

THE RELATIONSHIP BETWEEN ORAL HEALTH AND SYSTEMIC HEALTH IS NOT WELL UNDERSTOOD

Alan Patterson
Chief Dental Officer
NSW Health Department

Why has oral health received so little attention within the health system over the past two decades? Is it because the pre-1960s era, when tooth loss was considered inevitable, was followed in the 1970s by the advent of water fluoridation and the perception that flouridation ensured good oral health? Or does the fact that approximately 80 per cent of oral health care is provided by the private oral health sector promote a perception that the public health care system needs only to provide water fluoridation and a restricted welfare service?

The effects of oral neglect and oro-dental disease continue to manifest themselves as severely debilitating chronic conditions with the potential for long-term adverse health, social and economic consequences for a significant and growing number of people. The 1992 National Health Strategy and the 1998 Senate Inquiry into Public Dental Services both identified the extent of oral disease and the need for access to oral health care as an important community issue.^{1,2} The 1995 Australian *National Health Survey* found that oral disease was the sixth most frequent illness condition,³ with more than one million people visiting a dentist in the two weeks prior to the survey. In 1998, the Australian Institute of Health and Welfare reported a cost-of-illness analysis for dental services in 1993–1994 of almost \$1.8 billion, or six per cent of the total health budget.⁴ That included \$0.5 billion in costs to NSW alone.

Further, like so many other health indicators, the burden of oral disease is shared disproportionately by such disadvantaged groups as refugees, the home-bound, aged, and homeless youth. Oro-dental diseases are predominantly lifestyle diseases and, as Kickbusch recognised in 1989, 'Lifestyle diseases are no longer diseases of

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affluence but an unnecessary burden on the disadvantaged'.⁵

Of growing interest is the evidence suggesting a direct relationship between oral health and diseases such as cardiovascular disease and diabetes. This is potentially of great public health significance, as oro-dental disease is largely preventable, and in many instances readily treatable. If oral health is to be recognised within the broader health system as having an important part to play in improving the health of our community, it is imperative that this relationship between oral health and other diseases be examined more closely.

To this end, and beginning with this edition of the *Public Health Bulletin*, a series of articles will examine current and emerging issues in oral health. These issues are both exciting and challenging, not only for the oral health profession, but also for medical and public health professionals. Over four issues, the *Bulletin* will examine topics such as the oral manifestations of child neglect; the influence of oral health on the general health of the aged and those with special needs; oral carcinoma; and the impact of HIV-AIDS on oral health. The future workforce required to meet the changing patterns of disease resulting from our aging population; the economic

impact of oral disease; and research trends will also be explored.

This edition highlights two important areas. The first article examines the relationship between periodontal disease and systemic health, with particular reference to pregnancy, diabetes and cardiovascular disease. The second explores the impact on oral health of xerostomia (dry mouth), the most common cause of which is medication.

REFERENCES

1. *Improving Dental Health in Australia*. National Health Strategy Background Paper No. 9. Canberra: AGPS, 1992. ISBN 0 642 17468 7.
2. Australia, Parliament 1998, *Inquiry into public dental services*, Senate Community Affairs References Committee Report, Parliamentary Paper. Canberra: Commonwealth of Australia, 1998.
3. Australian Bureau of Statistics 1995, *National Health Survey*, Cat. no. 4364.0, ABS, Canberra.
4. Mathers C, Pen MR, Carter C, Stevenson C. *Health system costs of diseases and injury in Australia 1993-94*, Health and Welfare Expenditure Series No. 2, Canberra: Australian Institute of Health and Welfare, 1998.
5. Kickbusch I. Self-care in health promotion. *Soc Sci Med* 1989; 29(2): 125-130. ❏

PERIODONTAL DISEASES AND SYSTEMIC HEALTH: ASSOCIATIONS, DIRECTIONS, IMPLICATIONS

Barbara Anne Taylor

Head, Department of Periodontics
United Dental Hospital of Sydney, Surry Hills

Recent findings that suggest that periodontal disease may be an independent risk factor for a number of significant systemic diseases, including cardiovascular diseases, are so important that they have been referred to as 'the inversion of a paradigm'.¹ While it is well known that a number of diseases and normal physiological states are associated with an increase in the risk of periodontal disease, until now we have not recognised that the chronic infection and ulceration in periodontal disease could affect systemic health beyond certain limited and well-defined instances, such as bacterial endocarditis. This article describes the association between periodontal disease and systemic health, in particular the evidence for an effect in pregnancy, diabetes and cardiovascular disease; and the implications of this for public health.

THE PERIODONTAL DISEASES

The periodontal diseases are a family of chronic inflammatory diseases, including gingivitis and periodontitis, that involve the periodontium (the bone and soft tissues that support the teeth in the jaws). Gingivitis, an inflammation of the gums, is a very common condition. Periodontitis is also common and is a more severe condition that causes loss of bone that supports the teeth. It afflicts 15 to 20 per cent of the adult dentate (people with their natural teeth) population in Australia.² Research over the last 30 years has described the role of bacterial plaque in causing periodontal disease, and controlled clinical trials suggest that periodontal treatment (such as scaling and cleaning of teeth) usually stabilises the condition and improves periodontal health.^{3,4} Thus, periodontal management, with an emphasis on bacterial plaque control, is an evidence-based intervention with established health outcomes.

A wide range of systemic conditions, ranging from the hormonal changes of puberty and pregnancy to disease

entities involving immune dysfunction, connective tissue disease and malignancy, have manifestations in the mouth. The dynamics of the periodontium are a product of its circulation, hormonal changes and immune response mechanisms. Changes in systemic health that affect any of these factors can be reflected in changes in periodontal health. While this is well known, the main concerns of periodontics remain focused on oral causes, oral risks and oral remedies. Indeed, dental education, management and research have been limited by the dualistic notion that the oral cavity is separate from the rest of the body.

However, recent research suggests that periodontal diseases can influence systemic health through two mechanisms. The first is direct, by the pathogenic action of dental plaque bacteria that enter the bloodstream (bacteraemia). The second is indirect, by the distant effect of inflammatory mediators, such as cytokines, prostaglandins and serum antibodies, that are induced by periodontal disease.

RECENT RESEARCH

Preterm birth

Preterm birth is usually a result of rupture of membranes before 37 weeks gestation or preterm labour. The rate of preterm birth remains unchanged at approximately 6.3 per cent of all pregnancies in Australia despite improvements in antenatal care. Estimates are that one-third of premature births are of unknown causes. Approximately 75 per cent of spending on neonatal morbidity in Australia is as a result of preterm birth.

Maternal infection is associated with preterm labour. For example, infection in the maternal genito-urinary tract is associated with a higher risk of preterm birth. Periodontal disease, particularly gingivitis, is common among pregnant women. This is thought to be a result of an increased inflammatory response modulated by the effect of sex steroid hormones on the gums.⁵ The frequency of transient bacteraemias means there is a high risk of translocation of bacteria pathogens or the products of chronic periodontal infection, such as interleukin-1 β and prostaglandin E₂ to other parts of the body.

For example, *Fusobacterium nucleatum* has been isolated from amniotic fluid among women with preterm labour but who had intact membranes. *F. nucleatum* is prevalent in the space between the gum and the tooth in humans, but not prevalent in the vaginal microflora. This suggests that the *F. nucleatum* infection in the amniotic fluid may have originated in the mouth.⁶

Recently, a case-control study of 124 mothers found that mothers of preterm babies had poor periodontal health when compared with mothers of normal birth-weight infants.⁷ This association remained after controlling for

factors such as smoking, alcohol consumption, age, race and level of prenatal care, which suggests that periodontal infection is associated with premature labour. This association has not been verified in an Australian population.

Diabetes

Type 2, or non-insulin-dependent diabetes (NIDDM), is the predominant form of diabetes in developed countries. Its prevalence is increasing in Australia because of greater awareness of the condition (leading to increased diagnosis and reporting), shifts in lifestyle patterns, an aging population and a higher prevalence of diabetes in some recent immigrant groups. Further, Aboriginal people and Torres Strait Islanders have the fourth highest prevalence rate of NIDDM in the world.⁸

Periodontal disease is a complication of diabetes.⁹ Numerous studies have shown that being diabetic is associated with a greater risk and severity of periodontal disease, particularly if the diabetes is poorly controlled. This susceptibility is linked to elevated blood glucose, altered protein synthesis, and changes in the microcirculation of the gums.

Conversely, recent evidence suggests that periodontal disease may cause poor control of diabetes. The Gila River Indian Community in Arizona has a prevalence of NIDDM of 50 per cent among its adults, the highest reported anywhere in the world. Recent analysis of periodontal data and blood sugars from this group showed that severe periodontitis was a risk for poor blood sugar control.¹⁰ Prospective controlled clinical trials in an Australian population could help to determine whether periodontal treatment has a beneficial effect on the stability of blood sugar control.

Cardiovascular Disease

Eating, brushing teeth, flossing and dental treatment may all lead to transient bacteraemias with bacteria that reside in the mouth, such as the streptococcus species. Bacteraemia is a particular risk for people with damaged heart valves, prosthetic valves and various cardiac anomalies who may develop bacterial endocarditis if bacterial vegetations form in parts of the heart with turbulent blood flow. Maintaining good dental health in these patients will reduce bacteraemias from everyday activities and reduce the need for dental interventions.

However, of broader public health concern is the suggestion that periodontitis is an independent risk factor for cardiovascular diseases (CVD) such as atherosclerosis, coronary thrombosis, ischaemic heart disease and myocardial infarction. The fact that the periodontal diseases and CVD share many risk factors, such as smoking, low socio-economic status, stress, hypertension

and social isolation, complicates the study of the relationship between these diseases.

Two studies sponsored by the United States Department of Veterans Affairs—the Normative Aging Study and the Dental Longitudinal Study—permitted a prospective assessment of the effect of periodontal disease on CVD. The Normative Aging Study followed a group of 2280 male veterans from the greater Boston area who were living independently in the community and were healthy when they entered the study in 1961. The Dental Longitudinal Study started in 1968, and its 1221 participants were part of a subsample of the original Normative Aging Study population.

Comprehensive oral examinations were carried out at three-year intervals. A range of variables was assessed, including alveolar bone loss as an indicator of periodontal disease. Mortality and morbidity from coronary heart disease (myocardial infarction, angina pectoris) and stroke was measured, along with risk factors such as cigarette smoking, blood pressure, cholesterol levels and body mass index.

Statistical analysis indicated a strong association between alveolar bone loss, stroke and cardiovascular disease. Periodontal disease was found to be a risk factor that was additional to other risk factors for cardiovascular disease and an independent risk factor for stroke.

A possible causative mechanism has been suggested from evidence emerging from animal laboratory studies. The viridans group of streptococci are found in dental plaque and do not usually cause dental disease. However, they can enter the circulation through the mucosa surrounding the teeth and assume a pathogenic role. Rabbits infused with *Streptococcus sanguis* to determine whether infection would affect cardiovascular health exhibited dose-dependent changes in electrocardiograms, heart rate, blood pressure and cardiac contractility.¹² Further, platelet aggregation and fibrin formation was found on heart valve vegetations of endocarditis, together with areas of myocardial infarction. This suggested that dental plaque bacteria could adversely affect cardiovascular health by stimulating thrombogenic events. *Porphyromonas gingivalis*, a gram-negative periodontal pathogen, can also bind onto atheromatous plaques and modify or promote their development as it, like *S. sanguis*, expresses an antigen that can induce platelet aggregation.

IMPLICATIONS AND DIRECTIONS

The evidence that oral health modifies systemic health has implications not only for the future direction of dental

care, research and education in Australia, but also for the broader public health community. Dental health should be promoted not only for itself but also as an integral part of general health care. Indeed, a curriculum that acknowledges that oral health care is an integral part of general health care is being developed at the Faculty of Dentistry at the University of Sydney. Scant information is available about periodontal disease and systemic health in Australia, suggesting a need for appropriate, well-designed and adequately funded research. Public health care in Australia can only benefit from recognising the relationship between periodontal disease and systemic health.

REFERENCES

1. Page RC. The pathobiology of periodontal diseases may affect systemic diseases: Inversion of a paradigm. *Ann Periodontol* 1998; 3: 108–120.
2. Barnard PD 1993, *National Oral Health Survey Australia 1987–88*, Department of Health, Housing, Local Government and Community Services, AGPS Press, Canberra.
3. Axelsson P, Lindhe J. Effect of controlled oral hygiene procedures on caries and periodontal disease in adults. *J Clin Periodontol* 1978; 5: 133–151.
4. Suomi JD, Greene JC, Vermillion JR, et al. The effect of controlled oral hygiene procedures on the progression of periodontal disease in adults: Results after third and final year. *J Periodontol* 1971; 42: 152–160.
5. Mariotti A. Sex steroid hormones and cell dynamics in the periodontium. *Critical Reviews in Oral Biology and Medicine* 1994; 5: 27–53.
6. Hill GB. Preterm birth: Associations with genital and possibly oral microflora. *Ann Periodontol* 1998; 3: 222–232.
7. Offenbacher S, Katz V, Fertik G, et al. Periodontal infection as a possible risk factor for preterm low birth weight. *J Periodontol* 1996; 67: 1103–1113.
8. McCarty DJ, Zimmet P, Dalton A, Segal L, Welborn TA. *The rise and rise of diabetes in Australia, 1996: A review of statistics, trends and costs*. Canberra: Diabetes Australia, 1996.
9. Löe H. Periodontal Disease. The sixth complication of diabetes mellitus. *Diabetes Care* 1993; 16: 329–334.
10. Taylor GW, Burt BA, Becker MP, et al. Severe periodontitis and risk for poor glycemic control in patients with non-insulin-dependent diabetes mellitus. *J Periodontol* 1996; 67: 1085–1093.
11. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol* 1996; 67: 1123–1137.
12. Herzberg MC, Meyer MW. Effects of oral flora on platelets: Possible consequences in cardiovascular disease. *J Periodontol* 1996; 67: 1138–1142. ❏

ORAL AND DENTAL MANIFESTATIONS OF XEROSTOMIA: PUBLIC HEALTH IMPLICATIONS OF A COMMON AND UNDER-RECOGNISED ADVERSE DRUG REACTION

Mark Schifter

*Staff Specialist and Head, Oral Medicine Unit
Department of Oral Diagnostic Sciences
Westmead Centre for Oral Health
(formerly Dental Clinical School, Westmead Hospital)*

The use of medications that have significant adverse implications for oral health and the safe delivery of dental care is increasing.¹ Medication use in general is expected to rise as the population ages, as will the number of dentate patients in the over-65 age group.¹ Adverse drug reactions, the most common of which is xerostomia, or dry mouth, may have direct effects on oro-dental structures.^{1,2} Decreased salivary flow and function leads directly to increased dental decay (caries) and impairment of the integrity of the oral lining, and it compromises such functions as tasting, speaking and swallowing.^{3,4} As described in the article by Barbara Anne Taylor, these effects have broader public health implications. Poor oral health status has been linked recently to major systemic conditions, such as cardiovascular disease.^{5,6} As well, diabetes and infective endocarditis have a well-established association with dental disease.^{7,8} This article examines the major groups of drugs causing xerostomia and the potential impact on both oral and general health.

Xerostomia is a symptom and does not itself represent a pathological entity. It may be defined as dryness of the oral mucosa of varying severity and a reduced flow in saliva (salivary hypofunction).⁹ Up to 1.5 litres of this complex secretion is produced every 24 hours. Of this, 20 per cent is produced by the parotid glands, which increase their contribution by up to 50 per cent on stimulation; 65 per cent by the submandibular glands; and the remaining 15 per cent by the sublingual and minor salivary glands.⁹

Little is known of the prevalence or the severity of salivary hypofunction in Australia. In the United States, surveys have found that up to 60 per cent of institutionalised elderly people and 30 per cent of older community-dwelling individuals complain of a dry mouth.^{10,11}

The leading causes of xerostomia are iatrogenic, predominantly as an adverse side-effect of many medications. Although the exact mechanisms are not understood, up to 400 drugs have been shown to induce xerostomia.^{1-4,12} The main classes of drugs implicated include the anticholinergics; the anti-hypertensives, especially the diuretic agents; the anti-depressive agents, particularly the tricyclics; and most analgesics. These classes of drugs are used predominantly, often in

combination, by older Australians. Aging itself is associated with some diminution of salivary function; however, this physiological change is much less severe than previously thought. Another group of drugs that cause profound salivary hypofunction are the major anti-psychotics, especially the phenothiazines and lithium.¹²

The effects of reduced salivary flow are potentially profound. Loss of the essential lubricating quality of saliva results in impairment of speech, mastication, and the early phases of swallowing and digestion. One study demonstrated that some 70 per cent of members of a retirement village suffered from medication-induced xerostomia that led directly to their avoidance of foods that were drier and more difficult to masticate and swallow, such as bread and crunchy vegetables. Concern was expressed that this could contribute to under-nutrition in these populations.⁹

Saliva is essential for protection against dental caries. Saliva contains anti-bacterial substances that inhibit and destroy cariogenic (caries-causing) members of the plaque flora. Saliva buffers the acid produced by cariogenic bacteria that can lead to dental decay by dissolving the mineralised-calcified component of the teeth. As well, saliva is essential for the re-mineralisation process, which allows the early repair of non-cavitated carious lesions, effectively forestalling decay. Thus, lack of saliva can lead to the loss of teeth by dental decay.³

Replacing lost teeth with dental prostheses in xerostomia patients is unsatisfactory. Dentures are expensive to manufacture and are highly reliant on the lubricating properties of saliva for their retention and function. Further, they exacerbate the problem of decay by retaining food as well as the plaque containing the bacteria that process food debris into the acid responsible for dental decay.

Until recently, the morbidity and poorer quality of life associated with xerostomia has been limited to the resultant need to extract teeth and fit replacement prostheses. However, new evidence demonstrates that poor oral health may be linked to adverse systemic conditions such as coronary heart disease (CHD).^{5,6} A recent study demonstrated a statistically significant association between the diagnosis of CHD and such oral health parameters as the number of missing teeth and complaints of xerostomia. Indeed, these oral health parameters were more strongly associated with CHD than such recognised risk factors as elevated serum cholesterol levels, obesity, late-onset diabetes and tobacco use.⁵ The relationship between these oral health parameters and CHD is not

understood, but one hypothesis is that certain oral flora may play a role in the pathogenesis of CHD. Consequently, the risks associated with xerostomia may be more extensive than previously believed.

Therefore, while the management of xerostomia is highly problematic, it is very important. Education of general medical practitioners and community-based pharmacists regarding the detrimental and xerogenic effect of certain medications is vital. Patients should be advised of the effect of their medications; warned to avoid simple potentially cariogenic sugars, such as table sugar; and encouraged to use non-calorific sweeteners, improve oral hygiene practices and attend their dentist for review.

For specific cases of salivary hypofunction, treatment is aimed at relief of symptoms. The use of salivary stimulants such as pilocarpine has not been well studied in patients with medication-induced xerostomia. Artificial salivary replacements are by and large unsuccessful. They are poorly tolerated by patients and have none of the anti-bacterial and re-mineralising abilities of normal saliva. Intensive fluoride preparations are useful for limiting the severity and extent of dental caries; however, they are dependant upon dedicated and regular home use. Topical anti-microbials or specific anti-fungals may also be useful for gingival and mucosal care.

In the absence of Australian research, targeted epidemiological research, in combination with education programs for the professional and lay carers—particularly those responsible for the institutionalised elderly and those with major psychoses—should be a public health priority. The morbidity, potential major systemic health effects, and the social and financial costs of xerostomia with its resultant loss of function and oral comfort need to be more fully researched and publicised. What has been learned from the preventive reforms put in place for the younger members of our community, such as the benefits

of water fluoridation, needs now to be re-assessed and applied to the increasing number of older Australians.

REFERENCES

1. Smith RG, Burtner AP. Oral side-effects of the most frequently prescribed drugs. *Special Care Dent* 1994; 14: 96–101.
2. Lewis IK, Hanlon JT, Hobbins MJ, Beck JD. Use of medications with potential oral adverse drug reactions in community-dwelling elderly. *Spec Care Dent* 1993; 13: 171–176.
3. Papas AS, Joshi A, MacDonald SL, et al. Caries prevalence in xerostomic individuals. *J Can Dent Assoc* 1993; 59: 171–179.
4. Atkinson JC, Wu AJ. Salivary gland dysfunction: Causes, symptoms, treatment. *J Am Dent Assoc* 1994; 125: 409–416.
5. Loesche WJ, Schork A, Terpenning MS, et al. Assessing the relationship between dental disease and coronary heart disease in elderly US veterans. *J Am Dent Assoc* 1998; 129: 301–311.
6. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol* 1996; (Suppl) 67: 112–137.
7. Finney LS, Finney MO, Gonzalez-Campoy JM. What the mouth has to say about diabetes. *Postgrad Med* 1997; 102: 117–124, 126.
8. Strom BL, Abrutyn E, Berlin JA, et al. Dental and cardiac risk factors for infective endocarditis: A population-based, case control study. *Ann Int Med* 1998; 129: 761–769.
9. Crockett DN. Xerostomia: The missing diagnosis. *Aust Dent J* 1993; 38: 114–118.
10. Marcus SE, Kaste LM, Brown LJ. Prevalence and demographic correlates of tooth loss among the elderly in the United States. *Spec Care Dent* 1994; 14: 123–127.
11. Loesche WJ, Bromberg J, Terpenning MS, et al. Xerostomia, xerogenic medications and food avoidances in selected geriatric groups. *J Am Geriatr Soc* 1995; 43: 401–407.
12. Peeters FPML, deVries MW, Vissink A. Risks for oral health with the use of antidepressants. *Gen Hosp Psychiatry* 1998; 20: 150–154. ☒

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PUBLIC HEALTH WORKFORCE: RESULTS OF A NSW STATEWIDE CONSULTATION ON THE DEVELOPMENT OF THE NATIONAL PUBLIC HEALTH WORKFORCE

Lynne Madden

*Manager, Public Health Training and Development Unit
NSW Health*

Allison Salmon

*Public Health Officer (Drugs and Alcohol)
NSW Public Health Officer Training Program*

The National Public Health Partnership (NPHP) was established in 1996 by the Australian Health Ministers. The Partnership Group consists of the Chief Health Officers or the heads of the Public Health Divisions in each State or Territory and the chief executives of the National Health and Medical Research Council and the Australian Institute of Health and Welfare. A representative of the New Zealand Government participates as an observer. The initial focus of the NPHP has been to strengthen the national infrastructure of public health, including the development of the public health workforce. In August–September 1998, the NSW Department of Health, on behalf of the NPHP, undertook a statewide consultation seeking opinions on the development of the national public health workforce. This article describes the consultation process, highlights some of the findings and provides an update on how the NPHP intends to proceed with national public health workforce development.

THE CONSULTATION PROCESS

The NPHP established a planning group in late 1997 which oversaw: the production of a background paper that summarised recent public health workforce development in Australia; and a discussion paper that clarified some of the current issues in this area and suggested priorities and potential objectives for a national approach to workforce development.

These two documents were distributed to all the States and Territories, who were asked to consult with their public health workforces on the discussion paper. Results of all the consultations were to be used by the planning group to prepare a submission to the NPHP proposing future action on national public health work force development.

The NSW consultation consisted of three components: a request for written feedback, a forum of key stakeholders and a rural teleconference. The NSW Department of Health requested written feedback from public health groups within the health and academic sectors who were expected to be interested in the development of the public health workforce. Included were:

- branches within the Public Health and Policy Divisions of the Department of Health

- Divisions of Population Health
- professional groups, such as Public Health Unit directors, public health nurses, environmental health officers, and health promotion workers
- local health service special interest groups, such as the Epidemiology Special Interest Group
- academic groups, such as the Department of Public Health and Community Medicine at the University of Sydney
- bodies representing public health professionals, such as the NSW Branch of the Public Health Association
- non-government organisations, e.g. the National Heart Foundation
- members of the NSW Department of Health's Public Health Workforce Development Committee
- rural representatives of many of the preceding organisations
- professional bodies representative of general practice.

Representatives of these groups were invited to participate in a forum to discuss: themes emerging from the written responses; perceived gaps in the public health workforce in NSW; and examples of successful workforce development initiatives. While there was some rural representation at the forum, a teleconference was organised to seek the opinions of a number of rural representatives who were unable to attend.

RESULTS OF THE CONSULTATION

Respondents felt that the NPHP is well positioned to take a strategic view across the many services and academic structures that contribute to public health work and workforce development in Australia. There was also consistent and strong support for linking education and practice in public health training. Of the many themes that emerged from the process, we have selected four to profile here:

- core knowledge and skills shared by the public health workforce
- types of public health workers
- learning organisations
- rural public health workforce.

Core knowledge and skills shared by the public health workforce

There was a consensus regarding the need to articulate the specialised knowledge and skills required by the public health workforce. It was recognised that these skills are usually held at different levels of sophistication depending upon the responsibilities of the position and, consequently, that training should be directed at achieving

an appropriate level of skill. Figure 1 lists the knowledge and skills suggested by the consultation as core to public health work.

Types of public health worker

Three broad types of public health worker were identified, each with particular combinations of knowledge and skills: generalist public health workers, specialist public

health workers and health workers who have a public health component embedded within their professional practice.

Generalist public health workers are required to take a broad perspective of issues and are usually found in positions where resources or organisational structure demand multiple skills. Examples ranged from many public health service management positions to workers in areas of social need (e.g. drug and alcohol) and rural areas.

Specialist public health workers have knowledge or skills pertaining to either a particular area or setting or a higher level of skill than generalist workers. Examples included epidemiologists, health promotion workers and environmental health officers.

The third type is health workers who have a public health component embedded within their professional practice. There was agreement that most health workers required some public health knowledge and skills to be effective. For example, those in active clinical practice should have an understanding of evidence-based health care. The Royal Australian College of General Practitioners has recognised this need and has recently included population health in their training for general practice. Population Health Needs and Priorities is one of three dimensions used to develop their new curriculum.

FIGURE 1

CORE KNOWLEDGE AND SKILLS NECESSARY FOR PUBLIC HEALTH WORK

- Management skills, including management of change, resource management and organisational development
- Service planning
- Intersectoral working
- Whole-of-government approaches
- Team building
- Community consultation/community planning
- Communication and negotiation
- Establishing and maintaining information technology skills to support e-mail and Internet access
- Maintenance of data sets
- Descriptive epidemiology and surveillance
- Infectious diseases
- Evidence-based practice
- Program/service evaluation
- Intervention designs
- Health promotion

Learning organisations

Many respondents emphasised that workforce development is seldom achieved effectively in isolation from broader organisational concerns. They felt that to be effective workforce development needs to be integrated into the overall development of an organisation and its goals, a concept embodied by the term 'learning organisations'. In other words, learning that is system wide focuses not only on the needs of the individual, but also on the needs of the whole organisation. Figure 2 suggests a model that acknowledges the impact of the physical infrastructure of the organisation on workforce development.

FIGURE 2

ORGANISATIONAL INFRASTRUCTURE AND WORKFORCE DEVELOPMENT



Rural public health workforce

The rural public health workforce was identified as having a particular need for development. This group encompasses multi-skilled clinical and community health workers who usually undertake a wider range of activities than their colleagues in metropolitan areas. Because it can be difficult to recruit highly skilled specialists to rural areas, development of the existing work force is essential. Skills in data collection and the organisational aspects of public health surveillance were cited as areas in which further training is needed.

OUTCOME FROM THE NATIONAL CONSULTATION PROCESS

The NPHP executive prepared a final report that was forwarded to the NPHP for their consideration in November 1998. At that meeting, a number of priority areas were agreed upon, including health promotion, leadership skills and environmental health (particularly environmental toxicology). An infrastructure project to examine the monitoring and planning of the public health workforce and to identify core public health workforce disciplines was also supported. The original planning group has now been disbanded and the next phase will be taken forward by a number of different groups. The *Bulletin* will continue to report on the outcomes of this work. ❧

A paper summarising the results of the NSW consultation and including copies of the workforce documents produced by the NPHP was published by NSW Health in December 1998. The document, *NSW Consultation on the Development of the National Public Health Workforce for the National Public Health Partnership*, is available on the Public Health website at www.health.nsw.gov.au/public-health/nphw.

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TRENDS

Legionnaires disease

A cluster of patients with pneumonia due to **Legionnaires disease** was reported in February this year (Figure 3 and Table 1). The causative organism, *Legionella pneumophila*, commonly develops in collections of warm water and may infect people who inhale aerosols produced by the cooling towers air conditioning systems in large buildings. The elderly, people who are immunosuppressed and smokers are at increased risk for the disease. Between 33 and 105 cases are reported yearly in NSW.

Seven cases of pneumonia due to *Legionella pneumophila* serogroup one were reported in people living in different parts of western Sydney, with onset dates between the 7th and 20th of February. Four of these cases were confirmed by positive tests for urinary antigen, and three were confirmed by sputum culture. All seven patients required hospitalisation, four in intensive care. All have recovered.

Western Sector Public Health Unit investigators gathered exposure histories for each case, either by direct interview or, for two cases who were too ill for questioning, by interviewing close relatives. No single common exposure was found for the cases.

Three cases reported visiting the vicinity of a shopping complex, and two cases reported visiting a club. However, given the popularity of these institutions in the locality, no definite causal link could be drawn. As a precaution, six cooling towers within a one kilometre radius of the shopping complex and the two cooling towers at the club were inspected and sampled and it was advised that they be cleaned. While none of the samples from around the shopping complex were positive for *Legionella pneumophila*, one of the cooling towers at the club returned a high positive count that was probably associated with the exhaustion of biocide.

Subsequent tests at the Institute of Clinical Pathology and Medical Research (ICPMR) laboratory, Westmead, on the sputum isolates from the three culture-confirmed patients found that they had different DNA fingerprinting patterns, suggesting that the cases were unrelated.

Such reports of apparently sporadic cases of Legionnaires disease serve as a reminder of the importance of

minimising bacterial growth by careful inspection, cleaning and maintenance of cooling towers by building owners. For more information contact your local council or public health unit.

Arbovirus surveillance

During January and February, the NSW Arbovirus Surveillance and Vector Monitoring Program detected Kunjin virus in mosquitoes collected in the Griffith and Leeton areas of Greater Murray.

Kunjin is a flavivirus whose primary hosts are believed to be water birds who transmit it to mosquitoes. It is closely related to the Murray Valley encephalitis virus, although it causes a milder illness. Kunjin has been shown to cause a febrile polyarthritic illness and occasionally mild encephalitis.

Following the detection of the virus, disease prevention messages were intensified in the Leeton and Griffith areas and general practitioners were informed about the need to consider Kunjin virus when investigating suspected arboviral disease.

Flocks of chickens in selected sites across rural NSW are tested routinely in summer months to see if they have seroconverted, which would mean they had been infected with flaviviruses. No Kunjin virus activity was detected in the sentinel chickens located near Griffith and Leeton, and no acute cases of human disease due to Kunjin have been detected. This suggests that, although the virus was active in the area, the level of virus had not increased sufficiently to cause human disease.

The arbovirus season in the Greater Murray was characterised by high numbers of mosquitoes and a large number of cases of Ross River virus disease, particularly during November and December 1998. Residents are strongly advised to take appropriate personal protective measures (cover up, wear insect repellents, avoid mosquito exposures particularly at dawn and dusk) to reduce risk of infection.

For more information about the Arbovirus Surveillance and Vector Monitoring Program, visit the web site at www.arbovirus.health.nsw.gov.au.

FIGURE 3

REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, JANUARY 1994 TO FEBRUARY 1999, BY MONTH OF ONSET

These are preliminary data: case counts in recent months may increase because of reporting delays

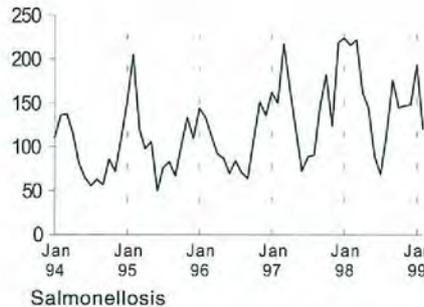
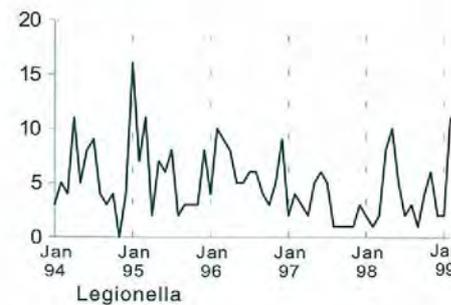
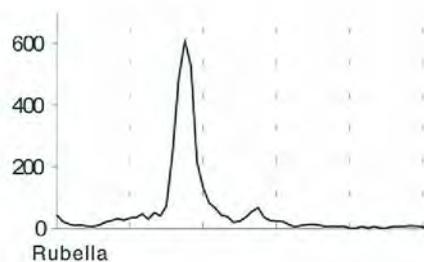
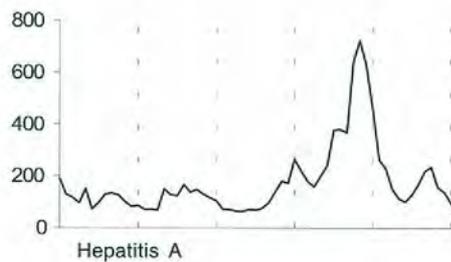
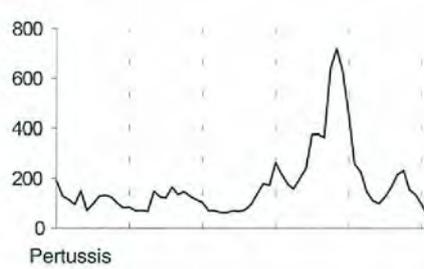
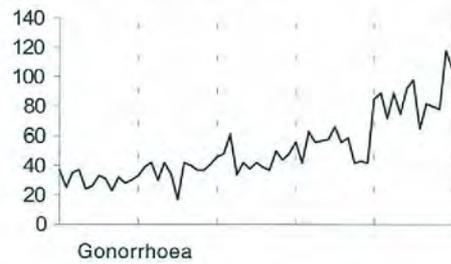
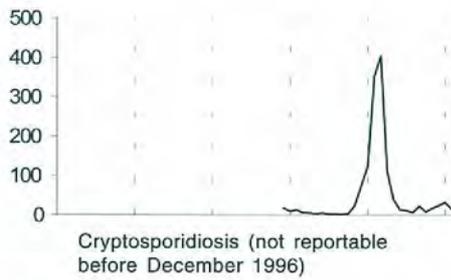
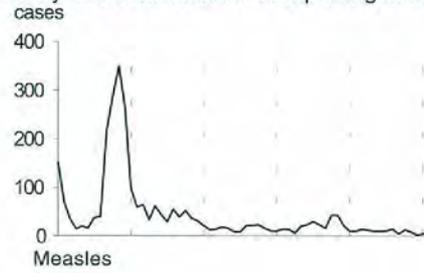
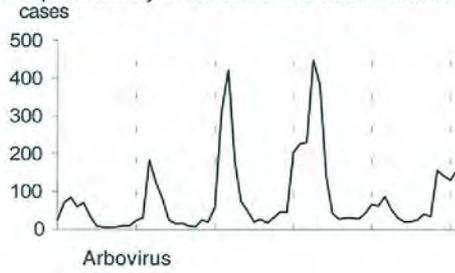


TABLE 1 INFECTIOUS DISEASE NOTIFICATIONS RECEIVED IN FEBRUARY 1999 BY AREA HEALTH SERVICES
Area Health Service (1999) Total

Condition	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	for Feb††	To date‡
Blood-borne and sexually transmitted																			
AIDS	2	1	-	-	-	-	-	-	1	2	-	-	-	-	-	1	-	9	55
HIV infection*	-	-	Notified in every				two months				-	-	-	-	-	-	-	-	16
Hepatitis B: acute viral*	-	-	-	-	-	-	-	-	2	1	-	-	1	-	-	-	-	4	9
Hepatitis B: other*	56	34	2	5	6	-	6	6	47	2	4	4	2	3	7	3	1	190	335
Hepatitis C: acute viral*	-	-	-	-	-	-	-	-	-	-	-	1	-	-	4	-	-	5	11
Hepatitis C: other*	58	42	1	47	5	-	63	18	80	36	28	10	5	25	3	28	29	486	995
Hepatitis D: unspecified*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hepatitis, acute viral (not otherwise specified)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)	11	7	-	4	-	-	20	13	37	12	3	5	-	6	3	3	3	129	275
Gonorrhoea*	22	9	-	2	3	-	-	2	41	3	2	1	-	2	5	-	-	94	208
Syphilis	6	5	-	-	-	-	3	-	11	3	1	2	1	2	5	-	-	39	70
Vector-borne																			
Arboviral infection*	4	5	4	-	2	-	21	7	2	16	22	3	10	3	13	35	8	157	300
Malaria*	2	5	-	2	1	-	-	1	5	-	-	2	-	-	-	-	-	18	34
Zoonoses																			
Brucellosis*	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	2
Leptospirosis*	-	-	-	-	-	-	1	-	-	1	1	-	-	-	-	-	-	3	8
Q fever*	-	-	-	-	-	-	-	-	1	4	2	3	2	2	-	-	-	14	23
Respiratory and other																			
Blood lead level	4	2	-	3	4	-	7	1	1	2	1	-	-	2	54	1	-	82	138
Legionnaires' disease	-	-	6	1	-	-	1	-	2	-	1	-	-	-	-	-	-	11	13
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Meningococcal infection (invasive)	-	-	2	-	1	-	1	-	2	-	-	-	1	-	-	-	-	8	26
Mycobacterial tuberculosis	4	5	6	-	2	-	-	-	5	-	-	-	-	1	-	-	-	-	2339
Mycobacteria other than TB	11	2	-	-	-	-	1	-	6	1	3	-	-	-	-	1	1	26	60
Vaccine-preventable																			
Adverse event after immunisation	-	-	-	1	-	-	1	-	-	-	1	-	-	-	-	-	-	3	18
<i>H. influenzae</i> b infection (invasive)	-	-	-	-	-	-	-	-	1	-	1	-	-	-	-	-	-	2	2
Measles	-	1	-	1	-	-	1	1	1	-	-	-	1	-	-	-	-	7	9
Mumps*	2	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	3	3
Pertussis	4	3	10	9	16	-	11	2	9	1	4	5	1	3	3	21	6	108	224
Rubella*	-	3	-	-	-	-	-	-	-	3	-	-	-	-	-	-	-	6	10
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Faecal-oral																			
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera*	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	1	1
Cryptosporidiosis	-	1	-	-	-	-	-	-	-	1	4	5	-	4	-	6	3	24	55
Giardiasis	2	19	13	5	2	-	5	4	21	10	2	4	5	-	2	3	3	100	187
Food-borne illness (not otherwise specified)	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1	1
Gastroenteritis (in and institution)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Haemolytic uraemic syndrome	-	-	-	-	-	-	-	-	2	-	-	1	-	-	-	-	-	3	3
Hepatitis A	8	6	9	1	6	-	-	3	11	-	1	-	-	1	-	2	3	53	89
Hepatitis E	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Listeriosis*	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	4
Salmonellosis (not otherwise specified)*	13	34	22	7	3	-	10	12	19	21	11	5	2	8	4	2	5	181	343
Typhoid and paratyphoid*	1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	2
Verotoxin producing <i>E. coli</i>	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

* lab-confirmed cases only

† includes cases with unknown postcode ‡ except CCA

CSA = Central Sydney Area
NSA = Northern Sydney Area
WSA = Western Sydney Area

WEN = Wentworth Area
SWS = South Western Sydney Area
CCA = Central Coast Area

HUN = Hunter Area
ILL = Illawarra Area
SES = South Eastern Sydney Area

NRA = Northern Rivers Area
MNC = North Coast Area
NEA = New England Area

MAC = Macquarie Area
MWA = Mid Western Area
FWA = Far West Area

GMA = Greater Murray Area
SA = Southern Area

HOW DOES HIV-AIDS SURVEILLANCE WORK IN NSW?

Rob Menzies

Senior Surveillance Officer
AIDS/Infectious Diseases Unit, NSW Health

Valerie Delpech

Medical Epidemiologist
South Eastern Sydney Public Health Unit

Lizzy Griggs

Surveillance Officer
AIDS/Infectious Diseases Unit, NSW Health

The monitoring of epidemiological trends in HIV-AIDS in NSW and Australia has, since 1982, provided information critical for the prevention, care and treatment of HIV disease. National surveillance is done through several mechanisms described previously,¹ the notification of cases to the Department of Health by laboratories, medical practitioners and hospitals being an important part. Australia's HIV-AIDS surveillance is among the best in the world, with notification of AIDS in all states since 1982 and of HIV since 1989. Most countries do not collect public health data on all HIV diagnoses. For example, the United States and most European countries rely on other sources, such as sentinel sites, population surveys and AIDS notifications. However, because of the success of combination therapies and the reduction in AIDS incidence, there is new emphasis internationally on implementing HIV notification.²

The documentation required of clinicians for HIV-AIDS is considerable. Notification requirements plus research studies, drug trials, S100 drug forms, etc. result in a sometimes confusing array of forms that collect similar information. The aim of this article is to clarify the system of HIV-AIDS notification in NSW.

HIV

Under the *NSW Public Health Act (1991)* persons newly diagnosed with HIV infection are notifiable by laboratories that carry out confirmatory HIV testing (reference laboratories). There are currently seven reference labs in NSW. Following confirmation of diagnosis, the reference laboratory sends a notification form to the requesting doctor to collect information including the demographics, risk exposure, previous tests and clinical status of the patient. To protect patient confidentiality, a name code is used (first two letters of the surname and given name) instead of the patient's full name. By law, medical practitioners must complete the notification form and return it to the laboratory. The information is then entered on the NSW HIV database, forwarded to the National Centre in HIV Epidemiology and Clinical Research (NCHECR) for inclusion on the National HIV database, and to the World

Health Organisation (see Figure 4). Data summaries are published in the *NSW Public Health Bulletin*, the *National HIV Surveillance Report*, and in peer review journals.³⁻⁶

Public Health Units (PHUs) follow up notifications that report risk exposures other than male-to-male sexual contact or mother-to-child transmission for more detailed assessment of risk exposure. PHU surveillance officers forward to the medical practitioner a detailed exposure assessment questionnaire which requests information on the patient's history of blood transfusions, injecting drug use and sexual exposures both in Australia and overseas. The questionnaire also enquires whether the patient would like the Department of Health to investigate the means of HIV infection. When this is requested, a comprehensive interview with the patient is carried out by appropriate staff and, where necessary, other investigations are performed. This mechanism of HIV risk exposure assessment is very important in tracking the emergence of new or unusual means of transmission, such as heterosexual contact, medical procedures or other rare exposures. Analyses of this data have been published on two occasions to date.^{7,8}

Completeness of HIV data, including risk exposure, increased dramatically from 1992 with the introduction of data collection by reference labs. However, the completeness of the data has deteriorated in recent years (see Figure 5), making the monitoring of the epidemic increasingly difficult. Paradoxically, this has also increased the paperwork load for clinicians. Where the risk exposure is not recorded, a form is forwarded to the clinician that seeks to record information describing sources of exposure (Exposure Assessment form). Most of these cases turn out to be due to male homosexual contact, and therefore the Exposure Assessment form would not have been required if the shorter laboratory notification form had been completed.

AIDS

Under the 1991 Act, AIDS is notifiable to PHUs by medical practitioners and hospital chief executive officers. The information requested is similar to that for HIV notification, with the addition of the AIDS-defining illness and the date of the last medical contact or death. As with HIV notification, a name code is used at all times. Notifications are entered on the Notifiable Disease Database at the PHU and forwarded to AIDB. AIDB forwards notifications to NCHECR for inclusion on the National AIDS Register (see Figure 6). The number of people notified as living with AIDS each year has been used as one determinant of AIDS care funding to the States by the Commonwealth Government. Therefore, a yearly audit is carried out by Area Health Services and AIDB to detect unnotified cases.

FIGURE 4

HIV NOTIFICATIONS FLOW CHART

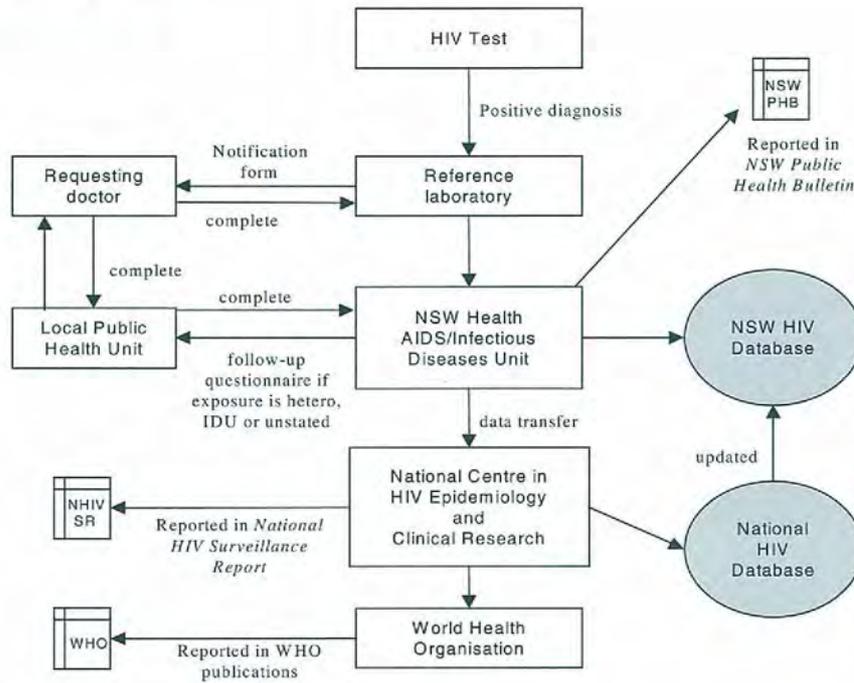
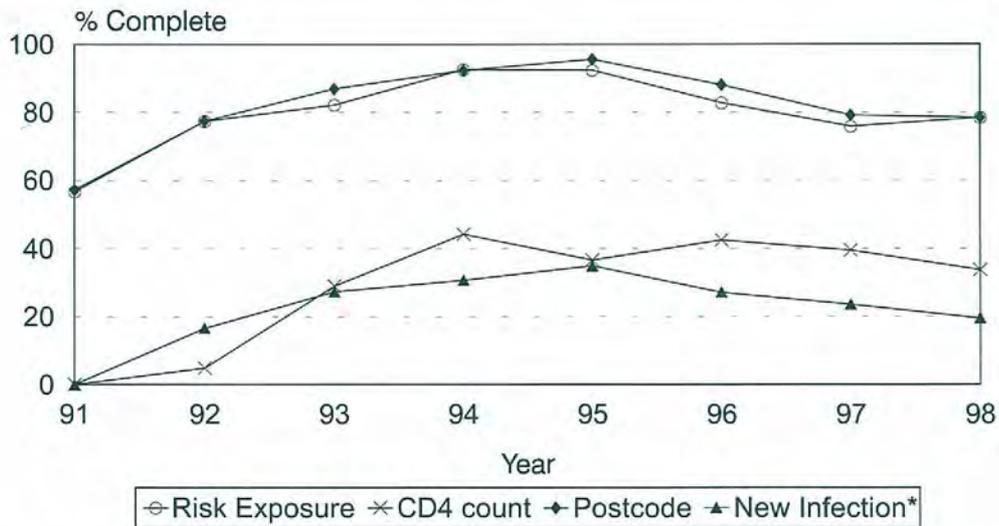


FIGURE 5

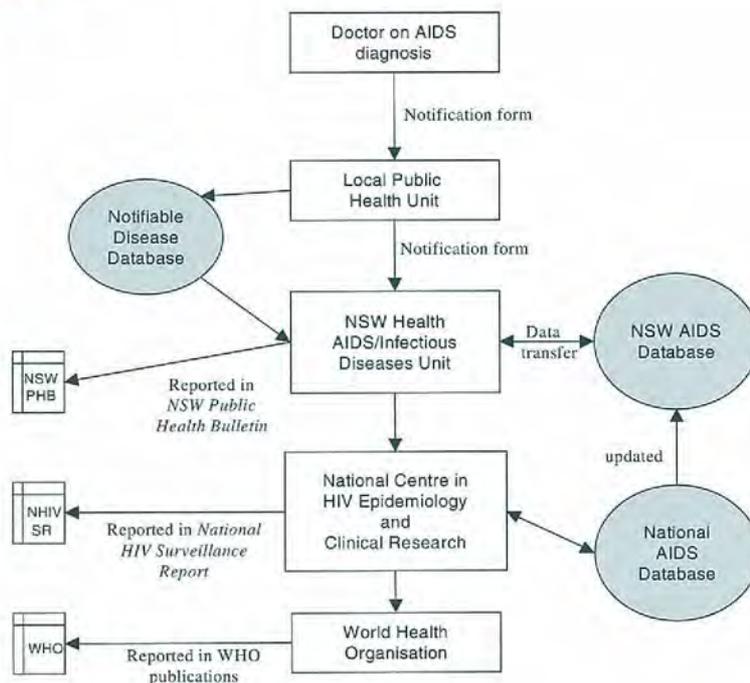
NSW HIV SURVEILLANCE DATA COMPLETENESS



* Defined as negative test within 12 months or documented seroconversion illness

FIGURE 6

AIDS NOTIFICATION FLOW CHART



The process includes cross checking the AIDS register with other data sources such as hospital inpatient data, clinic records and death registrations.

When a patient dies as a result of HIV disease, medical practitioners are requested to complete a specific notification form and forward it to their local PHU. Data on deaths are also updated using death certificates during the yearly audits.

The AIDS Register has provided a crucial means of monitoring the epidemic. Patients may be infected with HIV for many years before being diagnosed, and notified; therefore HIV notifications will underestimate the total prevalence of HIV. However, the development of AIDS almost always requires the patient to seek medical attention, which provides an opportunity for notification. As a result AIDS notifications have given the most reliable estimates of the population affected by HIV. However, as AIDS may develop many years after HIV infection this does not provide information on recent HIV transmission. In recent years, the successful introduction of combination therapies has delayed the development of AIDS in many patients indefinitely. This will result in AIDS data being used more as a measure of treatment failure than of the

natural clinical progression of HIV disease. Data from AIDS notifications are published in the *NSW Public Health Bulletin*, the *National HIV Surveillance Report* and peer reviewed journals.⁹⁻¹⁵

The AIDS case definition is outlined in the Australian National Council on AIDS Bulletin No. 18, which is available from PHUs. AIDS and AIDS death notification forms are also available from PHUs.

The authors would like to gratefully acknowledge the dedication and hard work of reference laboratory staff and clinicians in providing this information. This information gives NSW and Australia some of the best HIV–AIDS surveillance data in the world.

REFERENCES

1. Kaldor JM, Crofts N. Managing HIV. Part 8: Controlling the epidemic. 8.6 Epidemiological surveillance for HIV and AIDS. *Medical Journal of Australia* 1996; 165(5): 268–271.
2. Gostin LO, Ward JW, Baker, AC. National case reporting for the United States: A defining moment in the history of the epidemic. *New England Journal of Medicine* 1997; 337(16): 1162–1167.
3. McDonald AM, Cruickshank M, Ziegler JB, Elliott E, Kaldor

- JM. Perinatal exposure to HIV in Australia, 1982–1994. *Medical Journal of Australia* 1997; 166(2): 77–80.
4. McDonald AM, Gertig DM, Crofts N, Kaldor JM. A national surveillance system for newly acquired HIV infection in Australia. *American Journal of Public Health* 1994; 84(12): 1923–1928.
 5. McDonald AM, Crofts N, Blumer CE, et al. The pattern of diagnosed HIV infection in Australia, 1984–1992. *AIDS* 1994; 8(4): 513–519.
 6. Law MG, McDonald AM, Kaldor JM. Estimation of cumulative HIV incidence in Australia, based on national case reporting. *Australian & New Zealand Journal of Public Health* 1996; 20(2): 215–217.
 7. Raman S, Menzies RI, McDonald A, Griggs E, Levy M. Validation of reported risk exposure in persons with newly diagnosed HIV infection. *Communicable Diseases Intelligence* 1996; 20(1): 2–5.
 8. McDonald A. Assessment of reported exposure to HIV for HIV infection newly diagnosed in Australia in 1994. *Australian HIV Surveillance Report* 1995; 11(2): 9–13.
 9. Becker NG, Watson LF, Marschner IC, Motika M, Newstead SV, Carlin JB. Assessing the extent of the Australian HIV epidemic from AIDS surveillance data. *Australian Journal of Public Health* 1993; 17(3): 226–231.
 10. Li Y, Gold J, McDonald AM, Kaldor JM. Demographic pattern of AIDS in Australia, 1991 to 1993. The National HIV Surveillance Committee. *Australian & New Zealand Journal of Public Health* 1996; 20(4): 421–425.
 11. Whyte BM, Gold J, Dobson AJ, Cooper DA. Epidemiology of acquired immunodeficiency syndrome in Australia. *Medical Journal of Australia* 1987; 146(2): 65–69.
 12. Kaldor J, McDonald AM, Blumer CE, et al. The acquired immunodeficiency syndrome in Australia: Incidence 1982–1991. *Medical Journal of Australia* 1993; 158(1): 10–17.
 13. Elford J, McDonald A, Kaldor J. Kaposi's sarcoma as a sexually transmissible infection: An analysis of Australian AIDS surveillance data. *AIDS* 1993; 7(12): 1667–1671.
 14. Solomon PJ, Wilson SR. Predicting AIDS deaths and prevalence in Australia. *Medical Journal of Australia* 1992. 157(2): 121–125.
 15. Luo K, Law M, Kaldor JM, McDonald AM, Cooper DA. The role of initial AIDS-defining illness in survival following AIDS. *AIDS* 1995; 9(1): 57–63. ☒

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