

ENVIRONMENTAL CARCINOGENS IN NEW SOUTH WALES

GUEST EDITORIAL

Bernard Stewart

Cancer Control Program

South Eastern Sydney Public Health Unit

In 1974, an issue of *Newsweek* entitled 'What causes cancer?' suggested that 'At least 60 per cent, and as much as 90 per cent of human cancer is caused by environmental factors, probably chemicals'. This widely-accepted belief was a factor in establishing the United States National Toxicology Program, through which hundreds of chemicals were subjected to chronic toxicity testing in rodents. The goal was to identify those otherwise 'unknown' carcinogens that accounted for a significant proportion of human cancer.

This goal has not been realised. Rather, the intervening years have witnessed the development of a clearer understanding of what 'environmental factors' means. Such factors are now recognised to include not only a number of specific chemicals encountered in an occupational, medicinal or dietary context, but also complex mixtures encountered in foodstuffs, tobacco smoke, pollutants and workplace conditions. There is also a range of carcinogenic factors that do not readily, if at all, qualify as chemicals. These include ionising and non-ionising radiation, certain infections, chronic inflammatory states, some behaviours, reproductive status, and the competence of the immune system. All of that said, the term 'environmental carcinogen' is still often employed to categorise chemicals. It is in this limited sense that the term is used as the focus of this issue of the *NSW Public Health Bulletin*.

Implicit in the identification of any cancer-causing agent is the possibility of developing interventions for cancer prevention.

continued on page 194

CONTENTS

- 197 **Guest Editorial:**
**Environmental carcinogens
in New South Wales**
- 199 **Environmental carcinogen
control in Australia: the need
for a strategy.**
- 202 **Research and development
in carcinogen control**
- 205 **The identification,
assessment and control of
environmental carcinogens
in NSW and Australia**
- 208 **NSW Public Health Officer
Training Program: Public
Health and Risk Assessment
and Management Stream**
- 209 **Benzene: A case study of the
control of a carcinogen in
NSW**
- 212 **Can molecular epidemiology
help us better understand the
environment's role in
carcinogenesis? The
example of pesticides**
- 215 **Tobacco control in NSW:
evidence supporting
improved strategies to
reduce exposure to
environmental tobacco
smoke**
-
- 218 **FactSheet: Shigellosis**
-
- 219 **Communicable Diseases,
NSW: September 2002**
- 219 Trends
- 219 Enhanced influenza
surveillance
- 219 Syphilis reports
- 220 Focus on enteric disease

Prevention may be achieved by reducing or eliminating exposure to the agent in question. The scope for cancer preventive measures is broad, as illustrated through the following examples:

- solar radiation causes skin cancer; prevention may be achieved by avoiding intense sun exposure, taking advantage of shade, wearing protective clothing, and using sunscreens. A major challenge lies in facilitating widespread adoption of preventive behaviour and discouraging deliberate exposure (such as sunbaking);
- the brewing of beer may result in the formation of dimethylnitrosamine, a compound that is proven to be carcinogenic in a dozen animal species, including the non-human primates. It is metabolised by the human liver and by other tissues in humans. A marked reduction in the amount of dimethylnitrosamine in beer has been achieved by modifying brewing conditions.

Within the spectrum of cancer preventive measures, a recognised method of prevention is the adoption and implementation of regulatory measures; an option that often concerns (but is not restricted to) government departments and statutory authorities.

It is the prevention of cancer caused by environmental carcinogens that is addressed in the articles contained in this issue. The first three articles describe options for establishing that a hazard exists. Andrew Penman discusses the development of strategies that respond to the distribution of disease and which might lead to an increased understanding of those diseases. The means of identifying hazardous agents are considered by Bernard Stewart, while John Beard and Kathy Jong describe the means of emerging molecular epidemiological methods to elucidate the effect of pesticides. Julie Billett describes how here in NSW recognised hazards are addressed through regulatory action. In the articles that follow, two recognised hazards are discussed in detail. Benzene causes leukaemia and Julia Brotherton outlines control of exposure to this solvent by action through NSW and Commonwealth authorities. In their article, Elayne Mitchell and John Sanders concern themselves with one aspect of tobacco control, controlling exposure to tobacco smoke in the environment, and describe the NSW Tobacco Action Plan 2001–2004.

Determining which environmental carcinogens warrant regulatory action is a challenge; however, it is certain that, in specific contexts, cancer can be prevented through regulatory means. ☒

ENVIRONMENTAL CARCINOGEN CONTROL IN AUSTRALIA: THE NEED FOR A STRATEGY

Andrew Penman
Chief Executive Officer
The Cancer Council NSW

This article describes the need for a strategy for the control of environmental carcinogens in Australia, which extends from identifying causative agents through to the implementation and confirmation of measures that improve health outcomes.

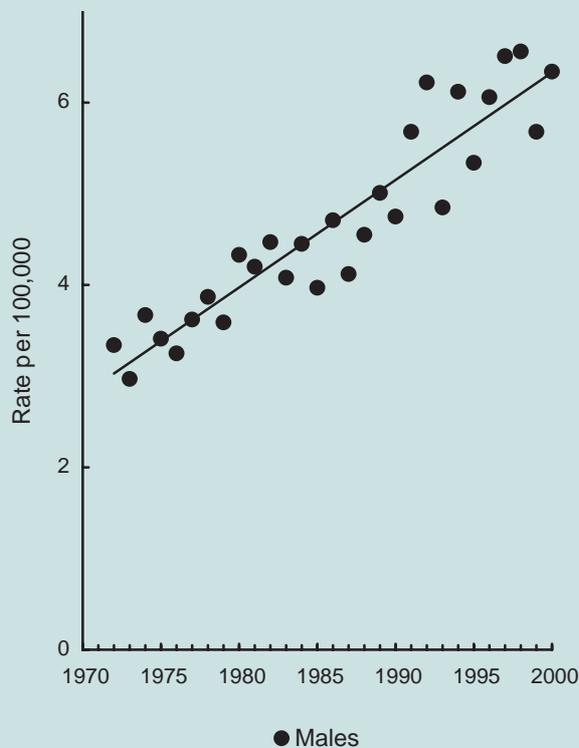
It is well accepted that, with current knowledge, many cancers are preventable by reference to a variety of risk factors.¹ Thus, smoking, over-nutrition, low intake of fruit and vegetables, sunlight exposure, lack of exercise, alcohol intake, and certain infections, all affect the burden of cancer and represent categories of risk that are avoidable to some extent. The challenges presented for control of these factors are widely recognised, often in the context of the so-called 'healthy lifestyle' and its outcome. However, in respect of cancer specifically, there are instances of individual disease, and some types of tumours generally,

which are not attributable to recognised causative agents or risk factors. Information is also limited regarding individual susceptibility, particularly in relation to genetic makeup or hormonal influences. Systems of addressing carcinogenic hazards rarely take account of these considerations, and the systems themselves are subject to marked variation. While the comprehensive regulatory approach to tobacco has been noteworthy, the degree of control for this substance may be perceived as lax by comparison to some current procedures limiting exposure to occupational asbestos, given that tobacco is one of the few substances proven to be carcinogenic in humans according to the International Agency for Research on Cancer.²

The distribution of cancer readily establishes strategic needs. Testicular cancer, non-Hodgkin's lymphoma and thyroid cancer (Figures 1 to 3) are common cancers, the incidence of which has more than doubled over 30 years.³ Asbestos use, and the consequent epidemic of mesothe-

FIGURE 1

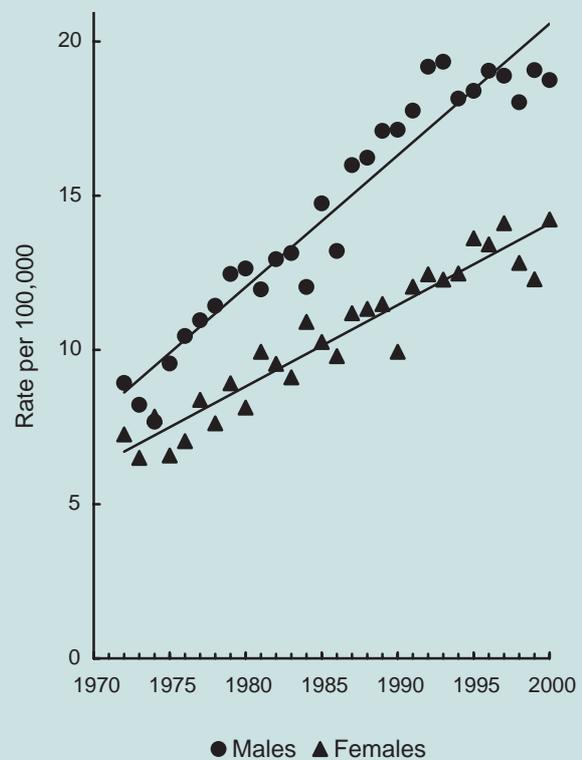
TESTICULAR CANCER, AGE-STANDARDISED INCIDENCE, NSW, 1970-2000



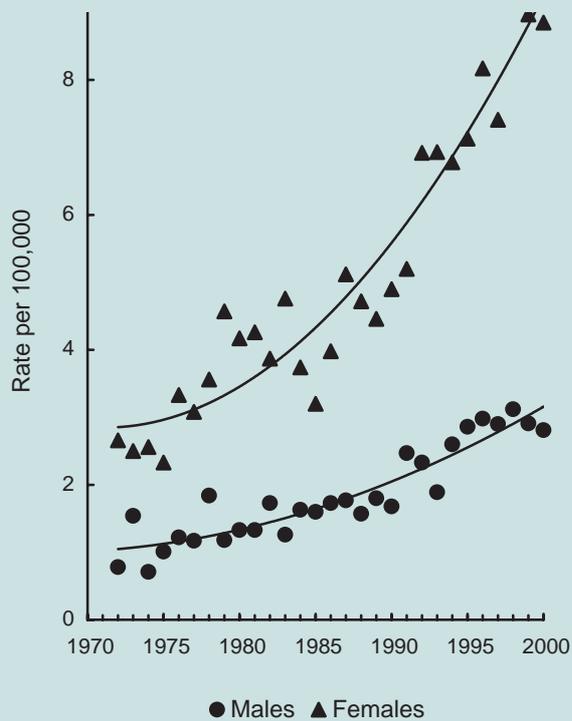
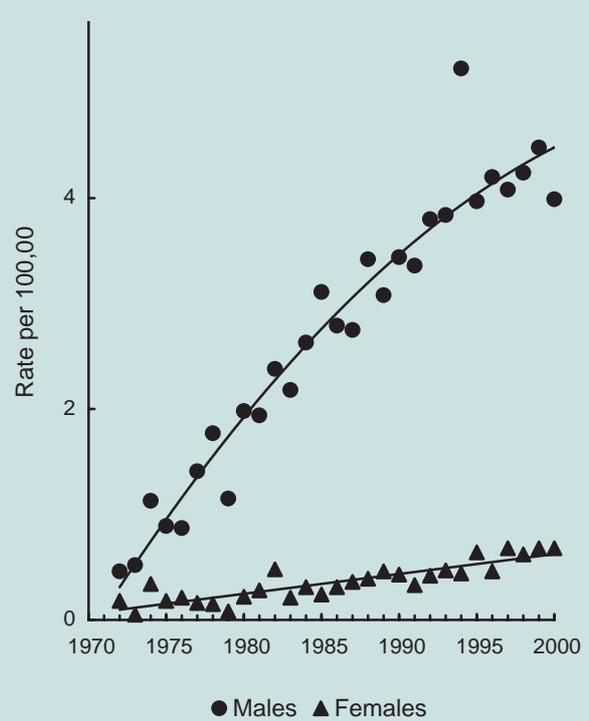
Source: The Cancer Council NSW.³

FIGURE 2

NON-HODGKIN'S LYMPHOMA, AGE-STANDARDISED INCIDENCE, NSW, 1970-2000



Source: The Cancer Council NSW.³

FIGURE 3**THYROID CANCER, AGE-STANDARDISED INCIDENCE, NSW, 1970–2000**Source: The Cancer Council NSW.³**FIGURE 4****MESOTHELIOMA, AGE-STANDARDISED INCIDENCE, NSW, 1970–2000**Source: The Cancer Council NSW.³

lioma, is another example (Figure 4). Indeed, there has been recent recognition that Australia has the highest rate of mesothelioma in the world.⁴

The conclusion that environmental factors have variously played a role in the increased incidence of these cancers is unavoidable. Also unavoidable is the inference that the science of quantitative carcinogenic risk assessment—and the considerable controls in place throughout the Western world that limit exposure to known hazards—have failed to prevent or even to predict these changes. These trends are not unique to Australia.

I venture to suggest that, if cancer were a communicable disease, we would spare little expense in funding programs of strategic research to find a cause; and that such a program would be wide-ranging, mobilising the considerable portfolio of scientific methods now available. Instead, we rely on investigator-initiated research that, in most cases, is only initiated after achieving success through fiercely competitive grant funding.

Of course, equally incisive scientific insights may be obtained from the study of positive trends in the incidence of malignant disease. Stomach cancer rates have

plummeted in Australia and elsewhere, but despite clear inferences about the reasons for this—among them, food preservation techniques and *Helicobacter Pylori* infection—other factors may still be revealed. These observations are but a few of the inferences concerning environmental carcinogenesis that can be drawn from descriptive epidemiology. Appropriate follow-up might illuminate our understanding of carcinogenesis and contribute to the refinement, focus, and development of those anticipatory controls that may be the responsibility of multiple departments within the structures of government.

Limiting or preventing exposure to environmental carcinogens is the responsibility of multiple authorities. Two decades of public sector reorganisation has seen the principal responsibility for carcinogen control fall to specific agencies (such as the National Industrial Chemical Notification and Assessment Scheme of the National Occupational Health and Safety Commission, and the National Registration Authority for Agricultural and Veterinary Chemicals), and the regulation of exposure settings assigned to environment and workplace portfolios (such as the NSW Environment Protection Authority and

WorkCover). While these changes have been for the better, specialisation has its disadvantages in terms of the development of clear, comprehensive strategies.

Currently the Commonwealth Department of Health and Ageing has no core focus on environmental carcinogens. NSW Health has a range of statutory and regulatory responsibilities, particularly in relation to the containment and to the control of environmental carcinogens. Yet there may be additional ways in which health departments can make a strategic contribution. The first of these, adequate response to population distribution of tumour types, has been considered. The second, the surveillance and assessment of particular hazards, builds on the traditional role of health departments in health surveillance, and may extend to the systematic collection and analysis of tissue specimens to demonstrate evidence of exposure.

Cancer is a preventable disease that kills prematurely, with 270,000 years of life lost (to age 75) nationwide in 1995.⁵ Cancer tops the health concerns of Australians,⁶ and the results of cancer are tragic, costly, and long lasting. There have been many proposals for a national cancer act. Certainly, in relation to environmental carcinogens, a

means is needed to harness the resources of government to address priorities and facilitate better surveillance and impact assessment. A national cancer act would be part of a national strategy for the control of environmental carcinogens in Australia.

REFERENCES

1. Peto J. Cancer epidemiology in the last century and the next decade. *Nature* 2001; 411: 390–395.
2. International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. IARC monographs may be located at <http://monographs.iarc.fr>.
3. Coates MS, Tracey EA. *Cancer in New South Wales: Incidence and mortality 1999 featuring 30 years of cancer registration*. Sydney: The Cancer Council NSW, 2001.
4. Leigh J, Davidson P, Hendrie L, Berry D. *Malignant mesothelioma in Australia, 1945–2000*. *Am J Ind Med* 2002; 41: 188–201.
5. Australian Institute of Health and Welfare. *Cancer in Australia 1991–94*. Canberra: Australian Institute of Health and Welfare, 1998.
6. Roy Morgan Polls Finding No. 3357, October 2000. ☒

RESEARCH AND DEVELOPMENT IN CARCINOGEN CONTROL

Bernard Stewart

Cancer Control Program

South Eastern Sydney Public Health Unit

There are two questions fundamental to the prevention of cancer by limiting or preventing exposure to carcinogenic agents: 'Which agents present a hazard?' and, if this is answered in the positive, 'Who, in consequence, is at risk?' Both of these questions have been the subject of research, in respect of particular substances and exposures, for more than half a century. Answers to these questions have the potential to reduce cancer-associated mortality and morbidity; however, the means of finding answers remains limited when considered against the background of progress in other fields of health research. This article describes which agents pose a carcinogenic hazard, who is at risk, and the future prospects of research in and development of carcinogen control.

WHICH AGENTS POSE A CARCINOGENIC HAZARD?

When presented with the question 'Which agents pose a carcinogenic hazard?' one assumes that the answer must involve a list. However, the answer to the question is not to be found in a list, and an understanding of why 'lists' of chemical carcinogens are a problem is fundamental to both the public health and the research aspects of carcinogenesis.

The most authoritative assessments of carcinogenicity data—the International Agency for Research on Cancer's (IARC) *Monographs on the Evaluation of Carcinogenic Risks to Humans*—arose because in the early 1970s the IARC was asked by governments from around the world to list known carcinogens. It became apparent that definitive biological criteria to generate such a list (both in respect of determining compounds to be on or off the list) were not available. Rather, the IARC initiated a program to evaluate carcinogenicity data for any given agent, using a protocol that ensured that all relevant findings were taken into account. While the *Monograph* series has given rise to 'lists' these were secondary to the individual evaluations and depend on the interpretation (sometimes disputed in individual cases) of the individual data sets.

The means of identifying carcinogens has not changed markedly over the last fifty years. During this time, understanding of the mechanism by which agents cause malignant transformation has moved from reference to tumours in particular animals to the structure and effect of altered gene sequences.² Operationally, knowledge has only marginally altered the generalisation that evidence

of carcinogenicity is drawn from appropriately designed epidemiological studies and testing of chronic toxicity in animals. Insight regarding a chemical of unknown biological potential can be gained using 'short term tests' for carcinogenicity, most commonly based on mutations of specifically-developed ultrasensitive strains of bacteria; or otherwise involving mutation or transformation of mammalian cells in culture.³ Tests are generally based on simulating the metabolism of carcinogens so that reactive intermediate products, capable of becoming bound to DNA, are formed in the presence of sensitive bacteria or other 'indicator' populations. While occupying a vital niche, for example, in toxicological evaluation of new drugs, short term test data are supplementary to epidemiological and animal testing data with respect to agents to which humans are already exposed. Finally, it must be acknowledged that, for the majority of specific chemicals, reliance is placed on animal studies, since the occurrence of human exposure to the agent in question—say, a specific pesticide—at high concentration and in the absence of other compounds, is rare.

Despite these generalizations, which concern all carcinogens, data for each compound must be considered on its merits. In some instances the findings are clear: for example, 1,3-butadiene, tris(2,3-dibromo-propyl) phosphate and 2,4-diaminotoluene present a carcinogenic hazard to humans and their use in children's sleepware and in hair dyes has been controlled; likewise sodium fluoride is not carcinogenic and its addition to water supplies is therefore appropriate.

Carcinogenicity data for other compounds, however, are far from clear, and there are plenty of examples that indicate that once relevant studies have been completed, an understanding (and a basis for action) does not necessarily follow. For example, exposure to trichloroethylene is associated with an increased risk of tumours at different sites, or with no increased risk, depending on the occupational context studied;⁴ causation of lymphoma by chloro-phenoxy herbicides may be inferred from agricultural and forestry work findings, but studies based on exposure to these compounds have generally failed to confirm this hypothesis, and dioxin (2,3,7,8-tetrachlorodibenzodioxin) appears to increase risk of cancer generally without being characterised as causing a particular tumour type. In all instances, the corresponding experimental data do not clarify the picture.

Apart from short-term tests, research has not contributed greatly to the assessment of putative carcinogenic hazards. Regarding electromagnetic fields, research data are

unhelpful.⁵ In this context, as in the testing of chemicals, it was supposed that artificially transferring genetic material from one animal species to another might provide an improved vehicle for chronic testing, but this hope has yet to be realised. And, for the purpose of elucidating specific mechanisms that account for increased risk of cancer in people occupationally exposed to complex mixtures of agents, novel effective methodologies have not emerged.

WHO IS AT RISK?

Regarding exposure to a specified carcinogen, the facile answer to the question 'Who is at risk?' is "Whoever is exposed". The dimensions of the category 'exposed' have increased markedly through the achievements of research. Such research addresses limitations inherent in the use of broad indicators to identify persons at increased risk by comparison with a wider comparative population. Thus the populations exposed to toxins produced by the fungus *Aspergillus flavus* (aflatoxins) growing on peanuts and maize, which causes liver disease (and especially cancer of the liver), are those living in tropical Africa and Asia.

Sometimes the carcinogen under consideration implicitly suggests who is at risk: painters and paint manufacturers are exposed to paint solvents; asbestos workers are exposed to asbestos. However, the limitations of such statements are well recognised: administrative staff at a paint factory may never come into contact with paint, while demolition workers (rather than asbestos workers) may have the highest exposure to asbestos.

Notwithstanding their limitations, broad categories will continue to usefully identify persons at risk. Thus, accumulation of lipophilic pesticides in breast milk may result in the newborn being exposed to relatively high concentrations of carcinogens: a scenario meriting intervention without waiting for direct evidence of harm. Other broad categories of individuals at risk include those who are immunocompromised by, for example, HIV infection or the administration of immune-suppressing drugs.

Finally, a separate body of evidence indicates that not all circumstances of carcinogen exposure result in increased risk. So far as is known, cigarette smokers receive 10 times the amount of benzene as non-smokers,⁶ but do not appear to suffer a commensurate increased risk of leukaemia.⁷ Ingestion of water containing asbestos derived from piping does not appear to present the hazard posed by respired asbestos. Indeed, the current reporting of a recognised carcinogen such as acrylamide, which is produced in some foods prepared at a high temperature, may be characterised as alarmist. However, if a new route of exposure can identify a higher risk than previously

recognised, publicity of relevant observations is justified. Research has contributed little in this context.

Quantitation of individual exposure to many carcinogens is assessable. Elucidation of relevant metabolic pathways has allowed detection of indicative compounds in body fluids. Much more significantly, and subject to on-going study, patterns of mutation attributable to specific carcinogens mark the interface between exposure and mechanistic analysis. Thus, patterns of mutation attributable to aflatoxin, benzo[a]pyrene, or ultraviolet radiation, not only indicate that a relevant exposure has occurred but provide insight into the mechanism of cancer causation⁸. Mutation is the commonest specific genetic alteration exhibited in human malignancy. Such insight is gained from studying tumours, and does not provide any simple immediate means of prevention. However, some progress is being made on the use of genetic information to indicate people at risk from environmental carcinogens. Intense effort has been directed toward the relationship between carcinogen metabolism (assessed genotypically or phenotypically) and risk of malignancy. Differences in risk are sometimes indicated, but variation is not so marked as to have public health implications.⁹

FUTURE PROSPECTS

In common with virtually every area of medical science, understanding of carcinogenesis is certain to be affected by advances in molecular genetics: we are in the postgenomic era. The recent publication *Cancer Cell* epitomises the focus of molecular analysis on the biology of malignancy.¹⁰ Discovery of a 'new gene' that is crucial is unlikely. But the capacity of available technology, such as microarrays (that is, matrices in which cDNA corresponding to 10,000 or more individual genes are 'arrayed' so that the expression of each gene may be evaluated relative to expression of the same gene in some reference context) to assess thousands rather than one or two genes on a single analysis, may change beyond recognition the identification of people at risk from a specific hazard. At the level of public health policy, the exploitation of chemoprevention—whether based on pharmaceuticals or micronutrients—has a limited history but continues to provide an opportunity for action outside the frame of simply 'preventing exposure'.¹¹

CONCLUSION

Environmental factors that influence cancer are known to include diet, certain infections and some behaviours. Nonetheless, causation of cancer by specific substances has been, and will remain, a singular opportunity to prevent malignancy. Hopefully, the design and implementation of such preventive measures will continue to be assisted by progress in research.

REFERENCES

1. International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. IARC monographs may be located at <http://monographs.iarc.fr>.
2. Yuspa SH. Overview of carcinogenesis: past, present and future. *Carcinogenesis* 2000; 21: 341–4.
3. Trosko JE. Challenge to the simple paradigm that 'carcinogens' are 'mutagens' and to the in vitro and in vivo assays used to test the paradigm. *Mutat Res* 1997; 373: 245–9.
4. Stewart BW. Trichloroethylene and cancer: A carcinogen on trial. *Med J Aust* 2001; 174: 244–7.
5. Anderson LE, Morris JE, Sasser LB, Loscher W. Effects of 50- or 60-hertz, 100 microT magnetic field exposure in the DMBA mammary cancer model in Sprague-Dawley rats: Possible explanations for different results from two laboratories. *Environ Health Perspect* 2000; 108: 797–802.
6. Wallace L. Environmental exposure to benzene: An update. *Environ Health Perspect* 1996; 104(S-6): 1129–36.
7. Doll R. Cancers weakly related to smoking. *Br Med Bull* 1996; 52: 35–49.
8. Hainaut P, Hollstein M. p53 and human cancer: The first ten thousand mutations. *Adv Cancer Res* 2000; 77: 81–137.
9. Mucci LA, Wedren S, Tamimi RM, Trichopoulos D, Adami HO. The role of gene-environment interaction in the aetiology of human cancer: Examples from cancers of the large bowel, lung, and breast. *J Intern Med* 2001; 249: 477–93.
10. Klausner RD. The fabric of cancer cell biology—weaving together the strands. *Cancer Cell* 2002; 1: 3–10.
11. Principles of chemoprevention. Stewart BW, McGregor D and Kleihues P (editors). *IARC Scientific Publication No. 139*. Lyon: IARC, 2002. ☒

THE IDENTIFICATION, ASSESSMENT AND CONTROL OF ENVIRONMENTAL CARCINOGENS IN NSW AND AUSTRALIA

Julie Billett

The Cancer Council NSW

Within NSW and Australia, a number of agencies are engaged in a wide range of activities—and in deploying a variety of tools—directed towards controlling and managing exposure to environmental carcinogens in industrial, domestic, and agricultural settings, and in the environment. In 2001, The Cancer Council NSW undertook a mapping exercise to establish ‘who does what’, with respect to the control of environmental carcinogens. The objectives of this exercise were to map out the functions and responsibilities of key state and national organisations involved in the control of environmental carcinogens; to describe the underpinning legislative and regulatory framework; and to identify possible weaknesses and limitations that may exist in the management of environmental carcinogens. This article describes the picture of environmental carcinogen control that emerged from this mapping exercise, which is a picture of a complex array of organisations—working at state and national levels—each with overlapping functions and responsibilities.

Based on current knowledge, over 200 substances, agents, or mixtures are known—or are reasonably anticipated to be—carcinogenic to humans.^{1,2} For the purpose of this exercise, we limited our focus to those chemical and physical agents and substances that are amenable to direct and socially-acceptable control measures, thereby excluding behavioural risk factors for cancer such as diet and physical activity.

Fifteen agencies involved in the control of environmental carcinogens were identified (Table 1). Relevant individuals from each agency were contacted and invited to participate in a semi-structured interview. Information obtained from these interviews was supplemented with information gathered from websites, annual reports, and other literature. In addition, semi-structured interviews were conducted with a second set of organisations with an interest or expertise in environmental carcinogens, including universities, consumer and environmental groups, trade unions, and associations of public and environmental health professionals.

WHO DOES WHAT?

The infrastructure and activities for the identification, assessment, and control of environmental carcinogens in NSW and Australia is complex. Responsibility is dispersed among multiple agencies, which reflects both the diversity of the substances and agents involved, and the range of

settings and routes through which potential exposures could occur. For the majority of agencies involved, their concern with carcinogens is part of a much broader responsibility for environmental and/or public health protection. None, with the possible exception of the Australian Radiation Protection and Nuclear Science Agency (ARPANSA), has a direct focus on carcinogen control per se; nor, unsurprisingly, is there any apparent sense of a community of organisations jointly responsible for environmental carcinogen control. There is also evidence of significant overlap in the organisational responsibilities of the key state and national agencies, with additional complexities imposed by Australia’s three-tiered system of government at the Commonwealth, state, and local levels.

Taking the two examples of radiation and (carcinogenic) atmospheric contaminants, Table 2 lists those agencies that have an organisational responsibility that includes the assessment, monitoring and/or control of these carcinogens, and provides an indication of the extent of each agency’s involvement.

The instruments and measures employed by each of the 15 key agencies for the control of environmental carcinogens reflect the nature of each agency’s particular responsibility, as well as any regulatory powers invested

TABLE 1

FIFTEEN KEY FEDERAL AND STATE AGENCIES RESPONSIBLE FOR THE CONTROL OF ENVIRONMENTAL CARCINOGENS

Australian Nuclear Science and Technology Organisation (ANSTO)
Australia and New Zealand Food Authority (ANZFA)
Australian Radiation Protection and Nuclear Science Agency (ARPANSA)
Commonwealth Department of Health and Aged Care, Environmental Health Section
Therapeutic Goods Administration (TGA)
Environment Australia
National Occupational Health and Safety Commission (NOHSC)
National Industrial Chemicals Notification and Assessment Scheme (NICNAS)
National Registration Authority for Agricultural and Veterinary Chemicals (NRA)
NSW Agriculture
NSW Environmental Protection Authority (NSW EPA)
NSW Health, Environmental Health Unit
Roads and Traffic Authority NSW (RTA NSW)
Sydney Water Corporation
WorkCover NSW

by underpinning legislation. A broad spectrum of approaches to control is represented including: the development of policy and guidelines; the setting of standards; the provision of expert advice to government, industry and the public; education, training and information; research, surveillance and monitoring; and the enforcement of regulatory and economic controls.

Besides the 15 agencies involved directly in the mapping exercise, participants identified other agencies with responsibilities that are directly relevant to the control of environmental carcinogens. These include: at the federal level, the National Drugs and Poisons Schedule Committee, the National Health and Medical Research Council and Agriculture, Fisheries and Forestries Australia; and at the state level, Planning NSW, Waste Services NSW, and local government.

LEGISLATIVE AND REGULATORY FRAMEWORK

The legislative and regulatory framework underpinning carcinogen control in NSW and Australia mirrors the complex picture of overlapping organisational responsibilities described above. This complexity is due to Australia's three-tiered system of government with numerous fields of regulation that have a bearing on carcinogen control. These include regulation in: environmental protection, occupational health and safety, transport and storage of waste, contaminated sites, radiation and nuclear safety, food, tobacco, public health, agricultural and industrial chemicals, urban planning, and land use. Although the Commonwealth Government has some important national responsibilities, particularly with respect to chemicals, therapeutics, food standards, tobacco, and radiation, legislation at the state and territory level gives those jurisdictions extensive responsibilities for managing and controlling exposures to environmental carcinogens in the home, at work, on the farm, and in the

environment. A convenient way of differentiating Commonwealth responsibilities from those of the states and territories is that the Commonwealth tends to deal with 'threshold' questions, through activities such as national standard setting and the registration of chemicals. The states and territories tend to regulate the application and/or use of chemicals in places and activities governed by relevant legislation. Local government also has responsibilities in the areas of waste disposal and land use and planning that can potentially affect environmental carcinogen control.

As with other public health issues, Australia's jurisdictions have endeavoured to achieve a degree of uniformity in the laws affecting environmental carcinogen control.³ Approaches to legislative uniformity in this area can be thought of as ranging along a continuum, from unitary pieces of Commonwealth legislation that provide for centralised, national controls, such as the *Commonwealth Agricultural and Veterinary Chemicals Act 1994*, to more cooperative approaches that enable states and territories to legislate for their own codes of practice and other measures, and which are developed through a process of collaboration and consultation with the Commonwealth.

LIMITATIONS AND WEAKNESSES

All participants in the mapping exercise were asked to highlight any perceived limitations and weaknesses within existing arrangements for environmental carcinogen control in Australia and NSW. A high degree of consistency was evident in the issues raised, despite the diversity of organisational perspectives. The common themes that emerged are summarised below.

System complexity and multiple jurisdictions

The complexity of the current system of carcinogen controls in NSW and Australia, and the multiple jurisdictions and agencies that contribute, engenders occasional gaps and duplications, and leads to variations

TABLE 2

ORGANISATIONAL RESPONSIBILITIES FOR THE PREVENTION AND CONTROL OF SELECTED ENVIRONMENTAL CARCINOGENS IN NSW AND AUSTRALIA

| Environmental carcinogen | Core business | Within organisational responsibility but not core business |
|---|--|--|
| Atmospheric contaminants, including air toxics and diesel exhaust | <ul style="list-style-type: none"> • Environment Australia • NSW EPA (ambient only) • NSW Department of Health (Environmental Health Branch) • RTA NSW | <ul style="list-style-type: none"> • ANSTO • ARPANSA • Commonwealth Department of Health and Ageing • NSW Agriculture • TGA |
| Radiation | <ul style="list-style-type: none"> • ANSTO • ARPANSA • NSW EPA | <ul style="list-style-type: none"> • Commonwealth Department of Health and Ageing • NSW Department of Health (Environmental Health Branch) • NOHSC • WorkCover NSW |

Note: See Table 1 for names in full.

in approach to the control of carcinogens, particularly in those areas where nationally uniform legislative controls are lacking, such as radiation. Achieving effective communication and coordination between the relevant agencies is perceived as vital to effective carcinogen control, yet is also regarded as a major challenge by the agencies involved. Nationally, a number of cooperative mechanisms have been established in recent years—such as the EnHealth Council, the National Public Health Partnership, and the Chemicals Clearing House—largely as a response to this challenge of improving coordination and communication. Increasingly, agencies operating at the state level see the need to develop more formalised, structured channels for inter-agency working, rather than relying on ad-hoc partnerships on specific projects. Participants also flagged the need for more ‘holistic’ whole-of-government approaches to environmental and public health policy development. The management of environmental carcinogens and other important environmental health issues requires recognition of their inter-sectoral and interdependent nature, and the development of approaches and structures that support and reinforce joint working. Transport, environmental health, and public health were highlighted by participants as policy areas in which there are clear contradictions and tensions between policy objectives.

Current controls are largely reactive rather than proactive

New environmental and public health issues are emerging all the time. Although some disease control programs have an element of active surveillance, these tend to focus on acute health problems rather than health outcomes that may result from long-term exposures such as cancers. Participants highlighted a lack of long-term surveillance programs linking environmental and occupational exposures to health outcomes data. Participants also underlined the importance of anticipating changes in the environment, which may predicate new or increased exposures to carcinogens, as a means of facilitating a more precautionary and proactive approach to carcinogen control. These environmental changes may be qualitative in nature; for example, changes to fuel formulations or engine design; or may be quantitative in nature, such as the increase in absolute numbers of diesel vehicles on our roads.

Specific limitations in the control of chemicals

Under current arrangements for chemical control, existing chemicals are not subject to the same regulatory scrutiny as new chemicals. While the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) and the National Registration Authority for Agricultural and Veterinary Chemicals (NRA) both maintain review schemes for existing chemicals, under which these are

reviewed on a priority basis, such schemes only scratch the surface of the thousands of industrial and agricultural chemicals already available for use in Australia. Moreover, there is scepticism among consumers about an assessment process that relies on industry-generated data, and evaluates chemicals on a one-by-one basis, rather than examining the health effects of the total ‘chemical load’ to which we are exposed. Also of concern is the difficulty of regulating and controlling the use of chemicals in the domestic environment. Reviews commissioned by the National Health and Medical Research Council (NHMRC) have highlighted widespread ignorance of chemical hazards in the home.⁴

Regulatory compliance in occupational settings

It has been estimated that in excess of 1250 deaths occur each year in Australia as a result of occupational exposure to carcinogens.⁵ Yet relatively little is known about the numbers of people exposed to carcinogenic or potentially carcinogenic substances in the workplace, and the extent and distribution of occupational cancer in Australia. Serious concerns were expressed by participants about the degree of compliance with occupational health and safety regulations and standards governing exposure to hazardous substances, including carcinogens. Several participants highlighted failures or inadequacies in implementing controls for a range of carcinogenic substances, including asbestos, trichloroethylene, and benzene. Itinerant or casual workers employed in small-scale enterprises are thought to be at greatest risk of hazardous exposures. Moreover, the resources available to oversee compliance with occupational health and safety standards are seen as inadequate by the key agencies consulted for this exercise.

Information and skills deficit

One of the most straightforward themes to emerge was the deficiency of scientific information on many actual and potential carcinogenic hazards. This deficiency was thought to span the range of scientific evidence necessary to undertake robust health risk assessments, from studies of carcinogenesis at the biological and molecular level, to human epidemiological studies of environmental and occupational exposures. Improving our understanding of the toxicity of chemicals and their effect on human and environmental health was seen as a key challenge, as were long-term studies of environmental exposures and an improved understanding of the synergistic effects of particular exposures. This deficiency of knowledge severely limits the ability of organisations to undertake health risk assessments, and to provide robust evidence-based policy advice and recommendations necessary to protect public health. Interviewees also observed a skills shortage in environmental and public health risk assessment, and particularly in relation to the scarcity of toxicological expertise available to public health units across NSW.

Risk perception and communication

At a societal level, perceptions of risk drive government policy and ultimately affect the nature of regulatory and other control mechanisms. There is significant public concern about the health risks posed by modern environmental health issues such as environmental pollution, ozone depletion, and chemical exposures.⁶ Yet we have only a limited understanding of environmental risk perception, risk thresholds, risk tolerability, and their determinants in an Australian setting. Improved understanding of public perceptions of environmental and behavioural risk factors, the determinants of those perceptions, and influences over the acceptability or tolerability of risk, would aid environmental and public health practitioners to communicate risk in a meaningful way to the public, and to develop targeted prevention strategies grounded in the evidence from behavioural research.

CONCLUSIONS

The picture of carcinogen control that emerged from this mapping exercise is of a complex array of organisations working at state and federal levels, each with overlapping responsibilities that include the direct or indirect control of environmental carcinogens. The legislative framework is similarly complex in NSW and Australia. Approaches to joint working on multi-sectoral carcinogen control issues range from formalised cooperative mechanisms to ad-hoc partnerships on specific projects. While no sense emerged of major gaps or holes in the network of controls dealing with established carcinogens, this mapping exercise highlighted some key challenges facing policy makers and practitioners working in the field. Those key challenges and areas for action can be grouped under four

broad themes: coordination and cooperation in carcinogen control at national and state levels; enforcement of and compliance with existing controls; information exchange, communication and public engagement; and active management of emerging risks. On a positive note, the key state and national stakeholders consulted saw considerable scope for addressing many of the limitations within existing carcinogen control arrangements, and have demonstrated a commitment to furthering this aspect of cancer control by actively engaging in a strategic program of work being carried forward under the auspices of The Cancer Council NSW.

REFERENCES

1. National Toxicology Program. *Ninth Report on Carcinogens*. Research Triangle Park, North Carolina: United States Department of Health and Human Services, 2000.
2. International Agency for Research on Cancer. *Monographs series on the evaluation of carcinogenic risks to humans*. Lyons, France: IARC, 1972–2001.
3. Centre for Comparative Constitutional Studies, University of Melbourne. *Implementation Options for National Legislative Schemes in Public Health*. National Public Health Partnership website at www.dhs.vic.gov.au/nphp/legtools/options.
4. National Health and Medical Research Council. *A National Plan for Domestic Chemicals*. Canberra: AGPS, 1991.
5. Morrell S, Kerr C, Driscoll T, et al. Best estimate of the magnitude of mortality due to occupational exposure to hazardous substances. *Occup Environ Med* 1998; 55: 634–64.
6. Starr G, Langley A, Taylor A. *Environmental health risk perception in Australia: A research report to the Commonwealth Department of Health and Aged Care*. Centre for Population Studies and Epidemiology, South Australian Department of Human Services. Canberra: AusInfo, 2000. ☒

NSW PUBLIC HEALTH OFFICER TRAINING PROGRAM: PUBLIC HEALTH AND RISK ASSESSMENT AND MANAGEMENT STREAM

In 2003, the NSW Public Health Officer Training Program will be offering a stream of training in public health risk assessment and management, in addition to its existing generalist and drug and alcohol streams.

The public health risk assessment and management stream is a response to the growing need for people with risk assessment skills, not only in public health emergencies but also for application in environmental and health impact assessments.

For further information about this stream contact the Public Health Training and Development Branch on (02) 9391 9204.

BENZENE: A CASE STUDY OF THE CONTROL OF A CARCINOGEN IN NSW

Julia Brotherton

NSW Public Health Officer Training Program

Even when a hazard, such as a carcinogen, is well established, reducing exposure to the hazard through regulation may be a complex process and involve multiple stakeholders working together. It is the health policy maker's job to put the issue, as a potential threat to health, on the agenda. This article describes a case study that illustrates the complexity of attempting to regulate known carcinogens, using benzene control in NSW as an example.

OCCURRENCE OF BENZENE IN THE ENVIRONMENT

Benzene is a volatile naturally occurring organic compound (C₆H₆). It is found in fossil fuels and is produced by the burning of organic material and, as such, occurs in fires, petrol refining, fumes from cooking oils, tobacco smoke, and waste incineration. Benzene is also formed, even during the combustion of lead free petrol, in car engines. Historically, benzene was widely used as a solvent, and is still used in the manufacture of plastics, synthetic fibres, detergents, pharmaceuticals, pesticides, and rubber. Low-level benzene exposure is ubiquitous, and the main route of exposure is through inhalation. Fortunately, benzene in the air is broken down naturally by chemical reactions over a period of hours to days.

THE CARCINOGENICITY OF BENZENE

Benzene is classified as a definite (that is, a Group 1) human carcinogen by the International Agency for Research on Cancer,^{1,2} and is a genotoxic substance that causes mutations in DNA. The National Occupational Health and Safety Commission has also classified benzene as a Category 1 carcinogen (that is, an established human carcinogen). Information about the carcinogenicity of benzene comes from animal studies, and from occupational cohort studies of workers who have been occupationally-exposed to high cumulative doses of benzene in previous decades (for example, shoe making, leather, rubber, and chemical industry workers.) In humans, there is clear evidence of a relationship between exposure to benzene and the development of acute myelocytic leukaemia (AML), which is a cancer of the white blood cells. The risk of leukaemia increases with exposure, with no known threshold but with a significantly elevated risk above 50 parts per million-years—for example, 1.25 parts per million (ppm) (time weighted average eight-hour exposure [TWA₈]) over 40 years).³ Benzene exposure is also associated with multiple myeloma and non-Hodgkins lymphoma. Locally, the Health Watch study, a long-term

cohort study of Australian petroleum industry workers, has reported an excess of lympho-haematopoietic cancers (that is, cancers of the blood and bone marrow cells), with a case-control analysis demonstrating that high cumulative exposures to benzene are associated with these cancers in this cohort.⁴

BENZENE EXPOSURE IN AUSTRALIA

Most exposure to benzene in the Australian population occurs through the air—indoors, inside vehicles, and outdoors—when the atmosphere is locally-contaminated with benzene from vehicle exhaust, petrol evaporation, and tobacco smoke.³ The estimated excess lifetime risk of leukaemia for the average urban Australian, due to the estimated 24-hour average lifetime exposure of 5.2 parts per billion, is one per 10,000 population, or 1.2 per cent of the lifetime risk of contracting leukaemia of any cause.³ In NSW, the lifetime risk of leukaemia by age 75 is one in 92 for men (1.1 per cent) and one in 162 for women (0.6 per cent).⁵

There are several industries in Australia where higher than ambient levels of benzene exposure could occur—such as the petroleum, steel and chemical industries—and in laboratories. Additionally workplaces where conspicuous exposures to vehicle exhaust or tobacco smoke occur—such as those of professional drivers, mechanics, or hospitality industry workers—need to be considered.

The control of benzene exposure in NSW is approached through three main avenues: as a poison, as an occupational hazard, and as an environmental pollutant (in air, water, tobacco smoke, and contaminated sites.)

CONTROL AS A POISON

The *Commonwealth Therapeutic Goods Act 1989* provides a framework for the states and territories to adopt a uniform approach to control the availability and accessibility, and ensure the safe handling, of poisons in Australia. Benzene is listed as a Schedule 7 poison under the 'Standard for the Uniform Scheduling of Drugs and Poisons' (the Standard). Schedule 7 substances are those with a high potential for harm at low exposure requiring special precautions during manufacturing, handling, and use. Products with benzene in concentrations below 1.5 per cent or in petrol up to 5.0 per cent are exempted from the poisons schedule. The legislation and schedule classification restrict access to benzene to authorised users.

In NSW, direct reference to the Standard is made in the *NSW Poisons and Therapeutic Goods Act 1966*, which means that poisons in NSW legislation are updated by

Commonwealth standards. The Pharmaceutical Services Branch of the NSW Department of Health administers the NSW Act. Unless one is an authorised person (doctor, dentist, vet, pharmacist, or holder of a manufacturer–wholesaler–general supplier’s licence), authority must be obtained from the Branch in order to obtain benzene.

CONTROL AS AN OCCUPATIONAL HAZARD

The National Occupational Health and Safety Commission is a tripartite statutory body with representation from government, employers, and employees. The Commission has the power to declare national occupational health and safety standards and codes of practice, but these need to be adopted into law at a state and territory level. National codes of practice relevant to benzene include the Control of Scheduled Carcinogenic Substances,⁶ with benzene under Schedule 2 (Notifiable), and Workplace Hazardous Substances.⁷ Benzene is included in the List of Designated Hazardous Substances [NOHSC:10005(1999)] as a Category 1 carcinogen.⁸ The Commission has also produced *Guidelines for health surveillance for benzene (1997)*.⁹

Since 1990, the occupational exposure standard for benzene has been 5 ppm. This standard is considered inadequate, given current knowledge about the hazard of benzene and is currently being reviewed with a view to lowering the standard to 1 ppm.¹⁰ Current Australian workplace exposures are estimated to be considerably lower than 5 ppm (mean long term exposures of <0.7 ppm across relevant industries).³ Substitution of benzene, engineering controls, ensuring adequate ventilation, and safe work practices such as personal protective equipment, are the main methods of workplace control of benzene exposure.

The review of the exposure standard is occurring, in the wake of a recent assessment of benzene as a priority existing chemical by the National Industrial Chemicals Notification and Assessment Scheme (NICNAS).³ Although NICNAS is not a statutory authority—that is, it cannot regulate—it can make recommendations as a statutory scheme and has a memorandum of understanding with states and territories.

NICNAS conducts its reviews in conjunction with Environment Australia and the Therapeutic Goods Administration—under whose auspices the scheme has recently come—and brings recommendations to the relevant parties for action.

In NSW, the Workcover Authority of NSW is responsible for ensuring compliance with occupational health and safety legislation. The *NSW Occupational Health and Safety Act 2000* places the onus on employers to conduct risk assessments of hazardous substances in the workplace, and thus they are responsible for identifying hazards,

disclosing them to workers, providing safety instructions, and labelling. In relation to benzene, its use as the main raw material in the manufacture of a product (>50 per cent volume) is notifiable and benzene cannot be supplied unless Workcover is notified or unless the benzene is used for research or analysis. Workcover requires employers to provide health surveillance for employees where indicated by risk assessment, to keep appropriate records, and to notify employees of any exposure or potential exposure at the end of employment. The use of greater than one per cent benzene for spray painting is specifically prohibited.

CONTROL AS AN ENVIRONMENTAL EXPOSURE

Lead agencies involved in measuring, monitoring and reducing benzene in the environment are Environment Australia and the NSW Environment Protection Authority (EPA). ‘Upstream’ control measures include new national fuel standards to reduce benzene emissions by over 50 per cent nationally by 2010, by reducing benzene in petrol to a maximum of one per cent by volume from January 2006.¹¹ The delay in introducing these standards reflects concerns regarding infrastructure costs to refineries. Industrial benzene emissions are reportable to the National Pollutant Inventory, with a summary of pollutants available publicly.¹² As part of the proposed National Environment Protection Measure for Air Toxics, an air standard for benzene in Australia is being considered. The NSW EPA has reported that air benzene levels in NSW comply with international air quality targets.¹³ The NSW Government’s 25 year Action for Air plan aims to provide an integrated strategy for improving air quality, with the NSW EPA working locally with initiatives such as regulations to reduce wood heater emissions, control of open burning and incineration, and working with the oil industry to supply low volatility petrol during summer. However, improving air quality clearly requires cross-sectoral action and involves agencies such as Planning NSW (transport and road infrastructure), the Roads and Traffic Authority, and the Environmental Health Branch of the NSW Department of Health.

Public health campaigns to reduce smoking have not focused on benzene, which is just one of many carcinogens in tobacco smoke. The success of tobacco control interventions in NSW has undoubtedly reduced population benzene exposure, both in smokers and non-smokers. In particular the *NSW Smoke Free Environment Act 2000*, which bans smoking in most enclosed public places, will reduce exposure to benzene from environmental tobacco smoke.

CONCLUSIONS

Population and subpopulation exposure to benzene is difficult to control comprehensively. This is because

exposure is ubiquitous and occurs in multiple settings, and because responsibilities for control vary according to these settings. Particular problems with the control of benzene in Australia relate to a lack of timeliness in occupational standard setting, to the multiplicity of small workplaces where significant (and unmeasured) exposures may be occurring (such as in petrol stations and car repair shops) and to insufficient data about low dose population exposures and the degree of such exposure in Australia. Concern over benzene in air pollution and in cigarette smoke has not been the driving force behind the control of these issues, and it is fortunate that these problems continue to be addressed for other reasons. The current initiatives, as part of Environment Australia's *Living Cities—Air Toxics Program*, to measure benzene exposures more accurately at the population and subpopulation level may facilitate focused control measures where they are most needed.

ACKNOWLEDGEMENTS

Thank you to the people consulted in preparing this overview including Vicky Sheppard, Graham Cox, Paul Byleveld, Stuart Clarke, Rich Harvey, Alan Yee, Jo McClellan, and Carolyn Vickers. In particular I would like to thank Margaret Hartley, Deborah Wilcocks, and Robert Kenyon for their helpful feedback.

Further Reading

The interested reader is referred to the recommendations of the recent report by the National Industrial Chemical Notification and Assessment Scheme,³ and to a recent report by the European Environment Agency,¹⁴ which uses benzene as a case study of failure to regulate appropriately in the face of significant evidence of harm.

REFERENCES

1. IARC. Benzene. *Some industrial chemicals and dyestuffs. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans*. Lyon, International Agency for Research on Cancer, 1982; 29: 93–148.
2. IARC. Benzene. *Overall evaluations of carcinogenicity: An updating of selected IARC Monographs from Volumes 1 to 42. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans*. Lyon, International Agency for Research on Cancer, 1987; Supplement 7: 120–122.
3. NICNAS. *Benzene Priority Existing Chemical Report No. 21*. Canberra: National Industrial Chemical Notification and Assessment Scheme, Commonwealth of Australia, 2001.
4. Monash University and Deakin University. *Lympho-haematopoietic cancer and exposure to benzene in the Australian petroleum industry: Technical report and appendices June 2001*. Available on-line at www.aip.com.au/pdf/case_study.pdf.
5. Tracey EA, Supramaniam R. *Cancer in New South Wales: Incidence and mortality 2000: Featuring projections to 2010 for selected sites*. Sydney: The Cancer Council NSW, 2002.
6. National Occupational Health and Safety Commission. *National Model Regulations for the Control of Scheduled Carcinogenic Substances. Control of Workplace Hazardous Substances, Part 2—Scheduled Carcinogenic Substances: National Model Regulations and National Code of Practice*. Canberra: Australian Government Publishing Service, 1995.
7. National Occupational Health and Safety Commission. *National Model Regulations for the Control of Workplace Hazardous Substances. National Model Regulations and National Code of Practice*. Canberra: Australian Government Publishing Service, 1994.
8. National Occupational Health and Safety Commission. *List of Designated Hazardous Substances, 2nd Edition*. Canberra: Australian Government Publishing Service, 1999.
9. National Occupational Health and Safety Commission. *Guidelines for Health Surveillance for Benzene*. 1997. Available at www.nohsc.gov.au/ohsinformation/nohscpublications/fulltext/docs/h4/01997ben.htm#guidelines.
10. National Occupational Health and Safety Commission. *Proposed amendment to the National Exposure Standard for Benzene. Public comment paper*. Commonwealth of Australia, 31 May 2002. Available at www.nohsc.gov.au/OHSInformation/Databases/ExposureStandards/expsearch.asp.
11. The Honourable Dr David Kemp MP, Federal Minister for the Environment and Heritage. *New fuel standards for cleaner air and better health* [media release]. 30 December 2001. Available at www.ea.gov.au/minister/env/2001/mr30dec01.html.
12. Environment Australia. National Pollutant Inventory. Available at www.npi.gov.au.
13. Environmental Protection Authority NSW. *NSW State of the Environment 2000 Report*. Sydney, 2001. Available at www.epa.nsw.gov.au.
14. European Environment Agency. *Late lessons from early warnings: The precautionary principle 1896–2000*. Environmental issue report No 22. Copenhagen: European Environment Agency, 2001. Available at www.eea.eu.int. ☒

CAN MOLECULAR EPIDEMIOLOGY HELP US BETTER UNDERSTAND THE ENVIRONMENT'S ROLE IN CARCINOGENESIS? THE EXAMPLE OF PESTICIDES

*John Beard and Kathy Jong
Department of Rural Health
University of Sydney, Northern Rivers*

Pesticides are widely dispersed in the environment and exposure to them is almost unavoidable, mainly through the food chain. During the peak period of its use, DDT was so ubiquitous that it could be detected in ice core samples taken in the Antarctic, even though it had never been used on that continent. Pesticides have been one of the most intensely studied of possible carcinogens in the environment. As with other environmental exposures, epidemiological research into the health effects of chronic pesticide exposure is subject to methodological challenges, and our understanding of the relationship between exposure and health remains limited. This article describes some of the challenges facing environmental epidemiology, and some of the recent developments in molecular epidemiology that may assist these challenges. To assist the reader, a glossary of terms is provided toward the end of the article.

SOME CURRENT CHALLENGES IN ENVIRONMENTAL EPIDEMIOLOGY

One of the challenges facing environmental epidemiology is getting accurate information on pesticide exposure. This challenge is exacerbated by the initiation and latency periods that are usually associated with cancer, which means that there may be lag periods of more than 10 years

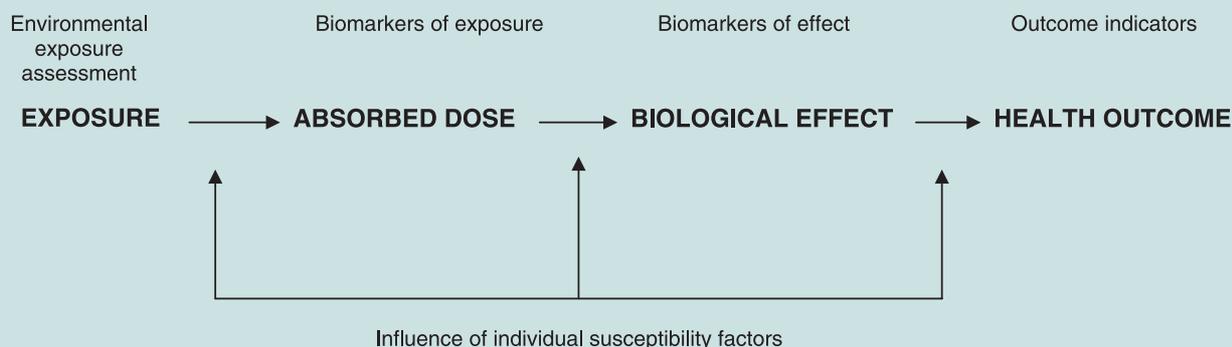
between an exposure and a particular outcome of that exposure. Any prospective study that looks at the health effects of a current exposure will necessarily involve long follow-up periods, even if the current exposure can be accurately determined, and any retrospective study will need to determine the effects of exposures many years in the past. Where data on exposure is dependent on the recall of those individuals subject to the exposure, measurement errors—in the form of 'recall bias'—are also likely.

Numerous surveys have been taken of pesticide levels in air, water, and food. However, because individuals vary in their behaviour—in relation to air, water, and food—it is difficult to extrapolate survey data to estimate individual exposure levels in a community setting. Also, exposures in air, water, and food are likely to be low, particularly since the 1960s when restrictions on the use of 'persistent' pesticides (that is, pesticides that persist in the environment) became widespread in western societies. At the levels likely to be faced in these communities, epidemiological studies would need to demonstrate large increases in carcinogenic risk to confidently identify an association between a particular pesticide and a form of cancer.

To overcome these difficulties, researchers have frequently turned to occupational settings to explore the relationship between exposure and health, since occupational exposures are likely to be higher and more predictable. However, even in an occupational setting, pesticide

FIGURE 1

THE EVENTS THAT OCCUR FROM EXPOSURE TO ENVIRONMENTALLY INDUCED DISEASE AND THEIR RELATIONSHIP WITH BIOMARKERS OF EXPOSURE, EFFECT AND SUSCEPTIBILITY *



*After Fowle & Sexton.²

exposure tends to be difficult to assess because the users of pesticides rarely have standardised work practices.

Information on individual exposures for pesticide users is also often limited. To overcome this, researchers have often turned to simple occupational categorisation to define exposure groups. However, to be most effective, such an approach requires homogeneity in the exposures likely to be experienced by individuals identified in each category. Where, for example, categories such as 'farm hand' are identified from census or other routinely-collected documents, heterogeneity of exposure lessens the ability of these studies to detect true associations.

One frequently-used method of getting more accurate information on individual exposure in occupational settings is biological monitoring. Biological sampling for persistent pesticides such as the organochlorines, or for contaminant pesticides such as dioxins, can give a meaningful picture of total exposure over a number of years. However, metabolism and excretion of modern pesticides is rapid, and results of biological monitoring may only reflect recent exposure in the last few hours or days.

Some of the issues around the assessment of pesticide exposure were examined by a review of studies of the possible effects of Agent Orange, which was used by American military personnel during the Vietnam War.¹ Until 1992, the assessment of exposure relied on categorisation of individuals into occupational groups that were thought likely to have worked with Agent Orange. Sometimes this assessment was supplemented by an individual's own estimate of the exposure. However, in 1992 the United States Air Force completed a study examining the relationship between individual serum TCDD (the dioxin contaminant of Agent Orange) and verified reproductive outcomes. TCDD levels correlated poorly with both self-reported exposure and exposure indices developed from military records, which confirms the limitations of research dependant on these surrogate measures and the need for a degree of scepticism when interpreting the findings.

Another challenge for epidemiological studies exploring the health impact of pesticides is the 'healthy worker effect', where relatively healthy individuals tend to be more likely to gain employment and remain employed. This effect has the potential to bias studies towards finding lower mortality rates in an occupational cohort, when compared with the general community, and thus mask true increases in mortality. When studying the impact of pesticides, the 'healthy worker effect' may be complicated by the unique dietary and lifestyle factors associated with residing and working on a farm, which is associated with mortality and cancer rates below those of the broader community.

Finally, a range of other factors may confound the relationship between pesticide exposure and cancer mortality. These possibly include smoking, carcinogenic animal viruses, and the lymphoproliferative effect of prolonged antigenic stimulus. Our understanding of these risk factors is currently limited and inconsistent.

SOME RECENT DEVELOPMENTS IN MOLECULAR EPIDEMIOLOGY

Recent developments in the field of molecular epidemiology may assist the challenges facing environmental epidemiology, by providing better information on exposure and earlier information on outcomes, and by identifying members of the community who may be most sensitive to exposures to pesticide. A framework for applying these recent developments is outlined in Figure 1, which is based on the work of Fowle and Sexton.²

Biomarkers of exposure

Accurate assessment of exposure to potential environmental carcinogens will contribute to the accuracy of studies, and will reduce the number of subjects required to identify possible health effects. As mentioned above, chemicals that persist in the environment can already be measured directly in body tissues (sometimes at considerable expense). However, direct measurement of pesticides that are rapidly metabolised, such as organophosphates, is less useful; and the surrogate measures currently used, for example, serum cholinesterase levels, only provide a crude indication of exposure.

These problems may be partly overcome if other exposure-specific patterns of physiological or chromosomal effect could be identified. For example, xenobiotic-specific DNA adduct formation (complexes that form when a chemical binds to a biological molecule such as DNA) has been demonstrated to correlate with exposure to a number of toxic compounds including polycyclic aromatic hydrocarbons and nitrosamines. While there is still more work to be done in this area, discovery of exposure-specific DNA adducts will significantly improve our ability to accurately estimate exposure to various environmental carcinogens. Unfortunately, adducts linked to specific pesticides have not yet been identified.

Measures of biological effect

Most prospective studies of environmental exposures have relied on crude and relatively rare measures of biological effect such as mortality or cancer. One of the early characteristics of carcinogenesis is genetic damage. By using such damage as an intermediate indicator of outcome, molecular epidemiology may allow shorter follow-up periods and smaller study sizes. Indicators that have so far been linked to both outcome and pesticide exposure include chromosomal aberrations, sister

chromatid exchange, and micronuclei in peripheral lymphocytes.

Exposure-susceptibility interactions

One of the most exciting new approaches allows a better understanding of individual variation and to identify populations at risk. Researchers already control for a

GLOSSARY OF TERMS

Lymphoproliferative effect of prolonged antigenic stimulus

The capacity of a chronic exposure to cause human white blood cells to proliferate in an uncontrolled way which can be a precursor to tumour development and growth.

Xenobiotic-specific DNA adduct formation

Toxins found in the human body but not produced by the human body. In susceptible individuals, xenobiotics can bind to DNA to form adducts that may lead to mutation and ultimately to cancer.

Polycyclic aromatic hydrocarbons (PAH)

Environmental carcinogens found commonly in tobacco smoke, outdoor air from automobile exhaust and emissions from power plants and other industrial sources.

Nitrosamines

Carcinogens formed from the reaction of amines (amino acids for example) with nitrite. Nitrite is commonly added to foods such as bacon, ham and sausages to inhibit the growth of harmful bacteria.

Sister chromatid exchanges (SCE)

The reciprocal interchanges of the two arms within a single chromosome. Assays of SCE in human blood (peripheral blood lymphocytes) can be used as a marker of chromosome damage.

Micronuclei in peripheral lymphocytes (MN)

Chromosome fragments or whole chromosomes left behind during the normal process of cellular division (mitosis). Assays of MN from human blood (peripheral blood lymphocytes) can be used as a measure of chromosome breakage and chromosome loss.

While assays of SCE and MN indicate chromosomal damage in cells, they do not reflect exposure to any specific chemical. However they are probably the best validated predictors of cancer risk and have thus been used as biomarker 'end points' in many epidemiologic studies.

number of key causes of variability: for example age, ethnicity, and gender. Molecular epidemiology may also soon allow for improved assessment of other non-genetic factors such as smoking, for example by the use of PAH adducts as markers of past exposure.

A number of genetic susceptibility factors are also worthy of incorporation into studies of environmental carcinogenesis, in particular the relatively common genetic polymorphisms that determine the metabolic fate of pesticides. Metabolism of most pesticides is undertaken in a two stage hepatic process and/or by serum paraoxonase. Polymorphisms of the enzymes used in these processes are common. Subjects with less effective metabolic phenotypes may be expected to face a greater internal dose following exposure to a particular pesticide and thus be more susceptible to any adverse effect. Identifying susceptible individuals and studying them separately increases the chance of an epidemiological study identifying a true association.

Such molecular approaches have already been used in a number of studies exploring the carcinogenic potential of pesticides. Typically, these studies have been small and have explored the relationship of quantified exposure to intermediate indicators.^{3,4} More recently they have also examined variations in these relationships between subjects with different metabolic phenotypes.^{4,5}

CONCLUSION

While the findings of these early studies in molecular epidemiology have been inconsistent they suggest that, as we become more familiar with the techniques, we will be better equipped to understand the role pesticides and other environmental exposures play in carcinogenesis.

REFERENCES

1. Wolfe WH, Michalek JE, Miner JC, et al. Paternal serum dioxin and reproductive outcomes among veterans of Operation Ranch Hand. *Epidemiology* 1995; (1): 17–22.
2. Fowle JR and Sexton K. EPA Priorities for Biologic Markers Research in Environmental Health. *Env Health Perspect* 1992; 98: 235–41.
3. Zeljezic D and Garaj-Vrhovac V. Chromosomal aberrations and single cell gel electrophoresis (Comet) assay in the longitudinal risk assessment of occupational exposure to pesticides. *Mutagenesis* 2001 4: 359–63.
4. Au WW, Sierra-Torres CH, Cajas-Salazar N, et al. Cytogenic effects from Exposure to Mixed Pesticides and the Influence from Genetic Susceptibility. *Env Health Perspect* 1999; 107: 501–05.
5. Cherry N, Mackness M, Durrington P et al. Paraoxonase (PON1) polymorphisms in farmers attributing ill health to sheep dip. *The Lancet* 2002; 359: 763–64. 

TOBACCO CONTROL IN NSW : EVIDENCE SUPPORTING IMPROVED STRATEGIES TO REDUCE EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

Elayne Mitchell and John Sanders

Tobacco and Health Branch

NSW Department of Health

Environmental tobacco smoke (ETS) is a mixture of sidestream smoke (emitted to the atmosphere from the tip of a burning cigarette) and mainstream smoke (inhaled and exhaled by a smoker). ETS contains at least 50 chemicals recognised to be carcinogenic, as well as thousands of other chemicals including many known to be developmental, reproductive, mutagenic, and cardiac toxins.^{1,2,3} Tobacco smoke is classified as a Group A carcinogen by the United States Environmental Protection Authority,⁴ and is classified as a Group 1 carcinogen (that is, a carcinogen that has been proven to cause cancer in humans) by the International Agency for Research on Cancer.²¹ Many compounds found in ETS are banned or regulated as occupational carcinogens, including arsenic, benzene, and vinyl chloride.³ The already extensive evidence of harm to the health of non-smokers who are exposed to ETS continues to mount. Reviews of the epidemiological evidence consistently confirm an association between exposure to ETS and passive smoking and the development of lung cancer, heart disease, and respiratory illness in non-smokers.^{1,5,6,7,8,9}

NSW has comprehensive legislation in place to minimise ETS-related harm in the community. The *Occupational Health and Safety Act 2000* requires all employers to ensure the health, safety, and welfare of their employees, consequently preventing smoking in workplaces. The *Smoke-Free Environment Act 2000*, which banned smoking in enclosed public places in NSW, was a major step forward in public health and reduced ETS exposure in restaurants, shopping centres, and other public places. Since September 2001, all table service dining areas of licensed hotels, licensed nightclubs, and registered clubs, have also been required to be smoke-free. This article presents some of the evidence that supports improved strategies to reduce exposure to environmental tobacco smoke. It also includes a description of the NSW Tobacco Action Plan (page 217),¹⁰ which outlines further areas for action to ensure that no-one is still exposed to ETS in the workplace.

AN ESTABLISHED HAZARD

A meta-analysis of the occupational risk to non-smokers of lung cancer from ETS concluded that there is a significant excess risk from occupational exposure to ETS.⁷ A review of hospitality employee exposure to ETS estimated that bar workers are exposed to 4.4 times the ETS experienced in domestic settings.¹¹ Cotinine, a chemical that is made by the body from nicotine, is a reliable biochemical marker of exposure to nicotine.

Measurement of cotinine levels in non-smokers is a valid and objective indicator of passive exposure to tobacco smoke, which permits estimation of risks to health. A recent New Zealand study of the exposure of hospitality workers to environmental tobacco smoke reported a clear association between changes in cotinine concentration during work and the workplace smoking policy.¹² Workers in premises permitting customer smoking reported a higher prevalence of respiratory symptoms and had higher concentrations of cotinine in saliva, compared to those from smoke-free workplaces. Further research on cotinine levels in non-smoking hospitality workers in NSW is needed.

In many settings throughout the world, rather than prohibiting smoking throughout premises, authorities have specified that smoking should not occur in particular places within these premises. This policy is attractive to some who perceive a prohibition on smoking to be a liability. However, evidence suggests that the protection provided by this approach is not comprehensive. An analysis by Repace et al. based on average number of smokers, number of cigarettes smoked, and room size, determined that ETS cannot be controlled by ventilation, air cleaning, or spacial separation.³ To achieve the minimum risk using ventilation alone would require, they concluded: 'in excess of one hundred thousand cubic feet per minute per occupant, which would need tornado-like levels of air-flow to achieve'.³ Consequently, attempts to separate indoor areas in hospitality venues into 'smoking' and 'non-smoking' areas are rarely effective, due to inherent difficulties in preventing the spread of smoke through air-conditioning and doorways.

INADEQUATE ACTION

In NSW, over recent years, a number of clubs and some hotels have voluntarily introduced smoke-free areas and auditoriums, and this has contributed to a reduction in ETS exposure for patrons and staff in those areas. Patrons can choose to utilise the smoke-free areas, but this same choice is not extended to employees who have to work in smoking sections. These workers may be exposed to high levels of ETS, if there is a concentration of smokers in areas where smoking is permitted. In 1999, a random sample of managers of registered clubs in NSW was surveyed regarding the smoking restrictions in clubs. Fifty-nine per cent reported that most or all of their staff were exposed to tobacco smoke at work, 43 per cent reported having no smoke-free areas, and more than two-thirds reported being concerned about litigation by patrons or staff for damage to health caused by passive smoking.¹³

The finding in the case of *Marlene Sharp v. Port Kembla RSL Club* represents an important international precedent as the first case of cancer of the larynx proven at law to be attributed to passive smoking in the workplace.¹⁴ It would be reasonable for the community to expect—in the face of the mounting medical, scientific, and legal evidence—that clubs and hotels would move swiftly toward voluntary self-regulation. However, in the more than 18 months since the court decision, the industry's voluntary progress toward smoke-free licensed premises has been piecemeal. Australian employers whose workers continue to be regularly exposed to tobacco smoke in their workplace must seriously consider the potential consequences of inaction, as permitting smoking indoors leaves employers exposed to potential litigation.

COMMUNITY SUPPORT

A recent National Drug Strategy Household Survey estimates that the vast majority (>80 per cent) of the Australian population are non-smokers.¹⁵ In the 1997 NSW Health Survey, respondents were asked about their attitude toward smoking in registered clubs, hotels, and bars. Over 90 per cent of respondents stated that smoking should either not be allowed or should only be allowed in special areas in registered clubs, and almost 85 per cent believed the same should apply to hotels and bars.¹⁶ The tobacco industry has engaged in intentional misrepresentation of the scientific evidence around passive smoking,^{17,18} by actively promoting fear of economic consequences for the hospitality industry as a result of tougher restrictions on smoking. International evidence refutes this claim, with numerous studies confirming community support for smoking restrictions, and that patronage and revenues were either maintained or increased.^{3,5,19,20}

CONCLUSION

There is overwhelming evidence of the risks to health caused by exposure to ETS. There is continuing involuntary workplace exposure to ETS among employees of licensed premises. A high level of support exists in the community for the introduction of further restrictions on smoking in clubs and hotels. There is no evidence to support tobacco company predictions regarding the economic outcomes for the hospitality industry as a result of further restrictions. The hospitality industry, the unions, and the community, have an opportunity to work together to ensure that no worker experiences involuntary ongoing exposure to tobacco smoke in their workplace, or has to make a choice to risk their health to earn a living. From a public health perspective, the elimination of exposure to ETS is achievable.

REFERENCES

1. National Health and Medical Research Council *The Health Effects of Passive Smoking—A Scientific Information Paper*. Canberra: Commonwealth of Australia, 1997.
2. Skolnick A. Draft of report on second hand smoke released. *JAMA* 1997; 277(13): 1026–1027.

3. Repace J, Kawachi I, Glantz S. *UICC Fact sheet on secondhand smoke*. Proceedings, 2nd European Conference on Tobacco or Health, Canary Islands 23–27 February 1999.
4. United States Environmental Protection Agency. *Respiratory health effects of passive smoking: Lung cancer and other disorders*. Publication EPA/600/6-90/006F. Washington DC: United States EPA, 1992.
5. The Cancer Council NSW. *When smoke gets in your eyes ... nose, throat, lungs, and bloodstream. A guide to passive smoking and the law in NSW*. Sydney The Cancer Council NSW, 2001.
6. Taylor R, Cumming R, Woodward A, Black M. Passive smoking and lung cancer: A cumulative meta-analysis. *Aus N Z J Public Health* 2001; 25(3): 203–211.
7. Brown KG. Lung cancer and environmental tobacco smoke: Occupational risk to non-smokers. *Environ Health Perspect* 1999; 107(S-6): 879–83.
8. Zhong L, Goldberg MS, Parent ME, Hanley JA. Exposure to environmental tobacco smoke and the risk of lung cancer: A meta analysis. *Lung Cancer* 2000; 27(1): 3–18.
9. He J, Vupputuri S, Allen K, Prerost M, Hughes J, Whelton G. Passive smoking and the risk of coronary heart disease—A meta-analysis of epidemiologic studies. *N Engl J Med* 1999; 340(12): 920–926.
10. NSW Department of Health. *NSW Tobacco Action Plan 2001–2004*. Sydney: NSW Department of Health, 2001.
11. Seigel M. Involuntary smoking in the restaurant workplace: A review of employee exposure and health effects. *JAMA* 1993; 270(4): 490–493.
12. Bates MN, Fawcett J, Dickson S, Berezowski R, Garret N. Exposure of hospitality workers to environmental tobacco smoke. *Tobacco Control* 2002 11(2): 125–9.
13. Muscatello D, Rissel C, Ward J. Smoking restrictions in New South Wales registered clubs: Current status and factors associated with high levels of restriction. *Health Promotion Journal of Australia* 2002; 13(1): 39–43.
14. Stewart B, Semmler P. *Sharp v. Port Kembla RSL club: Establishing causation of laryngeal cancer by environmental tobacco smoke*. *Med J Aust* 2002; 176(3): 113–116.
15. Australian Institute of Health and Welfare 2001. National Drug Strategy Household Survey Drug Statistics Series No. 9. Available at www.aihw.gov.au/publications/phe/ndshs01/index.html.
16. Epidemiology and Surveillance Branch. *NSW Health Surveys 1997 and 1998 (HOIST)*. Sydney: NSW Department of Health, 2000.
17. Hirschhorn N, Bialous S. Second hand smoke and risk assessment: What was in it for the tobacco industry? *Tobacco Control* 2001; 10: 375–382.
18. Dearlove JV, Bialous SA, Glantz SA. Tobacco industry manipulation of the hospitality industry to maintain smoking in public places. *Tobacco Control* 2002; 11(2): 94–104.
19. Chapman S, Borland R, Lal A. Has the ban on smoking in New South Wales restaurants worked? A comparison of restaurants in Sydney and Melbourne. *Med J Aus* 2001; 174(10):512–5.
20. Scollo M, Lal A. Summary of Studies Assessing the Economic Impact of Smoke-Free Policies in the Hospitality Industry. Melbourne: VicHealth Centre for Tobacco Control, 2002. Available online at [www.vctc.org.au/tc-res/Hospitality summary.pdf](http://www.vctc.org.au/tc-res/Hospitality%20summary.pdf).
21. International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. IARC monographs may be located at <http://monographs.iarc.fr>. ☒

THE NSW TOBACCO ACTION PLAN 2001–2004

The NSW Tobacco Action Plan 2001–2004 sets out the NSW Government's commitment to the prevention and reduction of tobacco-related harm in New South Wales. The Plan follows the successful NSW Tobacco and Health Strategy 1995–1999. It features a comprehensive range of strategies developed in response to the National Tobacco Strategy 1999–2003, which establishes a coordinated response for tobacco control by state, territory, and Commonwealth governments throughout Australia.

The goal of the Plan is to improve the health of the people of NSW by eliminating or reducing their exposure to tobacco in all its forms. The main objectives of the Plan are to:

- prevent the uptake of tobacco use in non-smokers, especially children and young people;
- reduce the number of users of tobacco products;
- reduce exposure to tobacco smoke;
- decrease the number of deaths and level of disease caused by smoking;
- decrease the economic cost of tobacco related illness.

The Plan has six areas of focus, which are in line with the key strategy areas of the National Tobacco Strategy, outlined below:

Focus 1: Community awareness and education

- implementation of public education campaigns
- establishment of the NSW Tobacco Control Network
- staff education and training

Focus 2: Smoking cessation

- continuation and enhancement of *Quitline* services
- training of health professionals
- incorporating routine delivery of treatment of nicotine dependence into patient care

Focus 3: Availability and supply of tobacco products

- continuation of Tobacco Sales to Minors prevention program
- enhanced enforcement of tobacco related legislation
- review of the tobacco control provisions of the *NSW Public Health Act 1991*

Focus 4: Marketing and promotion of tobacco

- implementation of tobacco advertising legislation

Focus 5: Tobacco product regulation

- collaboration with the Commonwealth government to develop a framework for the regulation of tobacco and nicotine products

Focus 6: Exposure to environmental tobacco smoke

- implementation of smoke-free public places legislation and the NSW Health Smoke Free Workplace Policy
- strategies to address exposure to environmental tobacco smoke in children.

Reducing smoking rates in the community will reduce death, disease, and associated health costs, and is therefore a major priority within the Plan. Specific strategies to target priority areas and population groups will be implemented. These population groups are:

- children and young people;
- Aboriginal and Torres Strait Islander communities;
- non-English speaking background communities with high smoking rates;
- people with a mental illness.

For further information about the NSW Tobacco Action Plan 2001–2004, contact the Tobacco and Health Branch on telephone (02) 9391 9111. The Branch is responsible for the statewide coordination of a range of programs aimed at reducing the harm associated with tobacco use. The Plan can be downloaded from the NSW Department of Health website at www.health.nsw.gov.au. Printed copies can be obtained by contacting the Better Health Centre on (02) 9879 0443.

For further information and support with quitting smoking, call the *Quitline* on 131 848 for the cost of a local call from anywhere in NSW. *Quitline* is a free 24 hour service.

S H I G E L L O S I S

WHAT IS SHIGELLOSIS?

Shigellosis is a bacterial disease caused by infection with *Shigella* bacteria. Shigellosis can affect anyone; however, children, people with poor immune systems, and the elderly are at the greatest risk.

HOW DO YOU CATCH SHIGELLOSIS?

Shigellosis is passed from person to person by the faecal–oral route, by direct or indirect contact with faecal material. This commonly occurs if hands are not washed properly, particularly after going to the toilet or changing nappies, and as a result of sexual contact. *Shigella* infections may also be acquired from eating food contaminated with the bacteria. Flies can also carry *Shigella* and can contaminate food.

A person can have *Shigella* present in their stools for some weeks and remain asymptomatic, and still pass on infection to others.

WHAT ARE THE SYMPTOMS?

Infection with *Shigella* usually results in diarrhoea, fever, nausea, vomiting, and stomach cramps. The stool may often have blood or mucus in it. The symptoms begin 1–7 days (usually 1–3 days) after exposure. Symptoms usually last 4–7 days but sometimes longer.

HOW IS SHIGELLOSIS DIAGNOSED?

Diagnosis of shigellosis requires the isolation of *Shigella* bacteria from a stool specimen. Your doctor may order this test.

WHAT IS THE TREATMENT FOR SHIGELLOSIS?

People with mild infections will usually recover without treatment. Drinking increased amounts of fluid is important to avoid dehydration. Young children (particularly infants) are susceptible to dehydration from diarrhoea, and parents

should seek medical attention. Antibiotics including ampicillin, trimethoprim–sulfamethoxazole and ciprofloxacin, can be used to treat severe *Shigella* infections. However, some *Shigella* have become resistant to antibiotics and using antibiotics to treat mild cases of shigellosis can make the bacteria more resistant in the future. For this reason, usually only severe cases of shigellosis will be treated with antibiotics.

The use of anti-diarrhoeal drugs is not recommended.

HOW IS IT PREVENTED?

Thorough washing of vegetables and fruit that is eaten raw is recommended.

Thorough handwashing with soap and water is the most important way to avoid contamination and infection. Hands should be washed after:

- going to the toilet;
- changing nappies;
- any exposures to faecal material.

People with shigellosis should avoid work and should not prepare food while they are sick. Sick children, particularly those in nappies, should be kept home from preschool while they have diarrhoea. Children and adults should avoid swimming until diarrhoea has stopped.

People who work as food handlers or who care for children or the elderly should not return to their duties until diarrhoea has stopped and two stool samples—taken at least 24 hours apart and at least two days after any antibiotics have finished—test negative for *Shigella*.

For further information please contact your local public health unit, community health centre, or doctor.

September–October 2002 ☒

COMMUNICABLE DISEASES REPORT, NSW: SEPTEMBER 2002

TRENDS

Notifications of communicable diseases through to the end of July (Figure 2 and Table 1) followed typical mid-winter patterns in NSW. Notifications of **arbovirus infections** and **cryptosporidiosis** were relatively few, but notifications of **invasive pneumococcal disease**, **meningococcal disease**, and **influenza**, were relatively frequent.

ENHANCED INFLUENZA SURVEILLANCE

To 3 August 2002, data from sentinel influenza surveillance sites in NSW showed increasing influenza A activity but decreasing influenza B activity in NSW (Figure 1).

For the week ending 3 August, 52 sentinel general practitioners in NSW reported diagnosing 123 patients with influenza-like illnesses [ILIs] among 5,469 total consultations (or 22.5 per 1,000 consultations). This is lower than the number of ILIs reported in the previous week. In the same week, six sentinel laboratories reported that 100 respiratory samples tested positive for influenza A and 10 for influenza B by either direct immunofluorescence or culture (a rate for influenza A, 16.9 positive per 100 samples; and for influenza B, 1.7 positive per 100 samples). These data represent an increase in the rate for

influenza A but a decrease in the rate for influenza B) compared with the previous week.

SYPHILIS REPORTS

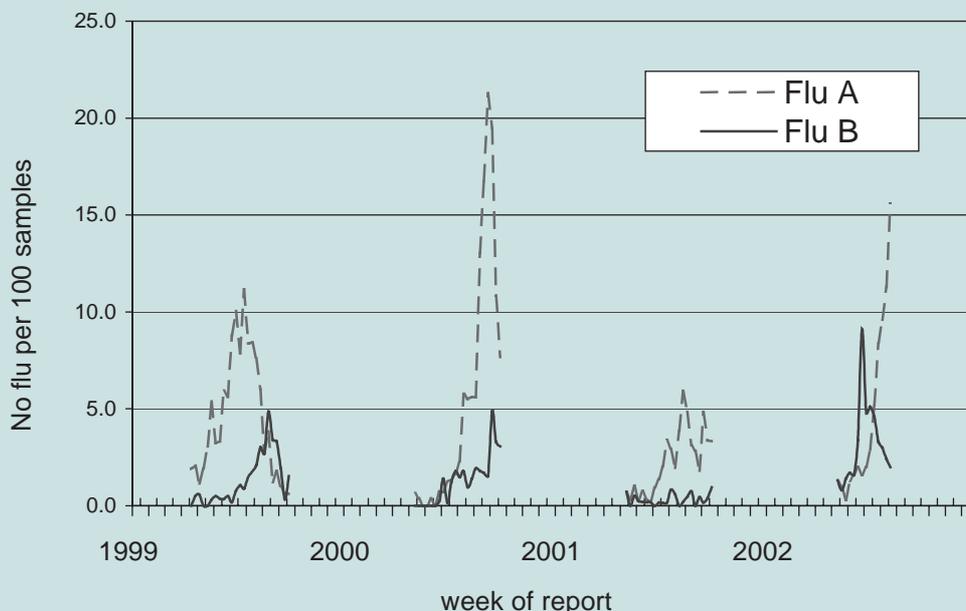
There have been several notifications of new cases of syphilis diagnosed in inner Sydney. Below are summaries of these cases from the staff of the Central Sydney and South Eastern Sydney Public Health Units.

The South Eastern Sydney Public Health Unit (SESPHU) began enhanced surveillance for syphilis in 2001. In 2001, 203 syphilis notifications were received by the SESPHEU. Of these, 19 cases were classified as having early syphilis (nine with the symptoms of primary syphilis [with chancre], four with the symptoms of secondary syphilis [with rash], and six with latent [asymptomatic] syphilis of < 1 year's duration). For 2002 to date, 119 syphilis notifications have been received (11 primary, four secondary, and four < 1 year's duration).

The Sydney Sexual Health Centre, a major public clinic in the SESPHEU, reports that early syphilis cases during 2001 were predominantly acquired overseas in countries with epidemics of syphilis among men who have sex with men. Some cases were also acquired locally, through sexual contact with visitors from these countries. Anecdotal reports from the Centre suggest that the number

FIGURE 1

REPORTS OF INFLUENZA DIAGNOSES BY SENTINEL LABORATORIES, NSW 1999–2002



Source: Communicable Diseases Branch, NSW Department of Health.

of locally-acquired cases in men has increased in 2002.

The Central Sydney Public Health Unit (CSPHU) began enhanced surveillance for syphilis in April 1999. CSPHU gathers information from the doctors of patients who have a positive serological result for syphilis. Data collected includes basic demographic information about the patient and information about the stage of disease.

The number of early syphilis cases has steadily increased from six in 1999 to 14 for the first seven months of 2002. Since March 2002 enhanced surveillance has identified six cases of primary or secondary syphilis, including four cases with chancre and two cases with rash, spots, and/or lymphadenopathy. The four cases with chancre have been diagnosed since June. All four were men and two of these individuals had acquired syphilis overseas. There was no link identified among these cases. Sexual Health Services in Central Sydney have since notified CSPHU of an additional four cases of primary syphilis in men who have sex with men.

This increase in the number of cases of early syphilis is consistent with trends in Europe, North America, and Britain, where increases in cases and outbreaks—often including men who have sex with men—have been reported since 1997.¹

The diagnostic classification of syphilis cases requires the interpretation of often-complex serological results and

patient histories, leading to the possibility of misclassification of cases. However, it is likely that many of these cases were infectious and these reports may be a harbinger of further cases. Both the SESPHEU and the CSPHU will be working closely with the NSW Department of Health, the AIDS Council of NSW, and the Sexually Transmitted Infections in Gay Men's Action Group (STIGMA) to develop prevention strategies.

Reference

1. Nicoll A, Hamers FH. Are trends in HIV, gonorrhoea, and syphilis worsening in western Europe? *BMJ* 2002; 324: 1324–1327.

FOCUS ON ENTERIC DISEASE

Enteric infections due to viruses, bacteria, parasites, and toxins may be transmitted via food and water and from person-to-person (directly and indirectly). Currently, 10 enteric infections are notifiable in NSW: botulism, cholera, cryptosporidiosis, giardiasis, haemolytic uraemic syndrome, verotoxigenic *E. coli* infections, hepatitis A, hepatitis E, listeriosis, and salmonellosis (including typhoid or paratyphoid).

Ms Jennie Musto has joined the Communicable Diseases Branch as epidemiologist for enteric infections. Future editions of the *NSW Public Health Bulletin* will include summaries of the surveillance for enteric infections. Readers' comments or suggestions for the content of this section would be appreciated. ☒

FIGURE 2

REPORTS OF SELECTED COMMUNICABLE DISEASES, NSW, JANUARY 1997 TO JULY 2002, BY MONTH OF ONSET

These are preliminary data: case counts for recent months may increase because of reporting delays. Laboratory-confirmed cases, except for measles, meningococcal disease and pertussis.

| | |
|----------------|-----|
| NSW population | |
| Male | 50% |
| <5 | 7% |
| 5-24 | 28% |
| 25-64 | 52% |
| 65+ | 13% |
| Rural* | 42% |

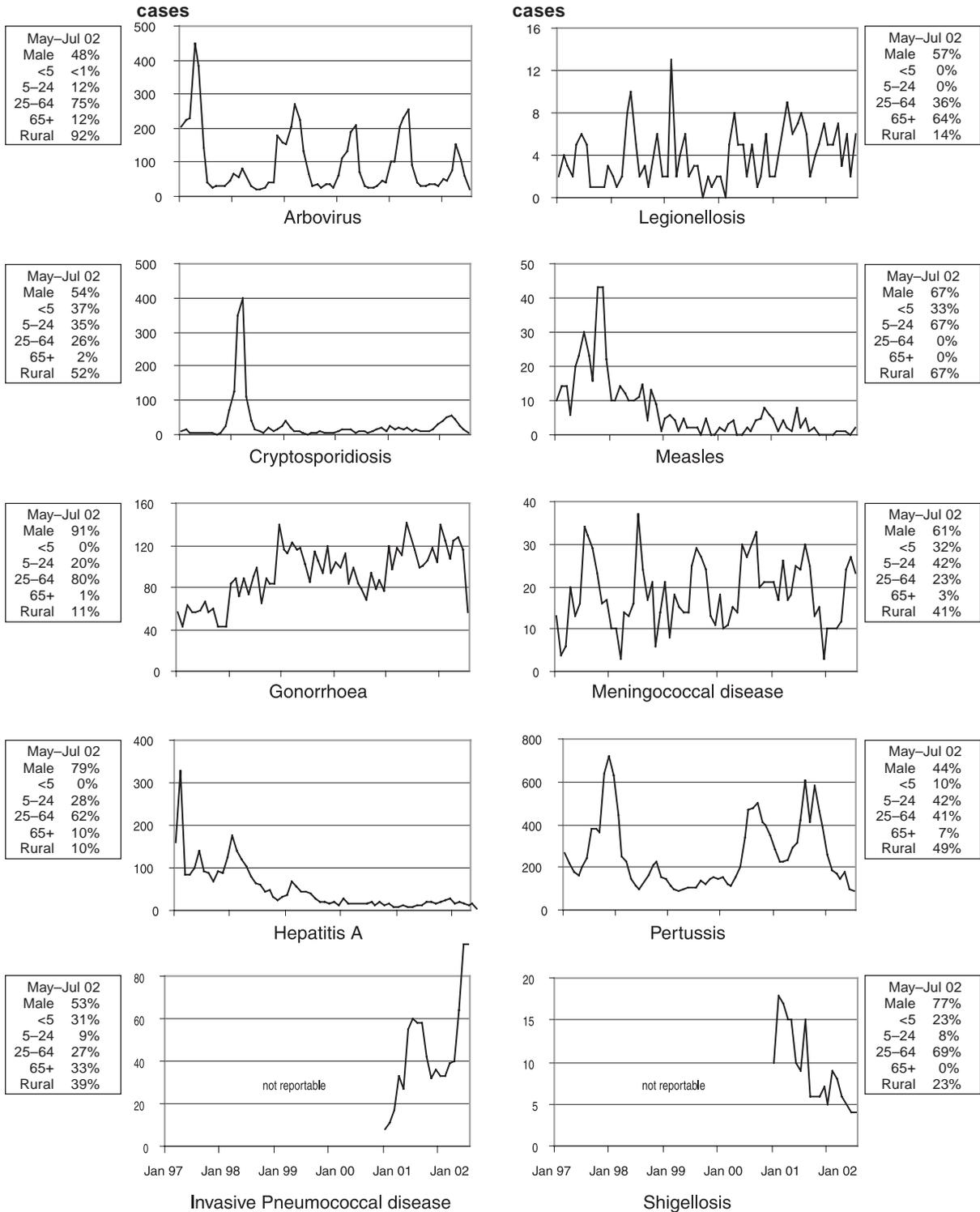


TABLE 1 **REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN JULY 2002 BY AREA HEALTH SERVICES**

| Condition | Area Health Service (2002) | | | | | | | | | | | | | | Total for Jul† | Total To date† | | | | | | | |
|--|----------------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|----------------|----------------|-----|-----|----|-----|-----|-------|-------|
| | CSA | NSA | WSA | WEN | SWS | CCA | HUN | ILL | SES | NRA | MNC | NEA | MAC | MWA | | | FWA | GMA | SA | CHS | | | |
| Blood-borne and sexually transmitted | | | | | | | | | | | | | | | | | | | | | | | |
| Chancroid* | - | 37 | 46 | 19 | - | - | 17 | 39 | 21 | 58 | - | - | 27 | 15 | 20 | 12 | 16 | 4 | 16 | 6 | - | 407 | 3,018 |
| Chlamydia (genital)* | 50 | 3 | 8 | - | 1 | 2 | 1 | 2 | 1 | 52 | 4 | 1 | 52 | 4 | 1 | 2 | 1 | 1 | - | - | 1 | 111 | 845 |
| Gonorrhoea* | 1 | 1 | - | - | 1 | - | - | - | - | 4 | 1 | - | 4 | - | 1 | - | - | - | - | - | - | 4 | 48 |
| Hepatitis B - acute viral* | 59 | 50 | 60 | 9 | - | 4 | 4 | 8 | 3 | 30 | 3 | 2 | 3 | 2 | 1 | 1 | 3 | 5 | 6 | - | 246 | 2,223 | |
| Hepatitis B - other* | - | - | - | - | - | 3 | 1 | 1 | - | 1 | - | 3 | - | 3 | - | 1 | - | 2 | - | - | - | 12 | 81 |
| Hepatitis C - acute viral* | 80 | 31 | 85 | 26 | 1 | 39 | 48 | 33 | 44 | 44 | 51 | 28 | 9 | 11 | 11 | 2 | 7 | 12 | - | - | 520 | 4,366 | |
| Hepatitis C - other* | - | 1 | 5 | - | - | - | - | 1 | - | 7 | - | - | - | - | - | - | - | - | - | - | 1 | 30 | 497 |
| Syphilis | 13 | 1 | - | - | - | - | - | - | 1 | 7 | 1 | - | - | - | - | 2 | - | - | - | - | - | - | - |
| Vector-borne | | | | | | | | | | | | | | | | | | | | | | | |
| Barmah Forest virus* | - | - | - | - | 1 | 1 | 1 | 7 | 1 | - | - | 4 | 18 | - | - | - | - | - | - | - | - | 33 | 318 |
| Ross River virus* | - | - | - | - | - | - | - | - | - | - | 1 | 5 | 2 | 2 | 2 | 2 | 1 | - | - | - | - | 14 | 166 |
| Arboviral infection (Other)* | - | 2 | 1 | - | - | 1 | - | - | - | 2 | - | - | - | - | - | - | - | - | - | - | 6 | 55 | |
| Malaria* | - | 1 | 1 | - | - | - | - | 1 | - | - | - | 1 | - | - | - | - | - | - | - | - | 5 | 81 | |
| Zoonoses | | | | | | | | | | | | | | | | | | | | | | | |
| Anthrax* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Brucellosis* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Leptospirosis* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Lysavirus* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Psittacosis* | - | - | - | 29 | - | - | 1 | 2 | - | 1 | - | 3 | 1 | - | 1 | - | 1 | - | - | - | 39 | 76 | |
| Q fever* | - | - | - | - | - | - | - | 1 | - | - | 4 | 5 | 1 | 4 | 3 | 1 | - | - | - | - | 22 | 136 | |
| Respiratory and other | | | | | | | | | | | | | | | | | | | | | | | |
| Blood lead level* | - | 2 | - | 5 | - | 1 | 19 | 9 | 9 | 1 | 1 | 2 | 1 | - | - | - | - | - | - | - | 48 | 258 | |
| Influenza* | 28 | 13 | 35 | 9 | 9 | 12 | 3 | 17 | 87 | 7 | 2 | 1 | 2 | 3 | 2 | 8 | 2 | 13 | - | - | 232 | 378 | |
| Invasive pneumococcal infection* | 8 | 9 | 15 | 12 | 12 | 3 | 19 | 3 | 14 | 3 | 3 | - | 2 | 2 | 2 | 1 | 7 | - | - | - | 119 | 400 | |
| <i>Legionella longbeachae</i> infection* | - | 4 | - | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 5 | 18 | |
| <i>Legionella pneumophila</i> infection* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 17 | |
| Legionnaires' disease (Other)* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Leprosy | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Meningococcal infection (invasive) | - | 1 | 2 | - | 1 | 2 | 4 | 2 | 7 | 2 | 3 | - | 2 | - | 2 | - | - | - | - | - | 26 | 116 | |
| Tuberculosis | 10 | 1 | 2 | 2 | 8 | 1 | - | 4 | 9 | - | - | 2 | 1 | - | - | - | - | - | - | - | 40 | 273 | |
| Vaccine-preventable | | | | | | | | | | | | | | | | | | | | | | | |
| Adverse event after immunisation | 4 | 6 | 1 | 2 | - | 1 | 4 | - | 2 | - | - | - | - | - | - | - | - | - | - | - | 21 | 110 | |
| <i>H. influenzae b</i> infection (invasive)* | - | - | - | - | - | - | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | 1 | 9 | |
| Measles | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 4 | |
| Mumps* | - | - | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 18 | |
| Pertussis | 10 | 10 | 16 | 4 | 12 | 4 | 19 | 4 | 16 | 9 | 8 | 2 | 10 | 5 | 6 | 4 | - | - | - | - | 139 | 1,442 | |
| Rubella* | - | - | - | - | 1 | - | - | - | 1 | - | 2 | - | - | - | - | - | - | - | - | - | 5 | 17 | |
| Tetanus | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Faecal-oral | | | | | | | | | | | | | | | | | | | | | | | |
| Botulism | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Cholera* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Cryptosporidiosis* | - | - | - | 1 | - | - | - | - | 1 | - | 1 | - | - | - | - | - | 1 | - | - | - | 4 | 253 | |
| Food borne illness (not otherwise specified) | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Gastroenteritis (in an institution) | 20 | - | 89 | 64 | - | - | 93 | - | 85 | - | - | - | - | - | - | - | - | - | - | - | 351 | 1,203 | |
| Giardiasis* | 3 | 15 | 14 | 3 | 4 | 2 | 2 | 5 | 10 | - | 1 | 2 | 1 | - | - | 3 | - | - | - | - | 65 | 557 | |
| Haemolytic uraemic syndrome | - | - | - | - | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 5 | |
| Hepatitis A* | 1 | - | 1 | - | - | 2 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 5 | 108 | |
| Hepatitis E* | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Listeriosis* | - | - | - | - | - | - | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | 1 | 3 | |
| Salmonellosis (not otherwise specified)* | 1 | 17 | 5 | 3 | 11 | 3 | 3 | 5 | 8 | 9 | 2 | 2 | 3 | 5 | 5 | 1 | - | - | - | - | 83 | 1,431 | |
| Shigellosis* | - | 3 | - | - | - | 1 | 1 | 1 | 1 | 1 | - | - | - | - | - | - | - | - | - | - | 7 | 47 | |
| Typhoid and paratyphoid* | - | - | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 1 | 21 | |
| Verotoxin producing <i>E. coli</i> * | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | 3 |

* lab-confirmed cases only + includes cases with unknown postcode * HIV and AIDS data are reported separately in the Public Health Bulletin quarterly

| | | | | |
|----------------------------|---------------------------------|----------------------------|---------------------------------|----------------------------------|
| CSA = Central Sydney Area | WEN = Western Sydney Area | WNS = Northern Sydney Area | WNS = Southern Sydney Area | WNS = Central Coast Area |
| NSA = Northern Sydney Area | SWS = South Western Sydney Area | CCA = Central Coast Area | HUN = Hunter Area | ILL = Illawarra Area |
| WSA = Western Sydney Area | SWS = South Western Sydney Area | CCA = Central Coast Area | SES = South Eastern Sydney Area | SES = South Eastern Sydney Area |
| | | | NRA = Northern Rivers Area | MNC = North Coast Area |
| | | | MNC = North Coast Area | NEA = New England Area |
| | | | MAC = Macquarie Area | MWA = Far West Area |
| | | | MWA = Mid Western Area | FWA = Far West Area |
| | | | GMA = Greater Murray Area | SA = Southern Area |
| | | | SA = Southern Area | CHS = Corrections Health Service |

NSW PUBLIC HEALTH BULLETIN

The *NSW Public Health Bulletin* is a publication of the NSW Department of Health.

The editor is Dr Lynne Madden, Manager, Public Health Training and Development Branch.

Dr Michael Giffin is the managing editor.

The Bulletin aims to provide its readers with population health data and information to support effective public health action.

The Bulletin is indexed by MEDLINE and *Index Medicus*.

Submission of articles

The preferred length of Bulletin articles is 1500 words. Tables and figures may be additional to that.

News, comments, and other reports should be 500–600 words.

All manuscripts should contain a short introductory abstract that reflects the structure of the manuscript.

References should be set out in the Vancouver style.

Send submitted manuscripts on paper and in electronic form, either on disc (Word for Windows is preferred), or by email.

The manuscript must be accompanied by a letter signed by all authors.

Full instructions for authors are available on request from the managing editor.

Editorial correspondence

Please address all correspondence and potential contributions to The Managing Editor, *NSW Public Health Bulletin*, Locked Mail Bag 961, North Sydney, NSW 2059, Australia or by email to phbulletin@doh.health.nsw.gov.au. Tel: 61 2 9391 9241, Fax: 61 2 9391 9232.

Distribution

To obtain copies of the *NSW Public Health Bulletin* please contact your local public health unit or by telephone at 61 2 9391 9942.

A new subscribers–change of address form is printed in most issues of the Bulletin. There is also an online subscription form available at the Bulletin's website.

The Bulletin can be accessed in both PDF and HTML formats from www.health.nsw.gov.au/public-health/phb/phb.html.

All back issues are downloadable from the website. Back issues of the printed version can be obtained from:

Public Health Training and Development Branch

NSW Department of Health

Locked Mail Bag 961

North Sydney, NSW 2059, Australia. ☒

Copyright © 2002 NSW Department of Health

NSW HEALTH
Working as a Team

NEW SUBSCRIBERS—CHANGE OF ADDRESS FORM

Please return to:

NSW Public Health Bulletin
NSW Department of Health
Locked Mail Bag 961, North Sydney NSW 2059, Australia

Fax: 61 2 9391 9232

SUBSCRIBE AND UNSUBSCRIBE

- I wish to be placed on the mailing list
 Please remove me from the mailing list

Name: _____

Organisation: _____

Mailing address: _____

City: _____ State: _____

Postcode: _____ Country: _____

Telephone: _____ Facsimile: _____

Email: _____

CHANGE OF ADDRESS

- I wish to change my mailing details, as follows:

FROM: _____

TO: _____

SUBSCRIBE TO THE WEB VERSION

The *Bulletin* can be accessed via the Internet from our Web site at www.health.nsw.gov.au/public-health/phb/phb.html. If you would like to be informed by email when new issues of the *Bulletin* become available, please subscribe to the Internet mailing list when you next visit the site.