specimens, i.e. those with faecal-oral or respiratory risk. The CDC guidelines, and these guidelines, are written specifically for protection against HIV and other blood-borne pathogens. The recommendations herein will in fact protect against transmission of faecal-oral pathogens but specimens that may potentially contain respiratory pathogens should be handled at all times with respiratory precautions documented elsewhere.[5] Many institutions will not yet have adopted a Universal Blood and Body Fluid Precaution policy for all staff. This should not preclude the laboratories of such institutions from implementing these guidelines. It is recognised that the staff of some institutions and laboratories will have difficulty with the initial acceptance of a universal policy. In these instances, a scheduled introduction of the policy may be required.

HANDWASHING

Attention to handwashing is an essential safety precaution. Washing with soap and water is recommended. Medicated soaps offer no additional benefit and may in fact lead to an increased disruption of skin integrity: the use of a moisturising handcream is recommended.

Hands must be washed:

(i) immediately they are contaminated with blood or body fluids;

(ii) before eating, drinking, smoking or leaving the laboratory to use toilet facilities;

(iii) after removing gloves or gown. If gloves are breached then they should be removed immediately and hands washed;

(iv) at completion of work-day or session.

PROTECTIVE CLOTHING

The use of protective clothing serves as a barrier to exposure.

Gloves: All staff should wear gloves when contact with blood or body fluids is envisaged. The gloves should be well fitting, disposable and readily available. Gloves should be changed if visibly contaminated or breached. Hands should be washed whenever gloves are removed. Gloves should be removed before handling telephones, performing office work, leaving the laboratory, etc.

Facial Protection: Staff should use facial protection, i.e. safety glasses, mask, or face shield, when there is a risk of splashing or spraying of blood or body fluids.

Gowns: Staff should wear gowns, preferably with a closed front, while performing laboratory tasks. Gowns should be changed immediately they are grossly contaminated. Before leaving the laboratory for non-laboratory areas, gowns should be removed and left in the laboratory. Routine laundering with render clothing non-infectious. Local regulations may require blood-stained gowns to be labelled "infectious".

Occlusive bandage: All skin defects (eg. exudative lesions, cuts, abrasions, etc.) should be covered with a waterproof dressing.

LABORATORY PRACTICES

In addition to specific precautions, staff should adhere to general principles of safe laboratory practice:

(i) Laboratory access should be restricted to authorised staff only

(ii) Eating, drinking, smoking and applying cosmetics are prohibited in the laboratory

(iii) Staff must discipline themselves to avoid hand or implement contact with their skin or mucosal surfaces (eyes, nose, mouth)

(iv) Food must be stored outside the laboratory work area. Food must not be stored in any cabinet or refrigerator containing specimens or reagents

(v) Mouth pipetting is prohibited. Manual devices must be used

(vi) **Sharps must be handled with extreme caution.** Percutaneous injury with sharps is the most common mode of blood-borne pathogen transmission in health institutions. The use of needles and syringes should be minimised. Needle recapping or removal must only be carried out using a device which eliminates the risk of percutaneous injury. Recapping, removal or manipulation of needles must never be performed with an unprotected hand. The wearing of latex gloves does not protect against needlestick injury. Other sharps, or potential sharps such as glass pasteur pipettes, should be handled with care.

(vii) Routine procedures with blood or body fluids can be performed on an open bench, however procedures that are likely to generate droplets should be eliminated or modified. In particular, the withdrawing of needles from blood collection bottles may generate sprays which can be averted by using tissues or gauze wads. Alternatively, the procedure can be carried out using a protective shield. Procedures that have a high potential for generating droplets such as blending, sonicating or vigorous mixing should be carried out with shield protection or in a safety cabinet (class 1 or 2).

(viii) Work surfaces should be decontaminated at least once a day and after any spill.

SPECIMEN TRANSPORT

Specimens for transport within institutions should be placed in a leak-proof sealable primary container. Snap-top closures should be avoided. The specimen container should then be transported in a secondary leak-proof container such as a zip-lock clear plastic bag. Request slips should be protected from contamination. The use of a double compartment clear plastic bag is recommended. The use by phlebotomists of a rack to transport blood specimens is permissible.

Personnel who transport specimens should be trained in safe handling practices and in decontamination procedures in case of a spill. Specimen reception staff should examine all specimens for leaks before opening the secondary container. Irreplacable specimens in containers that are found to be contaminated should be handled with gloves and decontaminated before being sent to the work area. If the request form is contaminated then, using gloves, it should be placed in a clear plastic bag and a photocopy taken. The copy should then be annotated and the original discarded (see section on Waste Disposal). If the specimen is able to be readily replaced the sender should be informed of the contamination and a fresh specimen procured.

Specific warning labels on specimens from patients with HIV or HBV are not recommended on the basis that all specimens are regarded and treated as infectious. Because it is often impossible to know a patient's infection status implementation of these guidelines assumes that all specimens are infectious and thus eliminates the unreliability of a warning label system.

Specimens for transport between institutions should be packed and labelled in compliance with the carrier's conditions, government and postal regulations and International Air Transport Association regulations, whichever is appropriate.[11]

DECONTAMINATION

The results of inactivation studies on HIV indicate that no changes in currently recommended sterilisation, disinfection and housekeeping strategies are required.[9] Chemicals effective against HBV are effective against HIV. Sodium hypochlorite is recommended as a safe and effective agent for surface disinfection. Some unpainted metals are corroded by sodium hypochlorite and a phenolic or iodophor may be substituted. In each case the manufacturer's instructions for the use of the disinfectant should be followed. Due to their toxicity it is not recommended that glutaraldehydes be used as surface disinfectants.

Spills:

(i) Gloves must be worn. Remove the blood or body fluid with absorbent material.

(ii) Using a detergent solution. Clean the spill site thoroughly.

(iii) Wipe down the spill site with disposable towels soaked in a disinfectant solution containing 500 mg/L (ppm) of available chlorine.

(iv) Dispose of all contaminated waste material into leak-proof bags (see section on Waste Disposal)

Note:

1. If the spill site is porous or cannot be adequately cleaned prior to disinfection then a solution containing at least 5000 parts per million (ppm) of available chlorine should be used.

2. Commercial laundry bleach (approx 5% available chlorine) is suitable and is diluted with tap water 1:10 to obtain 5000 ppm and 1:100 to obtain 500 ppm.

3. Hypochlorite solutions must be prepared daily.

Equipment: Instruments and equipment should be used in such a manner that minimises surface contamination or the production of droplets. Spillages should be immediately decontaminated. In the event of a breakage or leakage within a centrifuge, as with spills, gloves must be worn and the centrifuge decontaminated, remembering to disinfect the outside of all unbroken vials.

Equipment surfaces exposed to potential contamination should be disinfected daily. If necessary the equipment manufacturer's advice should be sought regarding compatibility of disinfectants with surfaces or functions. All instruments and equipment that require service or repair must be cleaned free of blood and disinfected before leaving the laboratory.

WASTE DISPOSAL

The disposal of laboratory waste must be in compliance with local and state regulations. Specimens containing blood, blood products or body fluids should be regarded as infectious as should all sharps. Needles should not be bent, broken or recapped before discarding. Recapping needles may reduce the risk of injury to staff disposing of waste but recapping must only be carried out with the aid of a protective device. Needles and sharps must be discarded into puncture-resistant containers which are kept close to relevant work areas. Infectious waste must be disposed of in leak-proof bags or vessels. In general, incineration of infectious waste is the preferred method of final disposal. If this is not practical then autoclaving and disposal as general waste is acceptable. Blood and body fluids may be disposed of by discard to sewer, taking care to avoid splashing.

Note: Of the 1200 HCW exposed to blood from HIV positive patients, **80% were due to needlesticks or sharps.**[1]

LABORATORY DESIGN

(i) Laboratory space and placement of equipment should not induce crowding or inhibit cleaning.

(ii) Laboratory surfaces and floors should be made of impervious material to facilitate disinfection.

(iii) Facilities for handwashing with elbow or foot operated taps should be provided in each work area. Eyewash stations should also be available in each work area.

(iv) Adequate and conveniently located biohazard containers for

disposal of contaminated materials or sharps should be provided.

(v) Adequate decontaminating containers for reusable articles should be provided.

(vi) Laboratory doors and specimen storage areas should be marked as containing a biohazard.

(vii) Offices and study areas should be separated from laboratory work areas.

STAFF SAFETY AND TRAINING

With regard to protective measures against blood exposure, the laboratory safety officer should ensure that all staff members, particulary new staff, receive adequate instruction. These instructions should ensure that the safety aspects of new practices and equipment have been considered and that standard practices are being followed. In many institutions the Infection Control Officer will be able to assist with the formulation of such protocols. It is recommended that an orientation check list is completed with each new staff member and that a copy of the laboratory's safety practices be signed by the new staff member when reading is completed. A Laboratory Safety Manual should be kept and updated at least annually. The Laboratory Safety Manual should identify specific hazards and provide detailed instructions to staff concerning the particular practices and protection measures required in all activities. Supervisors must ensure that their staff adhere to the documented safe working practices. It is recommended that the safety practices of staff be monitored at regular intervals.

Vaccination: It is recommended that all laboratory staff handling blood or body fluids receive hepatitis B vaccination and their response should be serologically confirmed one month after receiving the third dose. If the response is poor, a fourth dose should be offered and the response ascertained one month later. These results should be held by the institution's Occupational Health Unit.

Baseline Serum: It is not recommended that sera be taken routinely from new or current staff working in clinical laboratories. If specific laboratories feel their procedures or agents being handled are in a higher risk category than routine clinical laboratories, they may adopt biological safety level 3 for which the taking of baseline sera is recommended.[5] Such sera must be securely stored, bearing in mind the legal implications in compensation cases.

Accident reporting: Staff should formally report needlestick or other episodes of exposure to blood or body fluids. All such incidents should be recorded and a copy forwarded to the institution Safety Officer. Regular feedback on the incidence of accidents has been found to be very helpful for maintaining staff safety awareness.

MANAGEMENT OF BLOOD EXPOSURES

All institutions must formulate written protocols to be followed

when staff suffer blood or body fluid exposure. The protocol should include immediate first aid advice and must also deal with the issues of patient consent with regard to comatose patients.

If a laboratory worker has a parenteral or mucous-membrane exposure to blood or body fluid, the source material should be identified where possible, and tested for HIV/HBV presence.

Source material positive or unknown
If the source material is positive for HIV antibody/virus/antigen or HBV antigen, or is not available for examination, the worker should be counselled regarding the risk of infection and should be evaluated clinically and serologically for evidence of HIV and HBV infection.

HIV: Immediate therapy (within 24 hours of exposure) with azidothymidine (AZT) should be considered when the source is HIV positive. Although human clinical trials have not yet confirmed the prophylactic efficacy of AZT it is the only agent that has been shown experimentally to prevent infection in animals. The worker should be advised to report any acute febrile illness which occurs within 3 months of the exposure. An illness that is characterised by fever, rash, or lymphadenopathy and/or meningism may indicate recent HIV infection. The worker's serum should be retested for seroconversion at 3 months and if negative, again at 6 months.

HBV: If the worker responded effectively to vaccination and less than 5 years has elapsed since vaccination, then neither source nor worker need be tested further for HBV. If the worker did not respond effectively to vaccination then HBIG (0.06 ml/kg) should be given as soon as practical and repeated one month later. If the worker has never received vaccination then HBIG should be given and HBV vaccination commenced.

Source material negative

HIV: No follow up of worker is necessary unless the patient is at high risk of HIV infection and subsequently demonstrates HIV infection. In such cases the worker should be tested again at 3 months.

HBV: No investigation necessary. If worker has not been vaccinated then commence vaccination course.

REFERENCES

1. Marcus R, and the CDC Co-operative Needlestick Surveillance Group. Surveillance of health care workers exposed to blood from patients infected with the human immunodeficiency virus. N. Engl J Med 1988; 319:1118-23.

2. National Committee for Clinical Laboratory Standards. Protection of laboratory workers from infectious disease transmitted by blood and tissue; Proposed Guidelines. NCCLS Document M29-P, Villanova, Pennsylvania:NCCLS; 1987.

3. WHO. Laboratory Biosafety Manual. 1983, Geneva.

4. Code of Practice for the Prevention of Infection in Clinical Laboratories and Postmortem Rooms. DHSS, 1987. London, Her Majesty's Stationery Office.

5. U.S. DHHS: PHS. CDC and NIH. Biosafety in Microbiological and Biomedical Laboratories. 1984. U.S. Gov. Printing Office, Washington, D.C. 20402 USA.

6. CDC. Agent Summary Statement for Human Immunodeficiency Viruses (HIVs) Including HTLV- 111, LAV, HIV- 1, and HIV-2. MMWR 1988; 37 (Suppl S-4): 15-17s.

7. CDC. Occupationally Acquired Human Immunodeficiency Virus Infections in Laboratories Producing Virus Concentrates in Large Quantities. MMWR 1988;37 (Suppl.S4): 19S-22S.

8. CDC, Office of Biosafety, 1974. Classification of Etiological Agents on the basis of Hazards, 4th Ed. US Dept. of Health, Education and Welfare, Public Health Service .

9. CDC. Recommendation for prevention of HIV transmission in health-care settings. MMWR 1987; 36(Supp 2S).

10. CDC. Update: Universal Precautions for Prevention of Transmission of Human Immunodeficiency Virus, Hepatitis B Virus and Other Bloodborne pathogens in Health-Care Settings. MMWR 1988; 37: No 24, 377-388.

11. Guidelines for transportation of Specimens as Regular Commercial Freight 1987. National Pathology Accreditation Advisory Council publication, P.O. Box 658, Woden ACT 2606.

References 5, 6, and 7 have now been combined unaltered in a more recent publication (1988) of the same title as reference [5]. The separate references have been quoted because they are more readily available.

Enquiries regarding the Bulletin to Secretary, Australian National Council on AIDS, GPO Box 9848, Canberra ACT 2601, telephone (062) 89 7767.

A "FOODBORN" INVESTIGATION OF AN APPARENT FOOD POISONING OUTBREAK

(Contributed by Dr A S Cameron, A J Turner, I J Baldwin [South Australian Health Commission], M G Menadue, J T Rooney [City of Tea Tree Gully] and J Darzanos [City of Salisbury]).

INTRODUCTION

The following case report illustrates the use of the computer program "Foodborn" (1) during the investigation and analysis of an apparent food poisoning outbreak.

The index case was brought to the attention of the South Australian Health Commission (SAHC) when a woman telephoned to report that she had suffered an episode of vomiting and diarrhoea. She attributed this to a meal consumed at a local hotel two days prior to becoming ill. She had been one of a party of 16 persons at an engagement party and was aware of 6 others who were similarly ill.

METHODS

The relevant local council authority was notified of this initial report and was requested to contact the hotel and ascertain the total number of patrons who attended functions on the Saturday night. This would help with further case finding.

Hotel staff detailled a list of over 70 different foods offered to patrons in a smorgasbord format.

Using the menu provided by the hotel, a questionnaire was drafted to standardise the collection of data on the symptomatology and food consumption of patrons, both well and ill. The questionnaire was compiled using "Foodborn" for administration by SAHC epidemiologists and local authority environmental health officers. Guests identified by respective party organisers were contacted by

telephone and the information on food consumption and symptoms was entered into "Foodborn". Statistical analysis of these data, classified by food eaten and whether the consumer became ill, was by the chi-square test or Fisher's exact test where a cell contained an expected value less than 5.

Twelve residual food samples and 6 environmental swabs were collected from the hotel kitchen. Faecal specimens were also collected from 6 hotel staff members and 14 symptomatic guests. All samples were processed by the Institute of Medical and Veterinary Sciences.

RESULTS

Of the 177 or so persons possibly exposed, 84 were interviewed and 40 were determined to be cases (an attack rate of 48%). The median incubation period was 37 hours and the range of symptoms was:

Symptom	%	Symptom	%
Vomiting	74%	Fever/Temp	56%
Abdominal pain	82%	Back/Joint pain	46%
Diarrhoea	77%	Dizziness	41%
Headache	62%	Other	33%

Follow-up Of subsequent complaints to the council and the hotel indicated that persons who had eaten at the hotel on the following Sunday and Monday nights were also affected.

The analysis of the food histories using "Foodborn" showed that consumption of 30 foods was associated with attack rates of over 50%. Comparison of the attack rates between those who were ill and those not revealed only 2 food items with attack rates at least twice as high in persons that consumed those foods. Only one of these differences as significant at the 10% level, this being the potato salad (Chi-square=26; 0.000001). Bacteriological assays on the 12 food samples collected, which included the incriminated salad, were negative as were those of the kitchen environment swabs. Likewise no bacterial or viral pathogens were detected in the 20 faecal specimens.

Most of the implicated food had been prepared by two kitchen hands on the Saturday, with some having been prepared on the Friday along with other salads. It is likely that some of the batch was carried over for serving on the subsequent two nights, thus explaining the prolongation of the outbreak. The ingredients seemed beyond reproach, being freshly cooked diced potato, commercial mayonnaise from a batch widely distributed in Australia and fresh capsicum and parsley. The only illness reported in kitchen staff was diarrhoea in one person, not a salad cook, three weeks prior to this event.

DISCUSSION

The small number of cases spontaneously reported to health authorities in this outbreak of over 40 sick persons is typical. This reflects the stoicism of sufferers of "gastro" and the dilemma facing an attending doctor who is not to know that the case was infected by a food consumed two days or more previously. It also makes a nonsense of food poisoning statistics which may have some qualitative value but little quantitative validity.

The importance of collecting food histories from those exposed to an implicated meal, but who remained well, is once again emphasised, because it facilitates distinguishing foods that are associated with illness, from those that are frequently eaten by well and sick like.

The lack of an aetiological diagnosis, despite two cases being admitted to hospital and nine who consulted doctors, is not an uncommon circumstance. A recent review of foodborne outbreaks in England and Wales (2) noted that 30% of such episodes had no causal agent determined. The illness as described is consistent with a viral aetiology and this would explain the lack of apparent pathogens in faeces and food alike. A Norwalk-like virus, one of group of small round structured viruses (SRSV), is the most likely pathogen in this outbreak. The usual incubation prior for SRSV is 24 - 48 hours and the mean duration of illness is 12 - 60 hours (3). The symptoms reported in SRSV infections are comparable with those exhibited by cases in this outbreak. The Institute of Medical and Veterinary Science does not hold a stock of antigens of candidate viruses so no serological investigations have been performed.

The cryptic nature of these food-related episodes of illness means that the true costs of lapses in food hygiene are rarely calculated. An estimated cost of $10,000 has been calculated on the basis of two days hospitalization, 9 medical consultations, 20 hours of environmental health officers time and expenditure on microbiology tests. Further costs would include work time lost by a number of those persons who were ill and medical expenses incurred by those not interviewed.

CONCLUSION

Whilst the source of infection was unable to be proven by aetiological analysis, "Foodborn" provided an important tool to aid data collection and statistical appraisal of the epidemiological data. Potato salad was implicated as the most probable vehicle for infection.

The introduction of the viral agent to the potato salad may be explained by faecal or droplet contamination by the food handler whilst preparing and hand mixing the ingredients of the salad in bulk. The manner in which the ingredients of the potato salad were mixed could have resulted in non-uniform contamination and this, along with some pre-existing immunity would explain why not all clients who consumed the food were ill.

The authors emphasise that routine inspections of mass catering premises by dedicated environmental heath officers, willing to educate as well as prosecute, are probably society's only protection from more frequent unnecessary costs and loss of wellbeing.

REFERENCES

1. "Foodborn" - Epistat Services, 1785 Gattis School Road, Round Rock, Texas.

2. "Foodborne Disease Surveillance in England and Wales" - PHLS Communicable Disease Report Weekly Edition 90/15 (ISBN 0144-3186).

3. "Viral Agents of Gastroenteritis" - CDC MMWR April 199O Vol. 39/ No.RR-5.

TEN YEARS OF MRSA IN A MAJOR TEACHING HOSPITAL

(Contributed by J L Faoagali, Microbiologist; M P Nolan Technical Supervisor; M L Thong, Microbiologist; D Grant, Infection Control Nurse. Royal Brisbane Hospital)

INTRODUCTION

Methicillin resistant *Staphylococcus aureus* (MRSA) was firstdetected in 1961 shortly after methicillin became available for therapeutic use (1,2).

Prior to the availability of methicillin, *S. aureus* speciesexisted that were resistant to all the available oral and systemic antibiotics. This multi-resistant beta-lactamase producing staphylococcus had been responsible for many outbreaks of nosocomially acquired infections and was known as the "H" (hospital) strain or original "Golden Staph". Methicillin provided a useful adjunctive antibiotic therapy for infections with these organisms.

The MRSA strains detected in the 1960s (the "classic" strains) were different to the MRSA strains found in Australia and subsequently world-wide over the past 10 years (the "Eastern Australia" strains). The "classic" strains were only resistant to beta-lactam antibiotics and "normal" infection control techniques were able to contain these organisms.

The "Eastern Australia" strains were resistant not only to the beta-lactam antibiotics but also to erythromycin, trimethoprim/sulphamethoxazole and all the aminoglycosides. Some strains were also resistant to fusidic acid and rifampicin (3). Strains reputedly sensitive in vitro to cephalosporins and chloramphenicol have shown contradictory results to therapy with these drugs when tested in human infection and animal models (4-8). To date there have been no confirmed isolates resistant to vancomycin.

Significant clusters of MRSA infections did not emerge in Australian hospitals until the mid to late 1970s, nearly a decade after the problem became serious in Great Britain and other European countries, but around the same time as in the United States. Within 20 years of the introduction of methicillin, outbreaks of MRSA had occurred in all the major hospitals in Eastern Australia (9).

There is no good evidence to support either of the contentions that MRSA are less pathogenic or more pathogenic than non-MRSA. Patients colonized with MRSA pre-operatively are more likely to have MRSA isolated from post operative wound infections than patients not known to be colonized (unpublished studies from Royal Brisbane Hospital RBH). It is speculated that this is due to selection by perioperative antibiotic prophylaxis and there is a trend toward using anti-MRSA antibiotics in the perioperative prophylactic regime. However, there is no evidence to date that this reduces the post-operative wound colonization/infection rates.

STUDY

MRSA were first detected at RBH in 1975 and the number of new patients colonized/infected each year from 1975-1989 is shown in Table 1. The proportion of samples positive for MRSA peaked in 1987 and has gradually declined to 1984 levels (1.5%) in 1989.

Table 1: The number of new patients colonized or infected with MRSA at the Royal Brisbane Hospital, 1975-1989

Year	'New MRSA' Patients	Total Public Hospital Admissions	%Total
1975	1	NA*	-
1976	2	NA	-
1977	0	NA	-
1978	2	NA	-
1979	11	40 131	-
1980	30	41 465	-
1981	61	41 113	-
1982	447	42 289	1.0
1983	754	41 571	1.8
1984	595	38 785	1.5
1985	596	37 593	1.6
1986	602	38 450	1.6
1987	811	37 969	2.1
1988	711	41 594	1.7
1989	720	48 818	1.5

* NA - Not Available

Pathogenicity can possibly be inferred from blood culture isolates. Table 2 shows the number of blood cultures from which *S aureus* and MRSA have been isolated at the RBH between 1979 and 1989. The percentage of these isolates that were MRSA is also shown. The greatest proportion of isolates were in 1983 and 1984.

The total number of specimens screened for MRSA, for each year between 1981 and 1989, is shown in Table 3. Although screening swabs for MRSA have decreased since 1987 (Table 3) the number of new positive patients detected has increased (Table 2).

Table 2: *Staphylococcus aureus* and MRSA isolated from blood cultures at the Royal Brisbane Hospital, 1979 - 1989

Year	Total No. of Patients with *S. aureus* Bacteraemia	Patients with MRSA Bacteraemia	%MRSA
1979	40	0	0
1980	54	2	4
1981	58	6	10
1982	78	14	18
1983	93	32	34
1984	81	30	37
1985	70	17	24
1986	86	12	14
1987	72	17	24
1988	121	36	30
1989	138	39	28

Table 3: The total number of specimens screened for MRSA at the Royal Brisbane Hospital, 1981 - 1989, including the contribution due to screening swabs

Year	Total Number	Screening Swabs Number	Screening Swabs %
1981	1 427	893	63
1982	17 727	14 808	84
1983	29 563	NA	-
1984	27 926	NA	-
1985	31 476	NA	-
1986	37 737*	21 000	56
1987	41 915*	25 000	60
1988	34 263*	18 000	53
1989	33 898*	15 000	44

* includes specimens from the Royal Women's Hospital and the Royal Children's Hospital.

The number of MRSA isolated from 'routine' clinical swabs versus screening specimens has remained almost static between 1982 and 1989 (Table 4).

Table 4: New MRSA specimens v total specimens

	1982		1989	
Routine Clinical Specimens		271 (.43%)		448 (.40%)
Total Clinical Specimens Processed (excluding MRSA screening)	63 640		111 028	
Screening swabs positive		176 (1.2%)		272 (1.8%)
Total screening swabs	14 808		15 000	

The changing antibiotic sensitivity pattern of MRSA isolates between 1982 and 1989 is shown in Table 5. Sensitivity testing was performed using a standardized plate dilution technique (15). The pattern of antibiotic resistance and the increasing frequency of antibiotic resistant to fusidic acid and rifampicin has resulted in fewer therapeutic options available for clinicians treating patients infected with these organisms. The Australia-wide sensitivity patterns of MRSA have recently been published (12) and confirm the need for continued surveillance of this organism.

Table 5: Antibiotic sensitivities of RBH MRSA

		1982 n = 720		1989 n = 447	
ANTIBIOTIC	MIC	% S	% R	% S	% R
Penicillin	<1 mg/L	0	100	0	100
Methicilln	<4 mg/L	0	100	0	100
Erythromycin	<4 mg/L	0	100	0	100
Trimethoprim	<1 mg/L	0	100	0	100
Chloramphenicol	<8 mg/L	NA	NA	81.8	18.2
Gentamicin	<4 mg/L	9	91	8.2	91.8
Rifampicin	<1 mg/L	100	0	74	26
Fusidic acid	<1 mg/L	100	0	77	23
Vancomycin	<4 mg/L	100	0	100	0

CONTROL

There are many published papers reporting success in eliminating MRSA from specific areas and hospitals and these have been summarized by Brumfitt and Hamilton-Miller (10). Of the control methods described in the 25 papers they referred to, all included surveillance measures, isolation procedures and management of the carrier state.

At the RBH a five-pronged strategy was adopted in 1981 in an atttempt to control the spread of this organism:

1. Isolation of infected and colonized patients
2. Segregation of susceptible patients
3. Surveillance
4. Education
5. Control of antibiotic usage

Surveillance is essential to provide information on the extent and type of microorganisms in the environment, being carried by staff and colonizing or infecting patients. Only by careful surveillance can the extent of the problem be determined. Surveillance has formed a central focus for understanding and monitoring MRSA at the RBH. It involves routine patient and some staff screening, flagging of colonized/infected patients and physical separation of colonized/infected patients from non-colonized/non-infected patients. Table 3 indicates the numbers of surveillance swabs processed between 1981 and 1989.

Surveillance is used to provide information on who needs to be isolated. Most of the reported studies attest to the efficacy of strict surveillance and isolation policies (11). Centres with inadequate isolation facilities cannot prevent or contain outbreaks. MRSA is now endemic in the RBH. This endemicity has had to be accepted, except in a few specialised units where strict isolation procedures can be implemented whereby patients found or known to be colonized with this organism can be quickly moved to alternative areas.

Routine isolation i.e. use of a single room off a ward has not proved useful in containing MRSA. Strict isolation and separation of positive patients from negative patients is essential to control spread. Cohorting of positive patients and staff has proved to be a useful strategy.

Failure or inability to isolate patients colonized or infected with MRSA results in rapid spread of the organism amongst other patients in the ward, and the larger the number of colonized patients the greater the risk of invasive disease with the organism.

It is worthwhile attempting to prevent the establishment of MRSA. Many studies attest to the difficulty and expense of eradication of this organism once it has become established (9, 10, 11).

There is no widely accepted code of practice for the management of the carrier state. Disinfectants including chlorhexidine, hexachlorophene, povidone iodine and triclosan, topical antibiotics such as bacitracin, tetracycline, neomycin, and systemic antibiotics used singly and in combination have all been

used in an attempt to eliminate nasal or skin carriage. A newly available product, mupirocin, has been the subject of many published reports of successful eradication of the MRSA carrier state, but recent reports of the emergence of mupirocin resistance indicates this product is not the whole answer to the eradication of MRSA (10).

MRSA have had an important impact on patient morbidity and hospital practice but probably the major impact has been on infection control practices. MRSA are easily detected organisms which can provide a marker of the efficacy of the infection control practices used by a hospital (13,14).

MRSA is a costly organism to control. The costs of surveillance and the need to cohort and isolate patients all add to routine hospital costs. The shift of perioperative antibiotic prophylaxis from a simple (and cheap) beta-lactam or aminoglycoside antibiotic to vancomycin multiplies by 100 the antibiotic costs. The morbidity which follows infections with this organism adds to the length of the hospital stay and contributes toward the need for repeated surgery and long term antibiotic therapy.

The presence of MRSA in the hospital setting should not be ignored. Long term surveillance at RBH has shown that once a patient becomes colonized with MRSA this organism can be detected for up to 8 years. Initial attempts to eradicate the carriage of MRSA indicated that it was possible to lower the numbers of organisms to undetectable levels. Unfortunately subsequent antibiotic treatment, particularly with beta-lactam antibiotics always resulted in re-expression of MRSA within a few days of the commencement of the antibiotic therapy. It has not been possible to determine whether these patients are recolonized or demonstrating the selection of MRSA from their sensitive normal flora. It is hoped that in the future a number of these strains can be examined in an attempt to determine their origin and source.

CONCLUSIONS

Hospitals have probably been responsible for the selection and spread of methicillin resistant *S aureus* due to their failure to control the nosocomial acquisition of this organism by susceptible patients and possibly by the (over) use of beta-lactam and aminoglycoside antibiotics.

It is possible that these organisms are on the wane after ten years of activity and if so this is the pattern of many epidemic microorganisms throughout the years.

The antibiotic resistance patterns and numbers of MRSA isolated confirm the importance of this organism and the potential therapeutic problems posed by isolates capable of causing serious infections.

Continuing vigilance is essential to minimise their impact on patient morbidity and hospital patient care, antibiotic usage and laboratory support services budgets.

REFERENCES

1. Jevons MP. "Celbenin"-resistant staphylococci (Letter). Br Med J. 1961; 1:124-5.

2. Knox R. "Celbenin"-resistant staphylococci (Letter). Br Med J. 1961: 1:126.

3. Methicillin resistant S.aureus. Med J Aust. 1982; 1: (entire issue).

4. Frongillo RF, Bianchi P, Moretti A, Pasticci MB, Ripa S, Pauluzzi S. Cross resistance between methicillin and cephalosporins for staphylococci: A general assumption not true for cefamandole. Antimicrob Ag Chemoth. 1984; 25:666-8.

5. Traub WH, Spohr M, Bauer D. Gentamicin and methicillin resistant clinical isolates or *S. aureus*; comparative in vitro and in vivo efficacy of alternative antimicrobial drugs. Chemother. 1984; 30:102-12.

6. Hirschl A, Stanek G, Rotter M. Effectiveness of cefamandole against methicillin resistant strains of Staphylococcus aureus in vitro and in experimental infections. J Antimicrob Chemo. 1984; 13:429-35.

7. Cafferkey MT, Hone R, Keane CT. Antimicrobial Chemotherapy of septicaemia due to methicillin resistant Staphylococcus aureus. Antimicrob Ag Chemo. 1985; 28:819-23.

8. Klastersky J, Hensgens C, Daneau D. Therapy of staphylococcal infections: (A comparative study of cephaloridine and gentamicin). Am J Med Sci. 1975; 269:201-7.

9. Pavillard R, Harvey K, Douglas D, Hewstone A, Andrew J, Collopy B, Ashe V, Carson P, Davidson A, Gilbert G, Spicer J, Tosolini G. Epidemic of hospital acquired infection due to methicillin-resistant Staphylococcus aureus in major Victorian hospitals. Med J Aust. 1982; 1:451-4.

10. Brumfitt W, Hamilton-Miller J. Methicillin resistant Staphylococcus aureus. N Engl J Med. 1989; 320 (18):1188-1196.

11. Cafferkey MT, Hone R, Coleman D. Methicillin Resistant Staphylococcus aureus in Dublin 1971 - 74. Lancet. 1985; 2:705-8.

12. Turnidge J, Lawson P, Munro R, Benn R. A National Survey of Antimicrobial Resistance in Staphylococcus aureus in Australian Teaching Hospitals. Med J Aust. 1989; 150: 65- 72.

13. Turnidge J. MRSA - Is Control Worthwhile? M.J. Aust 1990; 152:225-226.

14. Brady LM, Thomson M, Palmer MA, Harkness JL. Successful Control of Endemic MRSA in a Cardiothoracic Surgical Unit. Med J Aust. 1990; 152: 240-245.

15. National Committee for Clinical Laboratory Standards. Methods for Dilution Antimicrobial Susceptibility Tests for Bacteria that Grow Aerobically; Approved Standard. NCCLS Publication

M7-A Villanova, Pa.: NCCLS 1985.

INFLUENZA PREVENTION AND CONTROL, USA

(Based on Recommendations of the Immunisation Practices Advisory Committee, CDC. MMWR 1990:39 no. RR-7)

INTRODUCTION

Influenza A viruses are classified into subtypes on the basis of two surface antigens: haemagglutinin (H) and neuraminidase (N). Three subtypes of haemagglutinin (H1, H2, H3) and two subtypes of neuraminidase (N1, N2) are recognised among influenza A viruses that have caused widespread human disease. Immunity to these antigens, especially to the haemagglutinin, reduces the likelihood of infection and lessens the severity of disease if infection occurs. Infection with a virus of one subtype confers little or no protection against viruses of other subtypes. Furthermore, over time, antigenic variation (antigenic drift) within a subtype may be so marked that infection or vaccination with one strain may not induce immunity to distantly related strains of the same subtype. Although influenza B viruses have shown more antigenic stability than influenza A viruses, antigenic variation does occur. For these reasons, major epidemics of respiratory disease caused by new variants of influenza continue to occur. The antigenic characteristics of strains currently circulating provide the basis for selecting virus strains to include in each year's vaccine.

Typical influenza illness is characterised by abrupt onset of fever, myalgia, sore throat, and nonproductive cough. Unlike other common respiratory infections, influenza can cause severe malaise lasting several days. More severe illness can result if primary influenza pneumonia or secondary bacterial pneumonia

occur. During influenza epidemics, high attack rates of acute illness result in increased numbers of visits to general practitioners, outpatient clinics, and emergency rooms and increased hospitalisations for management of lower-respiratory-tract complications.

Elderly persons and persons with underlying health problems are at increased risk for complications of influenza infection. If infected, such high-risk persons are more likely than the general population to require hospitalisation. During major epidemics, hospitalisation rates for high-risk adults may increase two- to fivefold, depending on the age group. Previously healthy children and younger adults may also require hospitalisation for influenza-related complications, but the relative increase in their hospitalisation rates is less than for persons who belong to high-risk groups.

An increase in mortality further indicates the impact of influenza epidemics. Increased mortality results not only from influenza and pneumonia but also from cardiopulmonary and other chronic diseases that can be exacerbated by influenza infection. At least 10,000 excess deaths have been documented in each of 19 different U.S. epidemics in the period 1957-1986; >40,000 excess deaths occurred in each of three of these epidemics. Approximately 80%-90% of the excess deaths attributed to pneumonia and influenza were among persons 65 years of age and over.

Because the proportion of elderly persons in the U.S. population is increasing and because age and its associated chronic diseases are risk factors for severe influenza illness, the toll from influenza can be expected to increase unless control measures are used more vigorously. The number of younger persons at increased risk for influenza-related complications is also increasing for various reasons, such as the success of neonatal intensive care units, better management of diseases such as cystic fibrosis and acquired immunodeficiency syndrome (AIDS), and better survival rates for organ-transplant recipients.

OPTIONS FOR THE CONTROL OF INFLUENZA

Two measures available in the United States that can reduce the impact of influenza are immunoprophylaxis with inactivated (killed-virus) vaccine and chemo-prophylaxis or therapy with an influenza-specific antiviral drug (e.g., amantadine). Vaccination of high-risk persons each year before the influenza season is currently the most effective measure for reducing the impact of influenza. Vaccination can be highly cost-effective when

a) it is directed at persons who are most likely to experience complications or who are at increased risk for exposure, and
b) it is administered to high-risk persons during hospitalisation or a routine health-care visit before the influenza season, thus making special visits to general practitioners or clinics unnecessary.

Recent reports indicate that when vaccine and epidemic strains of virus are well matched achieving high vaccination rates among closed populations can reduce the risk of outbreaks by inducing herd immunity.

Other indications for vaccination include the strong desire of any person to avoid influenza infection, reduce the severity of disease, or reduce the chance of transmitting influenza to high-risk persons with whom the individual has frequent contact.

The antiviral agent available for use at this time (amantadine hydrochloride) is effective only against influenza A and, for maximum effectiveness as prophylaxis, must be used throughout the period of risk. When used as either prophylaxis or therapy, the potential effectiveness of amantadine must be balanced against potential side effects.

Chemoprophylaxis is not a substitute for vaccination. Recommendations for chemoprophylaxis are provided primarily to help health-care providers make decisions regarding persons who are at greatest risk of severe illness and complications if infected with an influenza A virus. Use of amantadine may be considered:

a) as a control measure when influenza A outbreaks occur in institutions housing high-risk persons, both for treatment of ill individuals and as prophylaxis for others;
b) as short-term prophylaxis after late vaccination of high-risk individuals (i.e., when influenza A infections are already occurring in the community) during the period when immunity is developing in response to vaccination;
c) as seasonal prophylaxis for individuals for whom vaccination is contraindicated;
d) as seasonal prophylaxis for immunocompromised individuals who may not produce protective levels of antibody in response to vaccination; and
e) as prophylaxis for unvaccinated health-care workers and household contacts who care for high-risk individuals either for the duration of influenza activity in the community or until immunity develops after vaccination.

Amantadine is also approved for use by any person who wishes to reduce his or her chances of becoming ill with influenza A.

INACTIVATED VACCINE FOR INFLUENZA A AND B

Influenza vaccine is made from highly purified, egg-grown viruses that have been rendered noninfectious (inactivated). Therefore, the vaccine cannot cause influenza. Each year's influenza vaccine contains three virus strains (usually two type A and one type B) representing influenza viruses believed likely to circulate in the United States in the upcoming winter. The composition of the vaccine is such that it rarely causes systemic or febrile reactions. Whole-virus, subvirion, and purified-surface-antigen preparations are available. To minimise febrile reactions, only subvirion or purified- surface-antigen preparations should be used for children; any of the preparations may be used for adults. Most vaccinated children and young adults develop high postvaccination haemagglutination-inhibition antibody titers that are protective against infection by strains similar to those in the vaccine or the related variants that may emerge during outbreak periods. Elderly persons and persons with certain chronic diseases may develop lower postvaccination antibody titers than healthy young adults, and thus may remain susceptible to influenza upper-respiratory-tract infection. Nevertheless, even

if such persons develop influenza illness, the vaccine has been shown to be effective in preventing lower-respiratory-tract involvement or other complications, thereby reducing the risk of hospitalisation and death.

CDI Editorial Comment

As already reported (CDI 89/21) the Australian influenza vaccine composition for the 1990 winter season is:

- A/Victoria/36/88 (H1N1)-like strain, 15 micrograms haemagglutinin;
- A/Shanghai/11/87 (H3N2)-like strain, 15 micrograms haemagglutinin;
- B/Yamagata/16/88-like strain, 15 micrograms haemagglutinin.

Two (split/subunit) vaccines are currently available in Australia.

Recommendations for use

Annual vaccination is recommended for individuals in the following categories:

(i) persons of all ages with chronic debilitating disease, especially those with chronic cardiac, pulmonary, renal and metabolic disorders;
(ii) persons over 65 years of age;
(iii) persons receiving immunosuppressive therapy;
(iv) persons engaged in medical and health services, and essential public utilities.

Individuals in categories (i), (ii) and (iii) are at greater risk of complications or death from influenza than other members of the population.

Dosage and administration

Immunisation is normally undertaken in autumn, in anticipation of winter outbreaks of influenza.

One dose is sufficient for persons previously exposed to viruses of similar antigenic composition to the strain(s) present in the vaccine. In children and in those with some impairment of immune mechanisms, two doses separated by an interval of at least four weeks are recommended.

The vaccine should be administered by deep subcutaneous or intramuscular injection.

Side effects and adverse reactions

Children usually react more to influenza vaccine than adults.

Mild local reactions, consisting of swelling, redness, tenderness and/or pain may occur with low frequency during the 48 hours after vaccination.

Mild fever of short duration may occur occasionally.

Post-vaccination neurological disorders have been reported following the use of almost all biological products. In 1976, the United States Public Health Advisory Committee on Immunisation Procedures found that the Guillain-Barre syndrome (GBS) occurred after influenza immunisation at an incidence of approximately 1 in 100 000 and that the death rate in this 'series' was approximately 1 in 2 000 000. An association of GBS with influenza vaccination has not been documented in the United States since that time, and studies in other countries have failed to establish a relationship between influenza vaccination and GBS.

Amantadine

In Australia, amantadine hydrochloride is approved for use in the treatment of respiratory tract illness caused by influenza A. Practitioners should consult the relevant literature for prescribing information.

CDI corrigenda

CDI 90/10 contained an incorrect statement on dengue haemorrahagic fever (DHF). In the item on dengue in Vanuatu (Overseas Briefs No. 5 pg. 4) the final line should have read "DHF usually occurs when a patient is reinfected with a different dengue serotype".

CDI 90/11 suffered the apparently unavoidable problems that arise from the change to a new computer. Missing page numbers and the compression of table 2 page 22 were the more obvious results. It also appears a line at the bottom of page 2 may be missing in some copies. The final paragraph on page 2 should read:

> Areas of Angola, Gambia, Ghana, Guinea, Mali, Nigeria, Sudan, Zaire, Brazil, Columbia and Peru zare also currently considered to be yellow fever infected by the World Health Organisation.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 24/5/90 TO 6/6/90

1. CODE 018 - MICROBIOL DIAG UNIT, UNI MELB (VIC)
2. CODE 019 - FAIRFIELD HOSP (VIC)
3. CODE 065 - STATE HEALTH LAB (WA)
4. CODE 066 - PRINCESS MARGARET HOSP (WA)
5. CODE 110 - INST OF MED & VET SCIENCE (SA)
6. CODE 111 - ROYAL CHILDRENS HOSP (VIC)
7. CODE 112 - INST CLINICAL PATH & MED RES (NSW)
8. CODE 113 - PRINCE HENRY/PRINCE OF WALES HOSP (NSW)
9. CODE 114 - ROYAL ALEXAND RA CHILDRENS HOSP (NSW)
10. CODE 115 - STATE HEALTH LAB (QLD)
11. CODE 116 - WODEN VALLEY HOSP (ACT)

		019	065	110	112	114	115	116	TOTAL
0100	ADENOVIRUS NOT TYPED	1	4	0	6	0	8	0	19
0101	ADENOVIRUS TYPE 1	1	0	2	4	0	0	0	7
0102	ADENOVIRUS TYPE 2	1	0	1	3	0	0	0	5
0103	ADENOVIRUS TYPE 3	1	0	1	3	0	0	0	5
0104	ADENOVIRUS TYPE 4	0	0	1	3	0	0	0	4
0109	ADENOVIRUS TYPE 9	1	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	3	0	0	0	3
0122	ADENOVIRUS TYPE 22	0	0	1	0	0	0	0	1
0125	ADENOVIRUS TYPE 25	1	0	0	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	1	0	0	0	0	0	0	1
0128	ADENOVIRUS TYPE 28	1	0	0	0	0	0	0	1
0145	ADENOVIRUS TYPE 45	1	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	3	0	0	3
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	1	1
0299	INFLUENZA VIRUS - TYPING PENDI	0	1	0	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	6	0	16	2	1	32	0	57
0302	PARAINFLUENZA VIRUS TYPE 2	2	0	0	0	0	1	0	3
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	1	0	4	0	5
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	2	0	2
0400	RESPIRATORY SYNCYTIAL VIRUS (R	7	0	4	45	60	70	2	188
0500	RHINOVIRUS (ALL TYPES)	1	0	0	1	0	0	0	2
0600	MYCOPLASMA PNEUMONIAE	0	2	3	3	0	1	0	9
0900	COXSACKIEVIRUS GROUP B - NOT T	0	0	1	0	0	0	0	1
0901	COXSACKIEVIRUS B1	0	0	1	0	0	0	0	1
0902	COXSACKIEVIRUS B2	0	0	0	1	0	0	0	1
0903	COXSACKIEVIRUS B3	1	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	3	0	0	0	3
1007	ECHOVIRUS TYPE 7	1	0	0	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	2	0	0	3	0	0	0	5
1022	ECHOVIRUS TYPE 22	2	0	0	0	0	0	0	2
1025	ECHOVIRUS TYPE 25	1	0	0	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	0	1	0	2	0	0	3
1101	POLIOVIRUS TYPE 1	0	0	0	1	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	0	1	0	0	0	0	1
1104	POLIOVIRUS - MIXED VACCINAL ST	0	0	1	0	0	0	0	1
1200	MUMPS VIRUS	0	0	0	0	0	0	1	1
1300	HERPES VIRUS GROUP - NOT TYPED	2	1	0	0	0	0	0	3
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	1	0	23	0	1	6	31
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	12	11	4	4	3	1	35
1303	VARICELLA-ZOSTER VIRUS	5	8	0	4	1	4	0	22
1306	HERPES SIMPLEX TYPE 1	29	39	21	2	1	27	1	120
1307	HERPES SIMPLEX TYPE 2	49	77	26	14	0	33	0	199
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	1
1401	COXIELLA BURNETII	0	0	1	4	0	8	0	13
1502	PICORNIA VIRUS - NOT TYPED = E	0	7	3	0	1	36	0	47
1514	MOLLUSCUM CONTAGIOSUM	0	1	0	0	0	0	0	1
1515	CONTAGIOUS PUSTULAR DERMATITIS	0	0	0	1	0	0	0	1
1521	MEASLES VIRUS	0	0	1	0	0	0	0	1
1522	RUBELLA VIRUS	2	1	0	1	0	4	0	8
1532	HEPATITIS B ANTIGEN	12	36	9	44	0	2	2	105
1535	HEPATITIS A ANTIBODY	0	4	6	0	0	0	1	11
1541	CHLAMYDIA A - C. TRACHOMATIS	0	62	34	17	0	23	0	136
1553	LCM - LYMPHOCYTIC CHORIOMENING	1	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	30	3	1	3	5	18	1	61
1563	CORONAVIRUS	1	0	0	1	0	0	0	2
1564	ROTAVIRUS	2	1	0	0	1	0	0	4
1565	CALICI VIRUS	2	0	0	1	0	0	0	3
1566	NORWALK AGENT	1	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	3	0	0	3
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	0	1	1
9992	ROSS RIVER VIRUS	0	1	0	2	0	35	0	38
9993	ASTROVIRUS	1	0	0	1	0	0	0	2
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	1	1	1	0	3
9995	DENGUE	0	0	0	0	0	4	0	4
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	2	0	2
TOTAL		169	262	147	205	83	319	17	1202

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 24/5/90 TO 6/6/90

NSW: ICPMR; PHH PON; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP.
VIC: FAIRFIELD; RCH; MDU, UNI MELB
QLD: STATE LAB, BRIS; TOOWOOMBA PATH LAB; ROYAL BRIS HOSP.
WA: STATE LAB, PERTH; PMH.
SA: IMVS.
TAS: ROYAL HOBART HOSP; DIAGNOSTIC SERVICES, LAUNCESTON; LAUNCESTON GEN HOSP; DIAGNOSTIC SERVICES, HOBART; HOBART PATH; MERSEY GEN HOSP, LATROBE.
ACT: WVH.

		NSW	VIC	QLD	WA	SA	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	6	1	8	4	0	0	19
0101	ADENOVIRUS TYPE 1	4	1	0	0	2	0	7
0102	ADENOVIRUS TYPE 2	3	1	0	0	1	0	5
0103	ADENOVIRUS TYPE 3	3	1	0	0	1	0	5
0104	ADENOVIRUS TYPE 4	3	0	0	0	1	0	4
0109	ADENOVIRUS TYPE 9	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	3	0	0	0	0	0	3
0122	ADENOVIRUS TYPE 22	0	0	0	0	1	0	1
0125	ADENOVIRUS TYPE 25	0	1	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	1	0	0	0	0	1
0128	ADENOVIRUS TYPE 28	0	1	0	0	0	0	1
0145	ADENOVIRUS TYPE 45	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	3	0	0	0	0	0	3
0203	INFLUENZA B VIRUS	0	0	0	0	0	1	1
0299	INFLUENZA VIRUS - TYPING PENDI	0	0	0	1	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	3	6	32	0	16	0	57
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	1	0	0	0	3
0303	PARAINFLUENZA VIRUS TYPE 3	1	0	4	0	0	0	5
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	2	0	0	0	2
0400	RESPIRATORY SYNCYTIAL VIRUS (R	105	7	70	0	4	2	188
0500	RHINOVIRUS (ALL TYPES)	1	1	0	0	0	0	2
0600	MYCOPLASMA PNEUMONIAE	3	0	1	2	3	0	9
0900	COXSACKIEVIRUS GROUP B - NOT T	0	0	0	0	1	0	1
0901	COXSACKIEVIRUS B1	0	0	0	0	1	0	1
0902	COXSACKIEVIRUS B2	1	0	0	0	0	0	1
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	3	0	0	0	0	0	3
1007	ECHOVIRUS TYPE 7	0	1	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	3	2	0	0	0	0	5
1022	ECHOVIRUS TYPE 22	0	2	0	0	0	0	2
1025	ECHOVIRUS TYPE 25	0	1	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	2	0	0	0	1	0	3
1101	POLIOVIRUS TYPE 1	1	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	1	0	1
1104	POLIOVIRUS - MIXED VACCINAL ST	0	0	0	0	1	0	1
1200	MUMPS VIRUS	0	0	0	0	0	1	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	2	0	1	0	0	3
1301	HERPES SIMPLEX VIRUS - NOT TYP	23	0	1	1	0	6	31
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	8	0	3	12	11	1	35
1303	VARICELLA-ZOSTER VIRUS	5	5	4	8	0	0	22
1306	HERPES SIMPLEX TYPE 1	3	29	27	39	21	1	120
1307	HERPES SIMPLEX TYPE 2	14	49	33	77	26	0	199
1399	HERPES VIRUS TYPING PENDING	0	0	0	1	0	0	1
1401	COXIELLA BURNETII	4	0	8	0	1	0	13
1502	PICORNIA VIRUS - NOT TYPED = E	1	0	36	7	3	0	47
1514	MOLLUSCUM CONTAGIOSUM	0	0	0	1	0	0	1
1515	CONTAGIOUS PUSTULAR DERMATITIS	1	0	0	0	0	0	1
1521	MEASLES VIRUS	0	0	0	0	1	0	1
1522	RUBELLA VIRUS	1	2	4	1	0	0	8
1532	HEPATITIS B ANTIGEN	44	12	2	36	9	2	105
1535	HEPATITIS A ANTIBODY	0	0	0	4	6	1	11
1541	CHLAMYDIA A - C. TRACHOMATIS	17	0	23	62	34	0	136
1553	LCM - LYMPHOCYTIC CHORIOMENING	0	1	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	8	30	18	3	1	1	61
1563	CORONAVIRUS	1	1	0	0	0	0	2
1564	ROTAVIRUS	1	2	0	1	0	0	4
1565	CALICI VIRUS	1	2	0	0	0	0	3
1566	NORWALK AGENT	0	1	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	3	0	0	0	0	0	3
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	1	1
9992	ROSS RIVER VIRUS	2	0	35	1	0	0	38
9993	ASTROVIRUS	1	1	0	0	0	0	2
9994	SMALL VIRUS (LIKE) PARTICLE	2	0	1	0	0	0	3
9995	DENGUE	0	0	4	0	0	0	4
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	2	0	0	0	2
TOTAL		288	169	319	262	147	17	1202

NOTE: DIRECT COMPARISION BETWEEN STATES IS NOT POSSIBLE SINCE:
- SOME STATES HAVE MORE THAN ONE CONTRIBUTING LABORATORY; AND
- INTERSTATE REFERRRALS OCCUR REGULARLY.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 24/5/90 TO 6/6/90

1. CODE 00, 99 .... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 .... - ENCEPHALITIS
4. CODE M3 .... - MENINGITIS
5. CODE 04 .... - PARALYSIS
6. CODE 05, 13 .... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	4	0	0	0	11	0	0	0	0	15
0101	ADENOVIRUS TYPE 1	1	3	0	0	0	1	0	0	0	0	5
0102	ADENOVIRUS TYPE 2	3	2	0	0	0	0	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	0	2	1	0	0	0	0	0	0	0	3
0109	ADENOVIRUS TYPE 9	0	0	0	0	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	1	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	0	0	0	0	0	0	0	0	1	1
0145	ADENOVIRUS TYPE 45	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	1	0	0	0	1	0	0	0	0	2
0203	INFLUENZA B VIRUS	0	1	0	0	0	0	0	0	0	0	1
0299	INFLUENZA VIRUS - TYPING PENDI	0	1	0	0	0	0	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	1	56	0	0	0	0	0	0	0	0	57
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	1	3	0	0	0	0	0	0	0	0	4
0399	PARAINFLUENZA VIRUS TYPING PEN	0	2	0	0	0	0	0	0	0	0	2
0400	RESPIRATORY SYNCYTIAL VIRUS (R	9	179	0	0	0	0	0	0	0	0	188
0500	RHINOVIRUS (ALL TYPES)	0	2	0	0	0	0	0	0	0	0	2
0600	MYCOPLASMA PNEUMONIAE	2	5	0	0	0	0	0	0	0	0	7
0900	COXSACKIEVIRUS GROUP B - NOT T	0	0	0	0	0	0	0	0	0	1	1
0901	COXSACKIEVIRUS B1	1	0	0	0	0	0	0	0	0	0	1
0902	COXSACKIEVIRUS B2	0	0	0	0	0	1	0	0	0	0	1
0903	COXSACKIEVIRUS B3	0	0	0	1	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	1	0	0	0	0	2	0	0	0	0	3
1007	ECHOVIRUS TYPE 7	0	1	0	0	0	0	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	1	1	0	1	0	1	0	0	0	0	4
1022	ECHOVIRUS TYPE 22	0	1	0	0	0	0	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	3	0	0	0	0	0	0	0	0	3
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	1	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	2	2
1301	HERPES SIMPLEX VIRUS - NOT TYP	3	1	1	0	0	0	0	0	0	17	22
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	6	0	0	0	0	1	1	0	0	0	8
1303	VARICELLA-ZOSTER VIRUS	2	1	1	0	0	0	0	0	0	18	22
1306	HERPES SIMPLEX TYPE 1	2	6	0	0	0	0	1	2	1	73	85
1307	HERPES SIMPLEX TYPE 2	4	0	1	0	0	0	0	0	0	74	79
1401	COXIELLA BURNETII	6	2	0	0	0	1	0	0	0	0	9
1502	PICORNIA VIRUS - NOT TYPED = E	2	18	1	1	5	16	0	1	0	2	46
1515	CONTAGIOUS PUSTULAR DERMATITIS	0	0	0	0	0	0	0	0	0	1	1
1521	MEASLES VIRUS	0	0	0	0	0	0	0	0	0	1	1
1522	RUBELLA VIRUS	4	0	0	0	0	0	0	0	0	2	6
1532	HEPATITIS B ANTIGEN	64	0	0	0	0	0	28	0	0	0	92
1535	HEPATITIS A ANTIBODY	1	0	0	0	0	0	10	0	0	0	11
1541	CHLAMYDIA A - C. TRACHOMATIS	17	0	0	0	0	0	0	0	0	0	17
1553	LCM - LYMPHOCYTIC CHORIOMENING	0	0	0	0	0	1	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	2	13	0	0	0	1	7	1	4	0	28
1563	CORONAVIRUS	0	1	0	0	0	1	0	0	0	0	2
1564	ROTAVIRUS	0	0	0	0	0	4	0	0	0	0	4
1565	CALICI VIRUS	0	0	0	0	0	3	0	0	0	0	3
1566	NORWALK AGENT	0	0	0	0	0	0	0	1	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	2	0	0	0	1	0	0	0	0	3
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	0	1	0	0	0	1
9992	ROSS RIVER VIRUS	17	0	0	0	0	0	0	0	0	2	19
9993	ASTROVIRUS	0	0	0	0	0	2	0	0	0	0	2
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	3	0	0	0	0	3
9995	DENGUE	4	0	0	0	0	0	0	0	0	0	4
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	2	0	0	0	0	0	0	0	0	0	2
TOTAL		156	313	5	3	5	55	48	5	5	194	789

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 24/5/90 TO 6/6/90

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	2	1	0	0	0	0	0	1	0	0	4
0101	ADENOVIRUS TYPE 1	1	0	0	0	0	0	0	1	0	0	2
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	0	0	0	0	0	2
0104	ADENOVIRUS TYPE 4	4	0	0	0	0	0	0	0	0	0	4
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	0	0	2	0	2
0122	ADENOVIRUS TYPE 22	1	0	0	0	0	0	0	0	0	0	1
0125	ADENOVIRUS TYPE 25	0	0	0	0	0	0	0	0	1	0	1
0128	ADENOVIRUS TYPE 28	0	0	0	0	0	0	0	0	1	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	1	0	0	0	0	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	0	1	0	1
0600	MYCOPLASMA PNEUMONIAE	0	0	1	0	0	0	0	0	1	0	2
1011	ECHOVIRUS TYPE 11	0	0	0	0	0	0	0	0	0	1	1
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	0	0	1	0	0	1
1025	ECHOVIRUS TYPE 25	0	0	0	0	0	0	0	1	0	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	0	0	1	1
1104	POLIOVIRUS - MIXED VACCINAL ST	0	0	0	0	0	0	0	1	0	0	1
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	1	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	9	0	0	0	0	0	0	0	0	9
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	16	7	0	0	0	0	4	0	27
1306	HERPES SIMPLEX TYPE 1	6	25	0	0	0	0	0	1	3	0	35
1307	HERPES SIMPLEX TYPE 2	0	119	0	0	0	0	0	0	1	0	120
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	0	0	0	1
1401	COXIELLA BURNETII	0	0	0	0	0	0	1	3	0	0	4
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	0	0	1	0	1
1514	MOLLUSCUM CONTAGIOSUM	0	1	0	0	0	0	0	0	0	0	1
1522	RUBELLA VIRUS	0	0	0	0	1	0	0	0	1	0	2
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	13	0	13
1541	CHLAMYDIA A - C. TRACHOMATIS	4	115	0	0	0	0	0	0	0	0	119
1556	CMV - CYTOMEGALOVIRUS	0	2	0	1	0	1	0	1	28	0	33
9992	ROSS RIVER VIRUS	0	0	0	0	14	0	0	3	2	0	19
TOTAL		20	273	18	9	15	1	1	14	60	2	413