NSW Public Health Bulletin

Volume 14, Number 8
August 2003

NSW HEALTH
Working as a Team

ISSN 1034 7674
State Health Publication PH 030221

QUANTITATIVE HEALTH RISK ASSESSMENT

GUEST EDITORIAL

Stephen Corbett
Centre for Public Health
Western Sydney Area Health Service

This issue of the *NSW Public Health Bulletin* focuses on the application of quantitative health risk assessment in public health decision-making. Over the last decade, these assessments have become a common currency for government, industry, and public health officials. Risk assessment, however, is not a value-free science. Underpinning the practice are notions of what health is, what risks are tolerable, what constitutes evidence, and the legitimacy of government intervention in the management of risk.

Some recent developments in the methodology of risk assessment, in particular the application of genetic science to risk assessment, give cause for optimism that the credibility and usefulness of risk assessments will improve. Some of these developments are summarised in the short history of quantitative health risk assessment presented in this editorial.

There is no doubt that, in good hands, risk assessments can contribute to good decisions about risk. There is also no doubt that these decisions are increasingly the subject of close scrutiny by a scientifically literate and sceptical public. The articles in this issue of the Bulletin attest to the utility of the intelligent application of risk assessment to common problems in public health practice. First, Andrew Langley discusses some of the philosophical underpinnings of the methods of health risk assessment. Geoff Richards gives a 'worked' example of the calculations made in a typical request for risk information. Community consultation is an integral part of all risk assessment, and Alison Rutherford describes an example of this often difficult negotiation. An intriguing application of risk assessment is found in Craig Dalton's article on selenium contamination in Lake Macquarie in the Hunter region. Finally, Cris Hickey and Christine Cowie examine applications of risk assessment methods in the derivation of standards for recreational water quality.

continued on page 162

CONTENTS

QUANTITATIVE HEALTH RISK ASSESSMENT: A SHORT HISTORY

A quantitative approach to health risk assessment originated in the United States in the 1970s, in the context of the rising costs of environmental, food and drug legislation, and in the conviction that human cancers were largely attributable to chemical exposure.[1] In 1977, President Carter appointed an Inter-Agency Regulatory Liaison Group to coordinate regulatory activities in the environment, the workplace, product safety, and public health. A Risk Assessment Workgroup was charged with the responsibility for developing common criteria and approaches to the scientific aspects of risk assessment techniques. From the outset, there were fundamental objections to the use of quantitative risk assessment as a basis for decision making—it lacked a scientific foundation and it detracted from the efforts to reduce pollutants and contaminants using the best technology available.

The quantitative risk assessment approach was applied in five major areas: setting priorities; reviewing residual risk after application of best available technology; balancing risks with benefits; setting standard and target levels of risk; and estimating risks for specific populations.[2]

For carcinogens, there was a central controversy—the assumption that there was no threshold in the dose–response relationship for a carcinogen (that is, there is no safe minimum exposure and only a zero level of exposure is safe).[3] The first rigorous attempt to propose a non-zero level of exposure to a carcinogen was put forth by Mantel and Bryant in 1961.[4] They tackled the problem of extrapolating from high experimental doses in animal bioassays to the lower doses observed in human experience. Work on radiation exposure and leukaemia in atomic bomb survivors suggested that cancer risk could be extrapolated linearly from the 'no observed adverse effect level' (NOAEL) through zero with no apparent threshold. This approach was adopted as a default assumption in chemical risk assessment without strong evidence to support it.

To strengthen the scientific respectability of regulatory risk assessment, the National Research Council of the United States National Academy of Sciences published what is known as the 'Red Book', which defined four risk assessment disciplines: hazard identification, dose–response assessment, exposure assessment, and risk character-isation.[5]

Hazard identification

Hazard identification was a largely qualitative step aimed at evaluating the weight of evidence. Policies dictated that the most sensitive animal species be used for estimating the human response. Human epidemiologic data, though seldom available, was accorded the greatest 'weight'.

Dose–response assessment

Dose–response assessment evaluated the quantitative evidence from animal studies or, less commonly, epidemiologic studies to estimate risk of cancer as a function of exposure. Because environmental exposures are generally orders of magnitude less than those in either animal experiments or epidemiologic studies, extrapolation models were adopted to characterise risks for environmental exposures. A low dose linearity assumption was adopted as a default (that is, risks were assumed to decline to zero in a linear fashion from the lowest exposure known to cause health effect). Safety factors, usually in the range 10–100, were applied to account for the uncertainties of inter-species extrapolation and inter-individual variability.

Exposure assessment

Exposure assessment evaluated the character and level of exposure to substances in the population under consideration. This included the specific chemical forms, routes, and time course of exposure. Characterisation of the heterogeneity of exposure was by adopting conventions such as the maximally exposed individual (MEI) as an upper-bound exposure. The MEI was assumed to be exposed 24 hours per day for 70 years.

Risk characterisation

Risk characterisation is the quantification of risk based on information synthesised from hazard identification, dose–response assessment, and exposure assessment.

These four risk assessment disciplines have been applied to the assessment of non-carcinogenic chemicals over the last two decades.

NEW APPROACHES IN QUANTITATIVE HEALTH RISK ASSESSMENT

In each of these risk assessment disciplines, developments in genetics, toxicology, and statistical methods, have tried to address some of the more obvious problems of uncertainty and compounding conservatism. (Conservatism, in this sense, means that a standard is overly cautious; hence compounding conservatism is a situation where a series of cautious assumptions are used to derive a measure of risk, giving an ultra cautious result.)

Hazard assessment

Over the past two decades, research has recognised the importance of genetic and epigenetic (that is, processes that modify gene expression) mechanisms that determine responses to chemical hazards. These emerging genetic complexities will have a major effect on the simplifications inherent in current risk assessment practice.

Dose–response assessment

There have been great advances in the understanding of physiologically-based pharmacokinetic models in

toxicology. These advances enable a more accurate scaling of doses established in animal models to humans, by using the relevant determinants of pharmacokinetics such as tissue blood flow, tissue volume, and metabolic rate.

The 'benchmark dose' approach has been developed as an alternative to the 'no observed effect level' (NOAEL) approach, for both cancer and non-cancer health endpoints.[6] The benchmark dose corresponds to a pre-determined increase (usually five per cent) in the risk of an adverse health effect in a defined population. It has the advantages of taking into account the entire dose–response information, rather than a single dose. It is less influenced by the arbitrary choice of dose.

The International Program on Chemical Safety, as part of its Harmonization of Approaches to the Assessment of Risk of Exposure to Chemicals Program, has developed a guidance document for the use of chemical-specific adjustment factors for inter-species differences and human variability in dose–response assessment.

Exposure assessment

Developments in statistical modelling, and in particular the use of Monte Carlo modelling for incorporating exposure distributions into risk assessments, have been an important advance.

Risk characterisation

Improvements in hazard identification, dose–response assessment, and exposure assessment, have improved the way risk characterisation synthesises the quantification of risk.

Paustenbach has summarised some of the lessons learned in quantitative risk assessment and suggested areas for improvement in each of the four risk assessment disciplines (Table 1).[7]

A STRATEGY FOR IMPROVING THE QUALITY, CREDIBILITY, AND USEFULNESS OF QUANTITATIVE HEALTH RISK ASSESSMENT

Increased use of human data in health risk assessment

Much of the critique of risk assessment methodology revolves around the use of extrapolation of results from animals to humans. There is a clear need for better information on the effects of chemical hazards on human health.

In 1978, Saracci laid out a strategy for environmental epidemiology,[8] which called for:

- improvements in exposure assessment—there have been great advances in the availability and utility of biologic markers of previous human exposure;

TABLE 1

IMPROVING RISK ASSESSMENT: LESSONS LEARNED

Hazard Assessment	Dose–Response Assessment	Exposure Assessment	Risk Characterisation
Do not consider all animal carcinogens (equally) as a serious hazard	Present upper bound of risk plus best estimate of bounds.	Don't put too much emphasis on risk estimates for maximally exposed individuals.	Understand that one in a million increased risk is rarely a significant public health hazard.
Consider weight of evidence	Consider estimates from several low dose models.	Evaluate the uptake (absorbed dose) for both 50% and 95% persons.	Do not interpret low dose modelling results as an actual increase in risk (rather than a plausible upper bound).
	Consider reality check using epidemiological data.	Do not use repeatedly conservative or worst case assumptions. Use Monte Carlo techniques whenever possible.	Consider background levels of exposure when characterising incremental risk.
	Adjust for biological differences among species using physiologically based pharmacokinetic (PBPK) models.	Ensure a proper statistical analysis of environmental data, including a sensitivity analysis.	Do not assume the solution is remediation, destruction or substitution.
	Use low dose models to rank carcinogens rather than using models to predict cancer rate.	Understand the role of environmental fate when estimating exposure.	Put estimates of risk into perspective. Characterise risk using Monte Carlo analysis.
	Understand the fragility and sturdiness of low dose models.	Consider using biological monitoring to confirm exposure estimates.	Conduct uncertainty and sensitivity analyses.
		Consider all indirect pathways of exposure.	

Source: Paustenbach D. *The Practice of Health Risk Assessment in the United States.*[7]

- tackling the problems of the combined effects of multiple exposures—the disaggregation of the effects of dose–response, interactive effects, and induction periods, is a formidable task;
- integration of experimental and epidemiological evidence, which will require a more intense collaboration between toxicologists, environmental scientists, and epidemiologists.

A set of principles for evaluating epidemiologic data for use in risk assessment, known as the London Principles,[9] have been proposed and are summarised in Table 2.

Characterising individual susceptibility

An emerging issue in environmental epidemiology, and in both clinical and regulatory toxicology, is that of variation in susceptibility. This concept is not new. It constitutes the 'host' in the old paradigm of epidemiology that divided causes of disease into environment, host, and agent. It has, however, taken on a new dimension with the rapid developments in the characterisation of the human genome.[10]

Chemical toxicants have the potential to cause alterations at different organisational levels of a cell or tissue:[11]

- genome: the chromosomal information;
- transcriptome: the messenger RNA from actively transcribed genes;
- proteome: the entire protein complement of a biological sample;
- metabonome: the constituent metabolite in a biological sample.

Rapidly evolving technologies are enabling the characterisation of idiosyncratic responses to chemical toxicants; these include genomics, pharmocogenetics or toxicogenetics, functional genomics, and proteomics.

Genomics are the techniques for characterising the DNA sequence of the genome. The investigation of variable, or polymorphic regions of genes in an attempt to characterise idiosyncrasies in response to chemical insults is called *pharmocogenetics* or *toxicogenetics*. *Functional genomics* refers to a host of technologies that enables the functions of genes to be investigated. *Proteomics* is the characterisation of protein modifications that may lead to changes in the activity of gene products.

The application of these emerging technologies could assist risk assessment by:

- enhancing the ability to extrapolate accurately between animals and humans;
- enabling a more detailed understanding of molecular mechanisms of toxicity.

There is considerable optimism that these technologies can greatly enhance our understanding of the risks to health posed by chemicals in the environment.[12]

Prioritising health risk assessment: National and international practice

Chemicals used in food production, household products, textiles, medicines, and automobiles, underpin modern life. Global production of industrial chemicals has increased from one million tonnes in 1930 to 400 million tonnes today. The number of chemicals marketed in

TABLE 2

THE LONDON PRINCIPLES FOR EVALUATING EPIDEMIOLOGIC DATA IN REGULATORY RISK ASSESSMENT

Principles for Evaluating an Epidemiologic Report for Cause–Effect Relationship	
A1	The population studied should be pertinent to the risk assessment at hand, and it should be representative of a well-defined underlying cohort or population at risk.
A2	Study procedures should be described in sufficient detail, or available from the study's written protocol, to determine whether appropriate methods were used in the design and conduct of the investigation.
A3	The measures of exposure(s) or exposure surrogates should be: • conceptually relevant to the risk assessment being conducted; • based on principles that are biologically sound in light of present knowledge; • properly quantitated to assess dose-response relationships.
A4	Study outcomes (endpoints) should be clearly defined, properly measured, and ascertained in an unbiased manner.
A5	The analysis of the study's data should provide both point and interval estimates of the exposure's effect, including adjustment for confounding, assessment of interaction (for example, effect of multiple exposures or differential susceptibility), and an evaluation of the possible influence of study bias.
A6	The reporting of the study should clearly identify both its strengths and limitations, and the interpretation of its findings should reflect not only an honest consideration of those factors, but also its relationship to the current state of knowledge in the area. The overall study quality should be sufficiently high that it would be judged publishable in a peer-reviewed scientific journal.

Source: Federal Focus Inc. *Principles for Evaluating Epidemiological Data in Regulatory Risk Assessment*.[9]

volumes above 10 kg reported in 1981 was 102,806, of which 30,000 are marketed in volumes greater than one tonne per annum.

National and international chemical policies must ensure high levels of protection of human health for present and future generations. Adoption of the 'precautionary principle' is fundamental to achieving this objective. That is, whenever scientific evidence is available that a substance may have an adverse effect on human health and the environment, but there is still uncertainty as to the nature and magnitude of that effect, then decision-making must be precautionary.

The European Union and other countries have made a distinction between new and existing chemicals, in constructing mandatory regulatory requirements for the assessments of chemicals. Existing chemicals are subject to lesser scrutiny and account for over 99 per cent of the total number of chemicals. Some 140 of these substances have been listed as priority chemicals requiring comprehensive assessment.

The European Union White Paper outlines a strategy for a future chemicals policy.[13] It proposes a scheme that classifies and prioritises the vast list of existing chemicals—the REACH (Registration, Evaluation and Authorisation of Chemicals) Program. Registration of basic information is required for each of the 30,000 existing and new chemicals with production volumes greater than one tonne per annum. Evaluation of the registered information is required for all substances exceeding a production volume of 100 tonnes per annum. Authorisation of substances with certain hazardous properties that give rise to high levels of concern requires that authorisation be given before a substance can be used. Substances of concern include those that are carcinogenic, mutagenic, or toxic to reproduction, or substances with POP (persistent organic pollutants) characteristics.

This screening and risk classification does include some assessment of likelihood of human exposure, although production volume is used as the most convenient proxy for this measure. Some exemptions for assessment can be granted, if it can be demonstrated that human exposure is unlikely.

In Australia, the National Industrial Chemicals Notification and Assessment Scheme compiles detailed assessments of priority chemicals. Priorities are assessed by a consideration of published information on toxicity, volume of use, assumed frequency of exposure, and severity of health or environmental effects.[14]

CONCLUSION

Of public health, the historian Christopher Hamlin has said: 'What masquerades as an obscure offshoot of medicine or a marginal division of civil engineering is really a vast and unexamined part of our culture.'[15] Over the last two decades, quantitative risk assessment has, more or less by stealth, become a part of the culture of Australian risk management.

In June 2002, the enHealth council published Guidelines for Assessing Human Health Risks from Environmental Hazards.[6] This publication presents, for the first time, a considered national approach to health risk assessment practice, which will hopefully lead to a more consistent and critical application of this important technology.

REFERENCES

1. Landy MK, Roberts MJ, Thomas SR. Chapter 6—Forging a Cancer Policy: The Interagency Regulatory Liaison Group. *The Environmental Protection Agency: From Nixon to Clinton*. Oxford: Oxford University Press, 1994.
2. Anderson EL and the Carcinogen Assessment Group of the United States Environmental Protection Authority. *Risk Analysis* 1983; 3: 277.
3. Hrudey SE. Quantitative Cancer Risk Assessment: Pitfalls and Progress in Risk Assessment and Risk Management. Hester RE and RM Harrison RM (editors). *Issues in Environmental Science and Technology Number 9*. Cambridge: Royal Society of Chemistry, 1998.
4. Mantel N, Bryant WR. *J Natl Cancer Inst* 1961; 27: 455.
5. National Research Council. *Risk Assessment in the Federal Government: Managing the Process*. Washington DC: National Academy Press, 1983.
6. Environmental Health Risk Assessment, 2002. enHealth Council online at http://enhealth.nphp.gov.au/council/pubs/pdf/envhazards.pdf.
7. Paustenbach D. The Practice of Health Risk Assessment in the United States. *Human and Ecological Risk Assessment* 1995; 1(1): 29–75.
8. Saracci R. Epidemiological Strategies and Environmental Factors. *Int J Epidemiol* 1978; 7: 101–111.
9. Federal Focus Inc. Principles for Evaluating Epidemiological Data in Regulatory Risk Assessment. Washington DC: Federal Focus Inc., 1996.
10. Hertz Picciotto I. *Environmental Epidemiology in Modern Epidemiology*. Rothman KR, Greenland S (editors). Rven: Lippincott, 1998.
11. Pennie B, Ahr HJ, Braun C, Bars R, Jacob E. *Genomics, Transcript Profiling and Metabonomics: An Introduction*. European Centre for Ecotoxicology and Toxicology of Chemicals Document No 42.
12. Olden K, Guthrie, J. Genomics: Implications for Toxicology. *Mutation Research* 2001; 473: 3–10.
13. Commission of European Communities. White Paper: Strategy for a Future Chemicals Policy. Brussels: Commission of European Communities, 2001.
14. National Industrial Chemical Notification and Assessment Scheme. *Notifiers Handbook: Section 3—Priority Existing Chemicals Assessments* online at http://www.nicnas.gov.au/publications/pdf/handbook/chapter9.pdf.
15. Hamlin C. *Public Health and Social Justice in the Age of Chadwick: Britain, 1800–1854*. Cambridge: Cambridge University Press, 1997.

WHAT DOES IT MEAN WHEN THE RISK ASSESSMENT SAYS 4.73 X 10^{-5}?

Andrew Langley
Sunshine Coast Public Health Unit
Queensland Health

A number is a number is a number … and yet exactitude should not be confused with accuracy. This article describes some of the philosophical underpinnings of the methods of health risk assessment.

BACKGROUND

The development of risk assessment methodologies in the 1970s and 1980s proceeded along two paths. Qualitative risk assessment sought to categorise risk. In some situations, this was into broad categories such as 'safe' (or 'acceptable') and 'unsafe' (or 'unacceptable'); in other situations, a series of very well defined categories was used. An example of this is the grading of substances by WHO's International Agency for Research on Cancer into one of five levels of carcinogenicity.[1]

Quantitative risk assessment provides a numerical estimate of risk. It is emphasised that it is an estimate or calculation rather than an actual value. McKone and Bogen describe three types of risk: 'actual', 'calculated', and 'perceived'.[2] Ideally, these would be equivalent, but often risks are unquantifiable and unknowable as we have insufficient information on which to base the calculations, or our tools are not subtle (or accurate) enough. There are numerous quantitative risk assessment methodologies. The most prominent are probably the Cancer Risk Assessment Guidelines of the United States Environmental Protection Agency (US EPA) developed in the early 1980s.[3]

Quantitative risk assessment has been most controversial when applied to carcinogens, because of debates about the level of conservatism (that is, the caution associated with particular assumptions and default values chosen for the risk assessment) in estimates of risk. US EPA methodologies have been the most influential in the area of cancer risk assessment, but these contain a range of conservative assumptions that have been adopted for the pragmatic purpose of implementing cancer risk assessment rather than being established scientific fact.[4]

For exposure assessment, a series of 'high end' (that is, conservative) estimates of particular exposure factors is used in some assessments. The compounding effect of simultaneously combining several 'high end' estimates may result in 'an exceptionally rare value output' (that is, extremely conservative estimates).[4]

The US EPA has commented that the 'high end' risk estimates generated by its methodology are not 'necessarily a realistic prediction of risk' and that the 'true value' may be as low as zero.[4,5] The conservative nature of the methodology has been defended as necessary, in order to deal with the uncertainties in risk assessment, especially those relating to carcinogenicity data derived from feeding studies with limited cohorts of animals.

Having a number for the estimate of risk is somewhat meaningless, unless there are benchmarks such as an 'acceptable' level of risk against which the estimate can be judged. Frequently, a value of 1×10^{-6} is used to determine acceptability. Further, it needs to be clarified whether this is a risk per annum or over a lifetime, using a default life expectancy of 70 years. When quantitative risk assessment was first being used, an acceptable value of risk over a lifetime of 1×10^{-8} was arbitrarily proposed.[4] This was reduced to 1×10^{-6}, which was considered to be a *de minimis* risk, from the legal term *De minimis non curat lex* (the law does not concern itself with trifles). Paustenbach refers to a commissioner of the US Food and Drug Administration indicating that a risk of 1×10^{-6} did not mean that 1×10^{-6} exposed persons would develop cancer but rather that the risk was virtually nonexistent.[6] A review of US decision-making shows that risks between 4×10^{-3} and 10^{-6} have been deemed acceptable.[6]

The media, lawyers, and engineers like the concept of quantitative risk assessment but the risks and the context of the risk do not have the 'one dimensional' character of a number. While the public often just wants to know whether something is 'safe' or 'unsafe', regulatory risk assessors usually have to deal with uncertainty. While the US EPA methodology explicitly requires uncertainty assessments (that is, qualifications) around the risk characterisation, these are often not done.

Despite these problems, while a quantitative risk assessment model may lack accuracy in its risk characterisations, it may have sufficient precision to enable risks to be ranked, and for cost benefits to be compared for a variety of interventions. If the conservatism can be identified and taken into account it can reasonably be stated that the actual risk is unlikely to exceed this estimate and, hence, if the estimate falls below the criterion for acceptable risk, the actual risk is unlikely to exceed the criterion.

The US EPA is tending towards using narrative descriptions of risk, and has proposed this approach in its review of the methodology for assessing carcinogens.[7] A narrative description can provide more 'shading' to the nature and magnitude of the risk than a number that may not capture the 'subjectivity and multiple dimensions of risks'.[8]

RULES OF THUMB FOR ASSESSING QUANTITATIVE RISK ASSESSMENTS

It is important to determine whether the risk assessment has been documented clearly, coherently and completely and whether it has been exposed to peer review.[9] A range of questions can then be asked:

- Why was the risk assessment done? What was the societal and risk management context in which the risk assessment was done? What is the meaning of the risk to those involved in the situation? What information did the risk manager want? Will the risk assessment affect the management of the situation?
- Was there a better way of managing the issue than using a risk assessment?
- How will the results of the risk assessment be interpreted? Is there a need to determine an 'acceptable' or 'tolerable' level of risk?
- Is a qualitative risk assessment sufficient or more appropriate than a quantitative risk assessment?
- Were there sufficient data relevant to the local situation to be able to undertake a quantitative risk assessment? Was there an excessive reliance on default data rather than on data that is from the relevant population or situation?
- Are all the equations and default assumptions and values available and transparent or is it a 'black box' where the details of the methodology are unclear? This is particularly important where the results are presented as definitive.
- Has the risk estimate been calculated to too many decimal points?
- Is the risk estimate a 'best estimate' of risk, or does it reflect the inclusion of multiple conservative assumptions (for example, relating to exposures and the dose–response slope for carcinogens), the compounding effect of which is to provide a very conservative estimate of risk?
- Is the risk estimate derived for typical members of the population or only highly-exposed people?
- Does the model give a good appreciation of uncertainty for each stage of the risk assessment?
- What are the effects of doing a sensitivity analysis using changes in assumptions or different data selections?
- How could this risk estimate be improved?

CONCLUSION

To assist Australian risk assessors, the National Environmental Health Council (enHealth) has recently released a comprehensive risk assessment methodology, which includes a chapter on appraising risk assessment reports and risk characterisations.[10] It also describes techniques such as the Monte Carlo method,[10] which can—if done properly—help to improve the meaningfulness of quantitative risk assessments. The National Health and Medical Research Council (NHMRC) has established a Committee on Risk Assessment and Toxicity, which had its inaugural meeting in September 2002. Among its tasks is to advise the NHMRC on best practices in health risk assessment.

ACKNOWLEDGEMENTS

Bruce Markey assisted with the identification of historical references.

REFERENCES

1. International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. IARC monographs may be found at http://monographs.iarc.fr.
2. McKone TE, Bogen KT. Predicting the uncertainties in risk assessment. *Environmental Science and Technology* 1991; 25(10): 1674(8).
3. United States Environmental Protection Agency. *Federal Register* 1986; 51: 33–992.
4. Hrudey SE. Quantitative cancer risk assessment—Pitfalls and progress. Hester RE and Harrison RM (editors). *Issues in Environmental Science and Technology, No. 9*. Cambridge: Royal Society of Chemistry Risk Assessment and Risk Management, 1998.
5. Barnard RC. Major issues raised by the OMB report from the perspective of the regulatory agencies. *Regulatory Toxicology and Pharmacology* 1991; 14: 223–228.
6. Paustenbach DJ, Jernigean JD, Finley BL, Ripple SR, Keenan RE. The current practice of health risk assessment: Potential impacts on standards for toxic air contaminants. *Journal of the Air & Waste Management Association* 1990; 40(12): 1620–1630.
7. United States Environmental Protection Agency, Office of Research and Development. *Draft Revision to the Guidelines for Carcinogenic Risk Assessment.* Washington DC: United States Environmental Protection Agency, 1996.
8. Thomas SP and Hrudey SE. *Risk of Death in Canada: What we know and how we know it* Edmonton: University of Alberta Press, 1997.
9. Vose D. *Risk Analysis: A Quantitative Guide*, 2nd edition. Chichester, West Sussex: John Wiley & Sons, 2000.
10. enHealth. *Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards*. Canberra: Department of Health and Ageing, 2002.

SUSPECTED PESTICIDE POISONING: A BACK-OF-THE-ENVELOPE HEALTH RISK ASSESSMENT

Geoffrey Richards
Environmental Health Branch
NSW Department of Health

Not all health risk assessment is of a formal and extensive nature. At times a 'back-of-the-envelope', or quick screening assessment, based on few or limited data may be appropriate. It may consist solely of discussion with a member of the public over the phone concerning a soil, water, food or air sample and its comparison with some national or internationally accepted standard or guideline; or it may deserve slightly deeper consideration. This article describes such an assessment, using the example of a suspected pesticide poisoning—an environmental health issue typical of those encountered by the Environmental Health Branch and the public health units each day.

BACKGROUND

A woman who purchased a home in southern Sydney had the sub-floor area treated for termites, by a licensed pest control company, prior to moving in. She alleged that soon after entering the house with her primary school age children she experienced nausea and headache. After vacating the house for a few days she returned, but could still detect an odour and again felt nauseous. The Pesticide Branch of the NSW Environment Protection Authority (EPA), was contacted and an investigation was initiated.

The house was constructed on short piers with little sub-floor ventilation, and the pest control operator stated that he used bifenthrin, a synthetic pyrethroid termiticide. Soil samples taken from under the house indicated an accumulation of chemicals, including traces of organochlorine pesticides, from successive termiticide treatments over many years; not an unusual condition for older houses in Sydney. Analyses strongly suggested a very recent treatment with bifenthrin on top of a treatment with chlorpyrifos, an organophosphate pesticide, undertaken within the previous one to two years or possibly longer, depending on the soil conditions that existed.

The EPA took air samples from a child's bedroom, which had been identified as a room having the strongest odour. A Gilian air sampler and ORBO 49 'puffer' tube was used to collect a 24-hour, one litre (L)/minute sample. The ORBO 49 tube is sensitive to synthetic pyrethroids and is very sensitive to organophosphates. Results of the air sample showed 3.5 micrograms (µg) of chlorpyrifos in 1,440 litres of air and no detection of bifenthrin. The EPA, through the South Eastern Sydney Public Health Unit, requested an assessment of health risk should an individual be constantly exposed to chlorpyrifos in the air at the concentrations measured during sampling.

CHLORPYRIFOS

Chlorpyrifos is a member of the organophosphorus class of chemicals and is registered for use in various formulations as a termiticide. It has been used in Australia for over 30 years and was recently extensively reviewed by the National Registration Authority for Agricultural and Veterinary Chemicals (NRA) under its Existing Chemicals Review Program.[1] The toxicology and assessment of risks, particularly from non-occupational exposure to chlorpyrifos, has also been comprehensively addressed in several papers published recently.[2,3]

Like other organophosphorus pesticides, chlorpyrifos inhibits the cholinesterase enzyme systems essential in the normal functioning of the nervous system. The most commonly reported effects of chlorpyrifos poisoning include: headache, nausea, dizziness, salivation, excess sweating, blurred vision, chest tightness, muscle weakness, abdominal cramps, and diarrhoea.

If occupational exposure is discounted, most effects from entry into areas treated with chlorpyrifos for termites are reported to be more likely a result of odour rather than the ability of the termiticide to inhibit cholinesterases.[2] This may be due to the active constituent itself, which has a distinctive sulphurous odour, or to volatile organic or petroleum solvents,with which chlorpyrifos and pyrethroids such as bifenthrin are usually formulated, and that may smell as they evaporate during and after application.[4]

ASSESSING RISKS

The results of the bedroom air sample showed 3.5 µg of chlorpyrifos in 1,440 L of air collected over 24 hours at a sampling rate of one L/minute.

Therefore, chlorpyrifos concentration:

= (3.5 µg/1,440 minutes) x (1minute/1L) x (1,000L/1m^3)

= (0.0024) x (1) x (1,000)

= **2.4 µg/m^3**.

For comparison, the Australian Occupational Air Standard,[5] based on an average airborne concentration of chlorpyrifos over a normal eight-hour working day for a five-day working week, and which according to present knowledge should not cause adverse health effects, is 0.2 milligrams (mg)/m^3 or: **200 µg/m^3**.

Comparisons with occupational exposure criteria; however, should be made with care, as appropriate adjustments have not been made for differing durations of exposure or for susceptible groups such as children.

The United States Environmental Protection Agency has developed very conservative risk-based concentrations of contaminants for screening purposes.[6] The risk-based concentration for chlorpyrifos in ambient air, which will not pose either an acute or long term threat to human health, is: **11 µg/m^3**.

Another quick form of comparison that can be made is by relating the amount of chlorpyrifos calculated to have been inhaled in the bedroom with the acceptable daily intake (ADI) of the chemical. The ADI for humans is considered to be a level of intake of a chemical over an entire lifetime without any appreciable risk to health. Regardless of the route of exposure (oral, dermal, or inhalation) the toxic effects of chlorpyrifos are similar.

The Australian ADI for chlorpyrifos is 0.003 mg/kilogram (kg)/bodyweight (bw)/day.[1,7] This is based on a 'no observable adverse effect level' (NOAEL) for plasma cholinesterase inhibition of 0.03 mg/kg bw/day derived from human studies and is more conservative than estimates based on inhibition of red cell or brain acetylcholinesterase. Thus, the ADI itself is conservatively based with a safety margin built in. For comparison the World Health Organization ADI is 0.01 mg/kg bw/day.

The children of the house purchaser were primary school age. Using an enHealth exposure default value,[8] assume that a child of 10 years is present in the bedroom for 24 hours of the day with a daily inhalation volume of 15m^3 of air, therefore estimated chlorpyrifos exposure:

= (15) x (2.4 µg)

= **36 µg/day**.

Add chlorpyrifos intake from food of 1.1 µg/day derived from the mean estimated daily dietary exposure of 12 year old children.[9] (No estimated intake for 10 year olds was found).

Estimated chlorpyrifos exposure:

= 36 µg/day + 1.1 µg/day

= **37.1 µg/day** intake of chlorpyrifos for child in bedroom.

No intake of chlorpyrifos from drinking water need be considered because Sydney Water monitors for pesticides and has not detected them in raw water sources. Dermal intake would also be limited because application of the chemical was sub-floor and not to the general living area.

In comparison assume a 10 year old child weighing 32 kg is exposed to the Australian ADI for chlorpyrifos:

(32) x (0.003 mg/kg)

= 0.096 mg/day or

= **96.00 µg/day** acceptable daily intake of chlorpyrifos for this child.

The estimated intake for a primary school child present in the house is therefore approximately one third of the acceptable daily intake.

CONCLUSION

If the air sampling and analyses were accurate, and the bedroom was an appropriate site in the house for testing, the results indicate that even when using conservative parameters, constant exposure to levels of chlorpyrifos detected in the bedroom would not cause either acute or chronic health effects.

The most likely explanation for the purchaser's symptoms would appear to be exposure to odours; either through application of the bifenthrin or through reactivation by wetting of the previous chlorpyrifos treatment. Although the effects of the odour were unpleasant, odours from chlorpyrifos and bifenthrin formulations per se are not known to cause toxicological effects unless accompanied by harmful concentrations of the chemicals. In this example, that was not the case. The broader issue of pesticide odours and their effects, however, is still of concern and has recently been given some attention by the National Registration Authority for Agricultural and Veterinary Chemicals (NRA) and the Commonwealth Department of Health and Ageing.

The house was constructed on short piers with little sub-floor ventilation and this coupled with high humidity may partially explain the lingering smell.

A citrus deodoriser was used and fans installed underneath the home to try to disperse the odour.

ACKNOWLEDGEMENTS

Thank you to John Hall of the NSW Environmental Protection Authority for providing background material.

REFERENCES

1. National Registration Authority for Agricultural and Veterinary Chemicals. *Review of Chlorpyrifos: Interim Report*. Canberra: National Registration Authority for Agricultural and Veterinary Chemicals Review Series 00.5, September 2000. Online at www.nra.gov.au/chemrev/chlor.shtml.
2. Cochran RC. Appraisal of risks from nonoccupational exposure to chlorpyrifos. *Regul Toxicol Pharmacol* 2002; 35: 105–121.
3. Gemert Mv, Dourson M, Moretto A, Watson M. Use of human data for the derivation of a reference dose for chlorpyrifos. *Regul Toxicol Pharmacol* 2001; 33: 110–116.
4. Therapeutic Goods Administration and Department of Health and Aged Care. *Termite Protection: Available Treatments and Hazard Information About Termiticides*. Canberra: Therapeutic Goods Administration and Department of Health and Aged Care, 2002.
5. National Occupational Health and Safety Commission. *Exposure Standards for Atmospheric Contaminants in the Occupational Environment: 3rd Edition*. Canberra: National Occupational Health and Safety Commission, 1995.
6. Smith RL. Risk-based concentrations: Prioritizing environmental problems using limited data. *Toxicology* 1996; 106: 243–266.
7. Therapeutic Goods Administration and Commonwealth Department of Health and Ageing. *ADI List: Acceptable Daily Intakes for Agricultural and Veterinary Chemicals. Current to 31 December 2002*. Canberra: Therapeutic Goods Administration and Commonwealth Department of Health and Ageing, 2003.

   Regularly updated at www.health.gov.au:80/tga/docs/html/adi.htm.
8. Department of Health and Ageing and enHealth Council. *Environmental Health Risk Assessment. Guidelines for assessing human health risks from environmental hazards*. Canberra, Commonwealth Department of Health and Ageing and enHealth Council, 2002.
9. Australian New Zealand Food Authority. *The 19th Australian Total Diet Survey 1998*. Canberra: Australian New Zealand Food Authority, 2001.

BUT YOU DON'T HAVE TO LIVE HERE! RISK ASSESSMENT AND CONTAMINATED SITES: A CASE STUDY

Alison Rutherford
Central Sydney Public Health Unit

Mortlake is an inner-western suburb of Sydney, adjacent to the Parramatta River. The Mortlake Gasworks provided gas to most of Sydney for almost 100 years. After closure as a gasworks, the site remained heavily contaminated with waste typical of gasworks, such as tarry organic compounds. The cleanup of the 52-hectare site began in 1998, with the aim of selling the land for residential and commercial development. The NSW Environment Protection Authority (EPA) and the local council managed the statutory development approvals and remedial action plans under relevant legislation.

Human health risk assessments undertaken in relation to the site clean up included:

- baseline assessments to determine appropriate clean up criteria;
- occupational health surveys, including health screening of workers;
- modelling studies that assessed the risk to future apartment residents from residual tar in rock and groundwater;
- an assessment of the risks of tarry marine sediments to recreational users;
- an assessment of the risk of airborne contaminants leaving the site during the remediation.

The Central Sydney Public Health Unit (CSPHU) reviewed some of these assessments. This article describes the final assessment—the risk of airborne contaminants leaving the site during the remediation—in the context of the advantages and limitations of quantitative risk assessment and differing perceptions of what constitutes a 'risk to health'.

MY STREET STINKS!!

Residents living near the former gasworks began complaining of odours and health effects soon after the clean up began. Some residents reported immediate symptoms whenever the odours were present, such as headaches, itchy eyes, and nausea; other residents were concerned about the long-term health effects of odorous chemicals, particularly on their children's health. An independent environmental health consultant was contracted by the local council to investigate these symptoms.[1]

Many chemicals have different odour, irritative, and toxic thresholds. Some, such as benzene, are potentially toxic without any odour noticeable; others, such as naphthalene, are odorous at levels well below their toxic threshold.[2]

The consultant conducted a simple quantitative and qualitative risk assessment, involving:

- a comparison between the measured concentrations of contaminants at the site boundary and concentrations known to have a toxic effect (derived from toxicological studies);
- a detailed review of the risk associated with emissions of benzene from the site;
- interviews with affected residents and a qualitative assessment of their symptoms and potential causes.

The results demonstrated no risk from benzene emissions and suggested that the symptoms being experienced were not from direct chemical toxicity but rather that odours were initiating a physiological or olfactory–limbic response, both of which have been previously identified as mechanisms for the symptoms described.[3] However, it was predicted that the symptoms would not abate until the odours were abated, and that people could become sensitised to odour and experience symptoms even when exposed to very low odour levels.

This risk assessment was important because:

- some of the chemicals on site were potentially carcinogenic;
- symptoms were validated by a recognised physiological mechanism;
- the site managers directed attention towards more active odour mitigation strategies (such as working in odorous areas only during suitable wind conditions and limiting the size of work surfaces);
- the EPA was provided with evidence to back up stronger regulatory action;
- new monitors with lower detection limits for benzene were introduced, due to the concern of some residents that the detection levels of benzene were slightly higher than the adopted annual average ambient standard ($20mm^3$ versus $16mm^3$).

MY NEIGHBOUR HAS CANCER: IS THERE A LINK?

Despite the findings of the risk assessment, some residents were concerned that recent cases of cancer in people living near the site were caused by the site remediation. These residents perceived that the number of people with cancer in their community was higher than in other areas. The CSPHU was asked to review the risk assessment and to conduct an epidemiological study of cancer in the area.

The CSPHU was aware that:

- local government maps of all cancer types from NSW Cancer Registry data, although crude measures,

showed no significant difference between the local government area and the rest of NSW;
- there was no evidence that emissions from cleaning up the site could initiate or promote cancer (in contrast to any potential risk associated with past employment at the gasworks when operational);
- studies of incidence of disease in relatively small populations around industrial sites rarely produce definitive results.

In consultation with others, we reviewed the risk assessment and concluded that the assessment methodology was sound and there was no evidence of long term risk to health for residents living near the site. Rather than commence a lengthy and costly study without scientific justification, the CSPHU focused on communicating information about cancer, and about actual emission concentrations leaving the site, to concerned residents at a community consultation forum convened by the local council. The CSPHU received feedback that this consultation was helpful in alleviating the concerns of some people.

Following the implementation of better odour mitigation strategies, the number of odour complaints decreased but did not abate entirely. Other issues, such as dust and truck movements, became relatively more important to residents as the remediation progressed.

DISCUSSION

Concerns about risks tend to be heightened by risks that are:

- involuntary or imposed;
- man made;
- inescapable;
- controlled by parties outside the community;
- exotic or unfamiliar;
- the cause of dreaded health effects, such as cancer.[4]

The notion of something being a risk differs by age, gender, ethnicity, income, education, political persuasion, values, and perceived benefits of the issue at hand.[5,6] Flynn postulates that 'power, status, alienation, and trust are strong determinants of people's perception and acceptance of risk'.[5]

Surveys in Australia have shown that people express substantial concern about exposure to chemicals, perceive chemicals as being predominantly dangerous, and make a conscious effort to avoid chemicals in their daily life.[5] The threat of exposure to chemicals, while a site is being cleaned up, is almost a recipe for community concern.

Despite this concern, the evidence of serious health outcomes for residents exposed to environmental levels of chemicals is small, although there are methodological difficulties associated with these assessments, including small sample sizes, difficulty in quantifying exposure, and the lack of relevant biomarkers. NSW does have relatively strong environmental legislation, and a solid infrastructure to protect residential health during clean-ups of former industrial sites. There is a paradox between the clean up of contaminated sites being perceived as dangerous and the lack of evidence establishing this danger.

In this case study, the perception of risk differed markedly between the stakeholders. Some of these perceptions can be characterised as follows:

- there are no toxic emissions leaving the site, so there are no real risks to health (environmental engineers);
- people are experiencing health effects, but they can be reassured that these will cause no long term biological damage (health agencies);
- the problem is not one of health risk but rather of nuisance (environmental agencies);
- the site poses much less risk to health now than it did when it was operating as a gasworks, and the current concern is a lot of fuss about nothing (residents);
- it is okay for the professionals to think there is no health risk, because they don't have to live here (residents);
- we are experiencing significant effects on our health, and no-one is taking our complaints seriously. In particular, psychological effects are being ignored (residents);
- the risks to health in the geographical area are so severe that the only option is to move away (residents).

Quantitative risk assessment, while an essential tool in the assessment of hazard, does not always address 'risk to health' as perceived by the community. In fact, critics of risk assessment argue that risk assessment, even more than other forms of scientific enquiry, purports objectivity while failing to acknowledge the inherent subjectivity of risk assessors. Scientists are not immune from perceiving risks according to their own worldview and this can frame the way that a risk assessment is conducted.

The underlying premise of these criticisms is that there is no universal definition of risk, and that risks may look very different to the people living near a site than they do to the risk assessors. Slovic argues that defining what is a risk is an exercise in power: 'Whoever controls the definition of risk controls the rational solution to the problem at hand. If risk is defined one way, then one option will rise as the most cost-effective or the safest or the best. If it is defined another way, perhaps incorporating qualitative characteristics and other contextual factors, one will likely get a different ordering of action solutions.'[7]

WHAT'S A PUBLIC HEALTH UNIT TO DO?

It is clear that the gap between what a community wants to know and what a risk assessment will tell them needs to be bridged. Neutra suggests that there is a fundamental difference between traditional epidemiology and what he calls 'dump-site epidemiology',[8] the investigation of health effects around waste disposal sites. In the latter, he argues, the decision to do a study is often made by the affected community and the audience really is that community, rather than other scientists. Rather than arguing that studies unlikely to produce statistically significant results should not be conducted, Neutra argues that affected people should be involved from the outset in specifying what answers the community wants and what level of uncertainty can be tolerated.

Where quantitative risk assessment or epidemiological studies are unlikely to resolve concern, some commentators have proposed that democratic models such as stakeholder-based decision-making should be used. In these models, prior to any investigation beginning, the focus is clearly on the values of stakeholders, important outcomes, and the probabilities of these outcomes. Citizen juries and consensus conferencing are two models that have been used in relation to environmental issues. Stakeholders may be asked to specifically consider the needs of the entire community.

It may be cost-effective and beneficial for health agencies to consider using such qualitative strategies to resolve environmental health issues. These have the advantage of not entering an argument about what is or is not a health risk, but rather focusing on outcomes that would be acceptable to all involved parties. While residents in this case study wanted to be certain that their future health was not at risk, they also just wanted the smells to go away.

One thing that health agencies should note is that the concept of 'community' is at times homogenising and misleading. It is impossible to alleviate the concerns of the entire community, and those people with the most local power are likely to get their concerns about risks addressed while other people remain unheard.

The use of alternative models to resolve environmental health issues, instead of or in addition to traditional risk assessment or epidemiological methods, is not a radical concept. It is what gets done in public health agencies daily in the name of risk communication and community consultation. What is different in the models above is that they involve more active input from the community and a relinquishing of some power from environmental health professionals and governments. They involve a specific shift from public health practitioners being the only ones who define what is 'risky' to health. This can be threatening, particularly when there are political and economic agendas associated with the definition of risk.

CONCLUSION

The meeting of the scientific paradigm of risk assessment with lay sensibilities of good health can lead to a traditional stand off between the rationality of science and the supposed irrationality of community sentiment. Quantitative risk assessment does not necessarily address threats to health as the community perceives them, although there is often pressure on health agencies to undertake 'health studies' of some sort, in the belief that this will provide objective, supportive evidence of the problems being experienced by the community.

The challenge for public health professionals is to combine the valuable data provided by the structured methodology of risk assessment (with its subjective assumptions) with qualitative approaches that recognise that risk is a contested term. The outcome should be meaningful results for all the stakeholders, including the people who live there.

ACKNOWLEDGEMENTS

The author would like to thank the following for their assistance and support: Mr David Donehue (AGL), URS Australia Pty Ltd, Dr David Douglas, Dr Leena Gupta, Mr Graham Burgess and Dr Michael Staff (Central Sydney Public Health Unit), Dr Vicky Sheppeard (NSW Department of Health), Ms Helen Davies, Mr Niall Johnstone and Mr John Sparkes (EPA), Dr Lillian Hayes, Canada Bay (previously Concord) Council, and the AGL Mortlake Community Liaison Committee.

REFERENCES

1. Douglas D. *AGL Mortlake Rehabilitation Project Environmental Health Issues*, 1 February 1999.
2. Ruth JH. Odor Thresholds and Irritant Levels of Several Chemical Substances: A Review. *Journal of the American Industrial Hygiene Association* 1986; 47(March).
3. Shusterman D. Odor-associated Health Complaints: Competing Explanatory Models. *Chemical Senses* 2001; 26: 339–343.
4. Starr G, Langley A, Taylor A. *Environmental Risk Perception in Australia—A Research Report to the Commonwealth Department of Health and Aged Care*. Adelaide: Centre for Population Studies in Epidemiology, South Australian Department of Human Services, 2000.
5. Flynn J, Slovi P, and Mertz CK. Gender, Race and perception of environmental health risks. *Risk Analysis* 1994; 14(6): 1101–8.
6. Alhakami AS, Slovic P. A psychological study of the inverse relationship between perceived risk and perceived benefit. *Risk Analysis* 1994; 14(6): 1085–96.
7. Slovic P. Trust, Emotion, Sex, Politics and Science: Surveying the Risk Assessment Battlefield. *Risk Analysis* 1999; 19(4): 689–701.
8. Neutra RR. Epidemiology for and with a Distrustful Community. *Environ Health Perspect* 1985; 62: 393–975.

RISK ASSESSMENT FOR THE CONSUMPTION OF FISH WITH ELEVATED SELENIUM LEVELS

Craig Dalton and Phillip Bird
Hunter Public Health Unit

This article describes an application of risk assessment for the consumption of fish with elevated selenium levels, from Lake Macquarie, which is in the Hunter region of New South Wales.

BACKGROUND TO THE INITIAL STUDY OF SEAFOOD IN LAKE MACQUARIE

Concern about possible health risks associated with heavy metal contamination of seafood in Lake Macquarie was highlighted, following the release of a study by Boyd Roberts in September 1995.[1] The study reported elevated concentrations of selenium (Se) in fish caught near power stations in the southern part of the lake in 1993. These results were corroborated by studies commissioned by the operator of the power station, Pacific Power. Because all of these studies focused on areas of the lake in the vicinity of industry, it was decided that a more broadly-based study should be commissioned to look at a range of heavy metals in fish caught at multiple sites that would be more representative of the entire lake.

A steering committee comprising representatives of the Hunter Public Health Unit, the NSW Environment Protection Authority (EPA), NSW Fisheries, Pacific Power, Delta Power, Pasminco smelter, Lake Macquarie Council, commercial fishermen, recreational fishermen, and consultants from the University of Newcastle, developed a protocol for sampling fish from 10 separate sites in the lake, including some sites that were not in the immediate vicinity of any heavy industry.

Five different species of fish were sampled in each site in three different weight categories. Five fish in each species–weight category were to be caught and tested under the protocol. The five fish in each category constituted a 'batch'. The species sampled included Sea Mullet, Black and Yellow Fin Bream, Dusky Flathead, Trumpeter Whiting, and Luderick. The flesh from each batch was homogenised and tested for selenium, copper, lead, arsenic, cadmium, zinc, and mercury. Analysis of the data revealed that selenium was elevated in a wide range of fish caught in many sites throughout the lake.

A risk assessment for consumption of fish from Lake Macquarie was conducted, to determine the safe level of fish consumption for people regularly consuming fish from the lake over a lifetime. This risk assessment used a maximum safe intake of selenium of 0.005 mg/kg/day.

METHODS

To determine the maximum allowable intake of selenium, we reviewed recommendations from selected agencies around the world: the Agency for Toxic Substances and Disease Registry (ATSDR), Centers for Disease Control and Prevention, US Public Health Service;[2] the United States Environmental Protection Agency (US EPA);[3] the World Health Organization (WHO);[4] and the National Health and Medical Research Council (NHMRC), Australia (Table 1).[5]

The ATSDR and US EPA levels are based on a 'no observed adverse effect level' (NOAEL) of an intake of 0.015 mg Se/kg/day or 0.85 mg Se/day, based on Chinese subjects who had an average weight of 55 kg, established in a study by Yang et al.[6] Both the ATSDR and the US EPA divided the NOAEL by an uncertainty factor of three to allow for sensitive individuals, giving a maximum safe level of intake of 0.005 mg Se/kg/day. In 1994, an Expert Consultation between the Food and Agriculture Organization of the United Nations and the WHO,

TABLE 1

MAXIMUM ALLOWABLE INTAKE OF SELENIUM RECOMMENDED BY SELECTED INTERNATIONAL AGENCIES

Agency	Criteria	Upper estimated safe level chronic oral ingestion
ATSDR*	Minimal risk level	0.005 mg/kg/day
US EPA†	Reference dose	0.005 mg/kg/day
WHO**	Maximal daily safe intake	0.4 mg/day for adult of 55 kg (0.007 mg/kg/day)
NHMRC‡	Adverse effect level	0.4–0.6 mg/day for adult (0.006–0.009 mg/kg/day)

* Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, US Public Health Service.[2]

† United States Environmental Protection Agency.[3]

** World Health Organization.[4]

‡ National Health and Medical Research Council, Australia.[5]

accepted the NOAEL established by Yang et al. and their suggestion that a maximal safe dietary intake of selenium be set at 0.4 mg per day based on an uncertainty factor of two. For a 55 kg adult, this equals 0.007 mg/kg/day.

The end points of interest in the Yang et al. study were toenail changes consistent with selenosis. Although this may appear to be a minor health event, it is evidence of disordered function at the cellular level. There may be other adverse affects that are not clinically observable. There is evidence of effects on glutathione peroxidase activity, with selenium intake around 0.85 mg per day, again suggesting some alteration of function at the cellular level.

In the study by Yang et al.,[6] which formed the basis of the NOAEL, the actual proportion of ingested selenium that was absorbed was not known. In the Lake Macquarie risk assessment, no adjustments were made for absorption as it was assumed that the same proportion of selenium was absorbed from the fish as was absorbed from the diet of the subjects in the Yang study. The NOAEL is based on the level of selenium ingested, not the level absorbed. However, there may be differences in the level of absorption of selenium from Lake Macquarie fish that could bias the assessment in either direction. Absorption from some fish has been shown to be as low as 20 per cent in animal models; however, it is not known how this correlates with absorption by humans.

TABLE 2

CALCULATION FOR MAXIMUM SAFE INTAKE OF FISH FROM LAKE MACQUARIE FOR 70 KG ADULT, VALUE OF VARIABLES USED IN RISK ASSESSMENT

Variable	Value	Source
Mean selenium level in fish from Lake	1.2 mg/kg	Lake Macquarie Seafood study 1996
Mean intake of fish/day for adult = 11 g/day	77 g/week	National Dietary Survey, 1983
Mean selenium levels in fish in Australia	0.6317 mg/kg	Australian Market Basket Survey, 1994
Adult weight	70 kg	
Maximal safe weekly intake of selenium = 0.005 mg/kg/day	2.45 mg/ week for 70 kg adult	US EPA,†ATSDR,*
Total average weekly dietary intake of selenium per kg body mass, Australian male.	0.0125 mg/kg/week	Australian Market Basket Survey, 1994
Total average weekly dietary intake of selenium for a 70 kg Australian adult male	0.0125 x 70 = 0.875 mg/week	Calculation from above figure
Toxic threshold from lowest -observed-adverse-effect-level.(LOAEL)	0.016mg/kg/day	Yang et al.[6]
Toxic threshold for 70 kg adult per week	0.016 mg/kg/day x 70 kg x 7 days = 7.84 mg/week	Calculation from above figure

* Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, US Public Health Service

† United States Environmental Protection Agency

Background intake of selenium = total intake – fish contribution

Fish contribution = mean intake/week x mean selenium in Australian fish

$$= \frac{77 \text{ g} \times 0.6317}{1000}$$

= 0.0486 mg/week for 70 kg adult

Background intake (excluding fish) = 0.875 – 0.0486 = 0.8264 mg/week

Toxic threshold of intake of fish from Lake

$$= \frac{\text{Weekly intake at LOAEL for 70 kg adult} - \text{Background intake}}{\text{Mean selenium level in fish in Lake}}$$

$$= \frac{7.84 \text{ mg} - 0.8264 \text{ mg}}{1.2 \text{ mg/kg}}$$

= 5.85 kg

Allowable intake of fish from Lake

$$= \frac{\text{Maximal safe weekly intake} - \text{Background intake}}{\text{Mean selenium level in fish from Lake}}$$

$$= \frac{2.45 \text{ mg} - 0.8264 \text{ mg}}{1.2 \text{ mg/kg}}$$

= 1.35 kg/week

Another study that supports the safety, and likely conservative nature, of an intake of 0.005 mg/kg/day was conducted in an area of the United States with high selenium levels in soil and diet.[7] It revealed no morbidity in 76 subjects with a mean daily selenium intake of 0.239 mg, including 12 subjects with an intake in excess of 0.4 mg/day and one subject with an intake of 0.724 mg/day.

RESULTS

The findings of this risk assessment suggest that a 70 kg adult would be able to consume 1.35 kg of Lake Macquarie fish per week if they had the mean dietary intake of selenium from other sources found in the 1994 Australia New Zealand Food Authority's Market Basket Survey (Table 2).

DISCUSSION

The risk assessment based on conservative but reasonable assumptions allows an intake of fish from Lake Macquarie of 1.35 kg, which is more than 17 times the mean national weekly intake of 77 grams. This level of intake is only likely to occur among people and their families who fish commercially or recreationally from Lake Macquarie.

When this risk assessment was conducted the standard for selenium in food in Australia was 1mg/kg; however, this was under review as it was not based on health risk assessment. In the interim we thought it was appropriate to develop dietary guidelines for consumers of Lake Macquarie fish based on health risk assessment. This risk assessment allows a 70 kg adult to consume 1.35 kg of fish per week continuously for a lifetime. This allows for much greater than 1.35 kg of fish in any given week as long as the average is below 1.35 kg. It must be emphasised that this is the upper limit of safe intake, not the threshold for toxicity. The conservative assumptions, and the threefold safety factor used in deriving a safe upper limit intake of 0.005 mg/kg, should ensure that the threshold for toxicity in the average person is much higher than this.

No evidence for increased sensitivity to selenium in children or pregnant women exists so their intake of Lake Macquarie seafood should be based on the same risk assessment.[2,6] Smaller adults and children should consume fish in proportion to their weight; that is, a 35 kg child should be allowed 675 grams of fish, or half of the 1.35 kg allowed a 70 kg adult.

There was a limited number of Blue Swimmer Crabs, and no prawns were caught for this study. The mean selenium levels in the Blues Swimmer Crabs were similar to that in fish with fins, and in prawns, which have been found to have similar levels of selenium to fish with fins caught in the same location in other studies. Therefore, it would be reasonable to allow a total intake of 1.35 kg for all fish, crabs, and prawns per week from Lake Macquarie for a 70 kg adult until further data is available.

Because selenium and other metals may accumulate in the internal organs of fish,[1] consumers should be discouraged from consuming internal organs or using whole fish as stock.

Fact sheets informing fisherman of the outcome of the risk assessment, and the advice to eat only 1.35 kg per week, were distributed through fishing cooperatives, bait and tackle shops, and through public meetings; a press release and media conference received widespread media coverage. While not formally assessed the public appeared to accept the risk assessment and no groups disputed the process or the findings. Since the study, commercial fishing has been banned in Lake Macquarie, due to depletion of fish stocks and not for reasons of public health.

REFERENCES

1. Roberts B. The accumulation and distribution of selenium in fish from Lake Macquarie, NSW. Canberra: Faculty of Applied Science, University of Canberra, 1994 (Master's thesis).
2. Agency for Toxic Substances and Disease Registry. *Toxicological profile for selenium*. Atlanta: United States Department of Health and Human Services, Public Health Service, 1996.
3. Poirer K.A. Summary of the derivation of the reference dose for selenium. Mertz W, Abernathy CO, Olin SS (editors). *Risk assessment of essential elements*. Washington DC: ILSI Press, 1994; 57–165.
4. WHO. Chapter 6. Selenium. *Trace elements in human nutrition and health*. Geneva: World Health Organization, 1996.
5. NHMRC. *Recommended Dietary Intakes for use in Australia*. Canberra: National Health and Medical Research Council, Australian Government Publishing Service, 1991.
6. Yang G, Yin R, Zhou L et al. Studies of safe maximal daily dietary Se-intake in a seleniferous area in China. Part II: Relation between Se-intake and manifestation of clinical signs and certain biochemical alterations in blood and urine. *J Trace Elem Electrolytes Health Dis* 1989; 3: 123–30.
7. Longnecker MP, Taylor PR, Levander OA, et al. Selenium in diet, blood and toenails in relation to human health in a seleniferous area. *Am J Clin Nutr* 1991; 53: 12988–94.

TAKING THE PLUNGE: RECREATIONAL WATER QUALITY GUIDELINES

Cris Hickey
Beachwatch Programs
NSW Environment Protection Authority

Christine Cowie
Water Unit
NSW Department of Health

During the 1980s, the profile of recreational water quality at Sydney's beaches caused concern among Sydneysiders. Monitoring of recreational water quality indicated that most Sydney beaches had poor water quality, and this condition was attributed to the disposal of partially-treated sewage at cliff-face discharges.[1] An epidemiological study conducted in 1989–1990 attempted to determine the prevalence of disease that might have been attributed to the water quality at the time.[2] Since then, major improvements to sewage disposal practices in Sydney have seen corresponding improvements in water quality.[3]

This article describes the current move in Australia, and by the World Health Organization (WHO), to review existing recreational water quality guidelines. Anticipating the release of new draft National Health and Medical Research Council (NHMRC) guidelines for recreational water quality, which are likely to be influenced by the WHO guidelines, the approach was trialled by applying it to data collected by Beachwatch. Beachwatch is the recreational water quality monitoring program administered by the NSW Environment Protection Authority. The different approaches to estimating the health risk produced different results for the water quality at 35 Sydney beaches. Possible reasons for these differences are discussed.

HEALTH EFFECTS ASSOCIATED WITH RECREATIONAL WATER QUALITY

Recreational exposure to contaminated beach water has been associated with gastroenteritis, respiratory illness, eye infections, ear-nose-throat infections, and skin and mucosal infections.[4] A review of the literature conducted in 1998, on behalf of the WHO, evaluated the health risk attributable to recreational water quality.[5] The author reviewed 22 of 36 studies that met specific epidemiological criteria, two of these studies being randomised controlled trials (RCT). The two RCTs reported threshold levels of >32 faecal streptococci/100 mL for increased risk of gastroenteritis, 60 faecal streptococci/100 mL for acute febrile respiratory illness, and 100 faecal coliforms/100 mL for ear ailments. Gastrointestinal symptoms were the most common outcome for which significant dose–response relationships were reported in the WHO review. One overseas study reported higher attack rates for gastroenteritis in visitors to a locality compared to the resident population, suggesting that immune status may play a role in the presentation of illness. This suggests that populations may differ in their susceptibility to waterborne diseases.

TABLE 1

RECREATIONAL WATER QUALITY GUIDELINES IN USE IN NSW AND AUSTRALIA

Guideline	Requirement for Safe Swimming for each Indicator Bacteria	
	Faecal coliforms	Enterococci
NHMRC, 1990 [6]	median value <150 cfu/100mL for a minimum of 5 samples taken at regular intervals not exceeding 1 month AND 4 out of 5 samples <600 cfu/100mL *	geometric mean of 33/100mL for marine waters
ANZECC, 1992, 2000 [7]	median value <150 cfu/100mL for a minimum of 5 samples taken at regular intervals not exceeding 1 month AND 4 out of 5 samples <600 cfu/100mL	median value <35/100mL for a minimum of 5 samples AND 60–100/100mL maximum number in any one sample
Beachwatch [3]	median value <150 cfu/100mLfor a minimum of 5 samples taken at regular intervals not exceeding 1 month AND 4 out of 5 samples <600 cfu/100mL	median value <35/100mL for a minimum of 5 samples taken at regular intervals not exceeding 1 month AND 4 out of 5 samples = or <100/100mL

* cfu/100mL = colony forming units per 100 millilitres of water.

Although indicator organisms used in the WHO review studies varied, the organisms that correlated best with disease outcomes were enterococci and faecal streptococci for both marine and freshwaters, and *E. coli* for freshwater.[5] However, correlations were also reported for faecal coliforms and staphylococci.[5]

RECREATIONAL WATER QUALITY GUIDELINES

Although many of the symptoms associated with recreational water exposure are due to infection by enteric viruses, for pragmatic reasons recreational water quality is determined by 'indicator' bacterial organisms. Three recreational water quality guidelines currently used in NSW and Australia are listed in Table 1. Although quite similar, there are subtle differences in terms of frequency of monitoring and the statistics used. Beachwatch uses a combination of both NHMRC guidelines and Australian and New Zealand Environment Conservation Council (ANZECC) guidelines.[6,7]

APPLICATION OF THE WHO DRAFT GUIDELINES: METHODOLOGY AND RESULTS

Draft recreational water quality guidelines were released by WHO in 1998,[8] and more recently by the United States Environmental Protection Agency.[9] The NHMRC generally uses the WHO guidelines as a basis for developing or reviewing Australian guidelines, and is currently reviewing the national recreational water quality guidelines.

The draft WHO guidelines enable water managers to set guideline values for swimming, based on the risk of beach users becoming ill. The values are determined using a known relationship between bacterial density and illness rates (the dose–response relationship), and the distribution of bacterial levels at a swimming site or representative group of sites (the probability distribution function, or pdf). The values can then be used to develop a beach classification system that promotes informed choice as a risk management strategy.

The WHO guidelines use the dose–response relationship derived from one of the RCT studies cited in the WHO review, conducted in English waters by Kay et al.[10] This study reported a threshold level of >32 faecal streptococci/100 mL for increased risk of gastroenteritis. While the dose–response curve from this study is significantly steeper, and the threshold level lower, than those reported in previous studies, it is accepted by WHO on the basis that the study's robust epidemiological design minimises misclassification and more accurately measures the association between water quality and illness.[8]

The WHO approach was trialled,[8] using data collected at 35 Sydney beaches under the Beachwatch Program.[3] The Beachwatch Program measures levels of thermotolerant coliforms and enterococci only. For the purposes of this exercise, it was assumed that levels of enterococci in marine waters closely approximate levels of streptococci. This assumption is supported by the rapid die-off rate of the two streptococci species not included in the enterococci group.

A pdf for Sydney beaches was generated from data collected over the 1999–2000 summer season. Guideline values were then generated using the WHO methodology and these are listed in Table 2. Interestingly, the pdf distribution and guideline values for Sydney beaches were similar to those determined by WHO for European waters.[8]

WHO notes that its derived guideline values represent better water quality than presently encountered at many beaches worldwide.[8] Table 3 indicates that this is the case for Sydney beaches, with many beaches that currently have high compliance with existing water quality guidelines (100 per cent compliance) receiving B and C classifications when the WHO dose–response relationship is utilised.

DISCUSSION

Before applying the guidelines to a specific area, WHO recommends that a wide range of social, environmental, cultural, and technical issues be considered, such as the

TABLE 2

EXAMPLE OF CLASSIFICATION SYSTEM FOR BEACHES BASED ON WHO DRAFT GUIDELINES, SYDNEY, NSW

Classification	Enterococci density at 95th percentile	Illness rates	Contamination
A	Less than 14 cfu/100 mL *	< 2.5/1000	Low
B	14 to 49 cfu/100 mL	2.5–12.5/1000	⇓
C	50 to 198 cfu/100 mL	12.5–50/1000	Medium
D	199 to 1000 cfu/100 mL	> 50/1000	⇓
E	Greater than 1000 cfu/100 mL	Public health risk requiring immediate investigation	High

Source: Based on *World Health Organization Guidelines for Safe Recreational-water Environments: Coastal and Freshwaters. Draft for Consultation*, Geneva, October 1998.[8]

* cfu/100mL = colony forming units per 100 millilitres of water.

TABLE 3

CLASSIFICATION OF BEACHES AND PER CENT COMPLIANCE WITH BEACHWATCH GUIDELINES FOR ENTEROCOCCI DURING THE SUMMER SEASON, SYDNEY, NSW, 2001–2002

Beach	Enterococci 95 percentile (cfu/100mL) **	Classification using WHO categories	% Compliance with Beachwatch guidelines *
Palm Beach	74	C	100
Whale Beach	16	B	100
Avalon	32	B	100
Bilgola	26	B	100
Newport	22	B	100
Bungan	16	B	100
Mona Vale	14	A	100
Warriewood	28	B	100
Turimetta	22	B	100
Nth Narrabeen	14	A	100
Collaroy	50	B	100
Long Reef	6	A	100
Dee Why	110	C	100
Nth Curl Curl	54	C	88
Sth Curl Curl	16	B	100
Freshwater	100	C	97
Queenscliff	80	C	94
Nth Steyne	78	C	97
Sth Steyne	80	C	97
Shelly Beach (Manly)	84	C	88
Bondi	80	C	88
Tamarama	100	C	84
Bronte	60	C	100
Clovelly	120	C	75
Coogee	120	C	84
Maroubra	110	C	78
Malabar	170	C	84
Boat Harbour	130	C	72
Greenhills	16	B	100
Wanda	24	B	100
Elouera	26	B	100
Nth Cronulla	34	B	100
Sth Cronulla	40	B	100
Shelly Beach (Sutherland)	44	B	100
Oak Park	86	C	100

* Source: Beachwatch. *Beachwatch and Harbourwatch 2001–2002 State of the Beaches Report*. Sydney, NSW Environment Protection Authority, 2003.[3]

** cfu/100mL = colony forming units per 100 millilitres of water.

nature and seriousness of local endemic illness, population behaviour, and exposure patterns. Three key issues that should be considered, when results from the two methods are compared, are outlined below.

1. Is the 95th percentile an appropriate statistic?

Beachwatch collects samples every six days at Sydney beaches. Elevated bacterial counts are most frequently recorded during and immediately after heavy rainfall. As the pdf of bacterial data for Sydney beaches includes bacterial levels collected during wet weather, the 95 percentile represents the poorer water quality during wet weather. Anecdotal evidence indicates that most of the community generally does not swim during or immediately after rainfall, and it may be therefore inappropriate to determine health risk and a beach classification based on this statistic.

2. Is the WHO dose–response relationship appropriate?

WHO notes that the dose–response curve developed by Kay et al. may not cover all global climatic conditions nor all recreational water types.[8] As Kay's study was conducted in northern European waters,[10] it is possible

that this dose–response relationship is not applicable for the Sydney region where a threshold more representative of warmer waters may be more appropriate. Varying climatic and oceanographic conditions such as differing water temperatures can effect the spatial distribution and survival of pathogens in bodies of water.[11]

Sydney also has an extended swimming season (from beginning of October to end of April), compared to northern Europe, with many beachgoers visiting beaches frequently during the season. As a result of this greater exposure (longer and more often), it is possible that Sydney swimmers may have higher or a different immune response to swimming-associated illness.[2]

3. Is faecal streptococci the best indicator?

The results of the Sydney Beach Users Study differ from Kay et al. in that faecal coliforms were found to be a better predictor of reported symptoms than were faecal streptococci.[2] The study found that swimmers were almost twice as likely than non-swimmers to report symptoms, and that there was evidence of increasing reporting of symptoms for all symptoms (other than gastrointestinal symptoms) with increasing bacterial counts, suggesting a dose–response relationship.

CONCLUSION

As the success of the WHO approach relies on a dose–response relationship that accurately defines the illness rates associated with swimming for a specific population, it may not be appropriate to apply the WHO methodology in NSW before this relationship is accurately defined by a robust epidemiological study. Such studies are, however, costly and resource intensive to conduct. Further, it is anticipated that the application of the WHO guideline methodology could be onerous for many local councils to implement.

Other factors that need to be considered before applying the WHO guidelines are the levels at which acceptable or tolerable excess disease rates are set for the NSW community, and the pattern of variability in the distribution of bacterial levels at Sydney beaches over time.

REFERENCES

1. Kueh CSW, Grohmann GS. Recovery of viruses and bacteria in waters off Bondi beach: A pilot study. *Med J Aust* 1989; 151: 632–638.
2. Corbett SJ, Rubin GL, et al. The health effects of swimming at Sydney beaches. The Sydney Beach Users Advisory Group. *Am J Public Health* 1993; 83:1701–06.
3. Beachwatch. *Beachwatch and Harbourwatch 2001–2002 State of the Beaches Report*. Sydney, NSW Environment Protection Authority, 2003.
4. World Health Organization. *Bathing Water Quality and Human Health. Protection of the Human Environment—Water, Sanitation and Health*. Geneva: World Health Organization, 2001.
5. Pruss A. Review of epidemiological studies on health effects from exposure to recreational water. *Int J Epidemiology* 1998; 27(1): 1–9.
6. National Health & Medical Research Council. *Australian guidelines for recreational use of water*. Canberra: National Health & Medical Research Council, 1990.
7. Australian & New Zealand Environment & Conservation Council, Agriculture & Resource Management Council of Australia & New Zealand. *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*. Canberra: Environment Australia, 2000.
8. World Health Organization. *Guidelines for Safe Recreational-water Environments: Coastal and Freshwaters: Draft for Consultation*. Geneva: World Health Organization, 1998.
9. US Environmental Protection Agency. *Implementation Guidance for Ambient Water Quality Criteria for Bacteria. Draft*. Washington DC: US Environmental Protection Agency, 2002.
10. Kay D, Fleisher JM et al. Predicting likelihood of gastroenteritis from sea bathing: Results from randomised exposure. *Lancet* 1994; 344: 905–09.
11. Mourino-Perez RR. Studying the risks of ocean swimming. *Epidemiology* 1999; 10(4); 351–352.

FACT*SHEET*

I N F L U E N Z A

WHAT IS INFLUENZA?

Influenza (known as the flu) is a highly contagious acute respiratory illness caused by influenza viruses A, B, and rarely C.

HOW IS INFLUENZA SPREAD?

The virus is spread from person-to-person through microscopic droplets, when an infected person coughs or sneezes. It is easier to 'catch' influenza in crowded areas and in confined spaces.

WHAT ARE THE SYMPTOMS?

Symptoms include the sudden onset of: fever, headache, muscle and joint pain, feeling tired, sore throat, cough, runny or stuffy nose, and often extreme fatigue. Symptoms usually appear within 1–3 days of the person being infected. A person is considered contagious for another 3–4 days after symptoms appear. Most people recover within 2–7 days. Compared with other viral respiratory infections, such as common colds, influenza causes more severe complications such as pneumonia—particularly in children, elderly people, and other vulnerable groups.

HOW CAN INFLUENZA BE TREATED?

New medications for the treatment of influenza can be effective in reducing the severity and the duration of the illness. These must be taken early in the illness to be effective and are only available on prescription from your doctor. Otherwise, fever, headaches, and muscle pains, can be treated with fluids, paracetamol, and rest.

HOW CAN INFLUENZA BE PREVENTED?

Vaccination remains the most effective protection against influenza infection. Anyone who wishes to avoid the flu should think about getting vaccinated well before winter begins each year. Influenza vaccination is recommended for:

- all adults aged 65 years and over;
- Aboriginal and Torres Strait Islander people aged 50 years and over;
- adults and children older than six months with chronic diseases affecting the heart or lungs;
- adults, and children older than six months of age, with other chronic illnesses that require regular medical follow up;
- residents of nursing homes and other long-term care facilities;
- persons with immunodeficiency, including HIV–AIDS;
- adults and children older than six months who live in a household with a person who fits into any of the above categories;
- healthcare workers, and staff of nursing homes and long term care facilities, who look after people at high risk;
- children and teenagers (six months to 18 years) on long-term aspirin therapy;
- travellers, especially those in the above risk groups, if travelling to the northern hemisphere between October and March, should consider having an influenza vaccination prior to departure;
- women who will be pregnant in the second or third trimester during the influenza season.

WHEN SHOULD I BE VACCINATED?

The best time to be vaccinated against influenza is in autumn, prior to the winter influenza outbreaks. The vaccine is usually available from March each year.

WHERE CAN I RECEIVE MY VACCINATION?

Your doctor can vaccinate you with the current vaccine for the season.

WILL I HAVE TO PAY FOR THE VACCINE?

If you are 65 years or older, or are an Aboriginal or Torres Strait Islander aged 50 years or older, the vaccine will be free. However, the doctor may charge a consultation fee.

IS THE VACCINE SAFE?

Yes. The most frequent side effect of vaccination is soreness at the vaccination site, which may last up to 2 days. Influenza-like symptoms such as fever, fatigue, and muscle soreness, can also occur. These symptoms mimic the flu. Other serious side effects are rare.

IS IT POSSIBLE TO CATCH THE FLU FROM BEING VACCINATED?

No. The vaccine contains killed virus that cannot cause influenza.

HOW EFFECTIVE IS THE VACCINE?

It will take about two weeks for your body to develop immunity against the influenza virus after your vaccination. During this time you should avoid contact with people who may have influenza. The influenza virus changes from time to time and the vaccine is designed to match the current virus that is circulating among the population. The vaccine will provide about 70 per cent protection against infection for about one year. However, even if you do catch the flu, the likelihood of developing complications from the infection will be reduced.

WHO SHOULD NOT HAVE THE VACCINATION?

Those who should not be vaccinated are:

- people with allergies to eggs;
- people with a high fever (greater than 38.5° C) should wait until their fever has gone;
- people who have previously had Guillain Barré syndrome should discuss this with their doctor prior to proceeding with vaccination;
- children younger than six months of age.

DO I NEED TO RECEIVE A FLU VACCINE EVERY YEAR?

Yes. Annual vaccination is necessary to provide continuing protection against the most recent influenza virus.

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

August 2003.

NSW HEALTH Working as a Team

COMMUNICABLE DISEASES REPORT, NSW, FOR JUNE 2003

TRENDS

Summaries of case notifications through to June 2003 are shown in Figure 1 and Table 2. Note that for Figure 1, at the time of reporting, notifications for the Mid North Coast Area Health Service were not available for June because of local database replication errors within the Area.

Notifications of **Ross River** and **Barmah Forest virus** infection peaked in May, and declined in June with the onset of cooler weather. Most cases have been reported from the northern coastal areas of the state.

Two cases of **measles** were reported in June, as part of a cluster centred in the Wentworth Area. A summary of the outbreak, which began with a traveller who acquired the infection in Nepal, will be reported in a forthcoming issue of the *NSW Public Health Bulletin*. Although the proportion of people immunised against measles is probably at an all time high—unpublished data from the Australian Childhood Immunisation Register indicates that in June 2003, 94 per cent of NSW children aged between 2 years and 2 years and 3 months had received measles vaccination—the absence of complete immunisation and the relatively low levels of naturally circulating virus in NSW since the mid 1990s will lead to an increase in the number of susceptible people in the community over time. After a long period of quiescence, therefore, measles may start to re-emerge in NSW. Clinicians should make every effort to ensure all patients are fully up-to-date with measles vaccination, and remain alert for possible measles cases in people presenting with fever and a rash.

The Wentworth Public Health Unit reported a cluster of **pertussis** at a school in the upper Blue Mountains. There were eight children and one teacher with confirmed pertussis. Of the children, four were not immunised (aged from 6–9 years) and one had an uncertain immunisation history. There were two cases with onset in May, six cases with onset in June, and one case with onset in early July.

Viral gastroenteritis is commonly reported in the winter months. In June, five institutional outbreaks were reported from four health areas: South Eastern Sydney reported an outbreak of 23 cases from one hospital; Mid North Coast reported an outbreak of 19 cases in one hospital; Northern Rivers reported an outbreak of 16 cases in a nursing home; and Central Coast reported outbreaks in one nursing home and one hospital, involving a total of 27 cases. The Communicable Diseases Branch of the NSW Department of Health is currently developing a protocol and resources for the investigation of gastroenteritis in institutions.

Four cases of **listeriosis** were reported in June, continuing the sustained increase in notifications during 2003. To date, 16 cases have been notified this year, compared with 12 cases notified in 2002 and 13 cases notified in 2001. The majority of cases are in people with underlying immunocompromising conditions. The most recent cases were in the Central Coast, Illawarra, Macquarie, and South Western Sydney Areas. No links have been identified among cases.

Reports of invasive **pneumococcal disease** increased with the onset of winter, in line with seasonal expectations.

INFLUENZA SURVEILLANCE

The NSW Influenza Surveillance Program began in May and will continue through to the end of the first week in October 2003. This year data sources include:

- clinical reports of influenza-like illness (ILI) by NSW general practitioners from the Australian Sentinel Practice Research Network (ASPREN), and five Public Health Units (Central Coast, Illawarra, New England, Northern Sydney, and Southern NSW);
- virological and serological reports of influenza, parainfluenza, adenovirus, rhinovirus, and respiratory syncytial virus (RSV) by major public laboratories: South Eastern Area Laboratory Services (SEALS), Institute of Clinical Pathology and Medical Research (ICPMR), South Western Area Pathology Service (SWAPS), Pacific Laboratory Medicine Services (PaLMS), Hunter Area Pathology Service (HAPS), and the New Children's Hospital (NCH);
- the Directed Virological Surveillance (DVS) scheme, involving general practitioners from metropolitan and rural area health services who submit samples from patients with ILIs for viral testing at SEALS and ICPMR;
- international and national influenza activity regularly updated from the WHO Collaborating Centre for Reference and Research on Influenza, Melbourne, at www.influenzacentre.org;
- National Notifiable Diseases Surveillance System, Australian Department of Health and Ageing, at www.health.gov.au.

Through to the end of June 2003, little influenza activity had been reported by laboratories and low rates of ILIs were reported by general practitioners. Towards the end of June there was a modest increase in influenza A reported by laboratories. RSV infection was the major cause of ILIs through to the end of June.

TABLE 1

CHARACTERISTICS OF MENINGOCOCCAL CASES, NSW, 2001–2003

Case	Characteristics	Week ending 4 July 2003	1Jan–4July 2003	Total 2002	Total 2001
Serogroup *	B	0	35	104	92
	C	1	11	53	38
	Other–Unknown	3	26	57	103
Gender	Male	2	38	124	111
	Female	2	34	90	122
Age Group	0–4	2	24	57	71
	5–14	0	5	44	33
	15–24	1	18	61	47
	25–44	0	9	26	52
	>45	1	16	26	30
Residence **	Sydney Area	3	31	126	129
	Other	1	41	88	104
Deaths		0	2	19	7
Total		4	72	214	233

Source: Data is based on date of onset and excludes cases of meningococcal conjunctivitis.

* Serogrouping of cases may change from unknown to a serogroup as laboratory results become available.

* Other serogroups include A, W135, and Y.

**Sydney area covers Central Sydney, Northern Sydney, South Eastern Sydney, South Western Sydney, Western Sydney and Wentworth Area Health Services.

MENINGOCOCCAL DISEASE

Each year, meningococcal disease affects between 200–250 people in NSW. It occurs more commonly in winter and early spring. Those most at risk are close contacts of other cases, young children, and young adults. Up to 10 per cent of patients with meningococcal disease die as a result of the disease. In NSW, group B meningococcal bacteria are responsible for about half of the cases of meningococcal disease and group C is responsible for about one third of cases. Hospitals and laboratories are required to notify their local public health unit as soon as a provisional diagnosis of meningococcal disease is made.

As of 4 July 2003, there were 72 cases of meningococcal disease notified in NSW for 2003, including two deaths. Of these 72 cases:

- 35 (49 per cent) had group B disease, 11 (15 per cent) had group C disease, and 26 (36 per cent) had other or unknown serogroups;
- 38 (53 per cent) were male and 34 (46 per cent) were female;
- 24 (33 per cent) were aged up to four years, five (seven per cent) were aged 5–14 years, 18 (25 per cent) were aged 15–24 years, nine (12 per cent) were aged 25–44 years, and 16 (22 per cent) were aged over 45 years.

Recorded information about meningococcal disease is available by calling 1800 150 061. A fact sheet on meningococcal disease can be found at: www.health.nsw.gov.au/public-health/cdscu/facts/pdf/meningococcal.pdf.

Updates on meningococcal disease incidence in NSW can be found at: www.health.nsw.gov.au/public-health/alerts/meningococcal/index.html

UPDATE ON INVESTIGATIONS OF SEVERE ACUTE RESPIRATORY SYNDROME

The worldwide epidemic of Severe Acute Respiratory Syndrome (SARS) continued in June; however, there were signs that international disease control measures were effective. By 30 June 2003, 8,447 probable SARS infections and 811 deaths had been reported to the World Health Organization (WHO), and only Toronto and Taiwan were considered to be 'SARS-affected' areas.

In NSW there were no further notifications of possible SARS cases in June. Australia had reported a total of five probable cases, one of whom was from NSW. No laboratory-confirmed cases of SARS were reported in Australia through to the end of June 2003.

FIGURE 1

REPORTS OF SELECTED COMMUNICABLE DISEASES, NSW, JANUARY 1998 TO JUNE 2003, BY MONTH OF ONSET

Preliminary data: case counts in recent months may increase because of reporting delays.
Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis
BFV = Barmah Forest virus infections, RRV = Ross River virus infections
LI = Legionella longbeachae infections, Lp = L. pneumophila infections
Gp C and Gp B = disease due to serogroup C and serogroup B infection,
other/unk = other or unknown serogroups

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

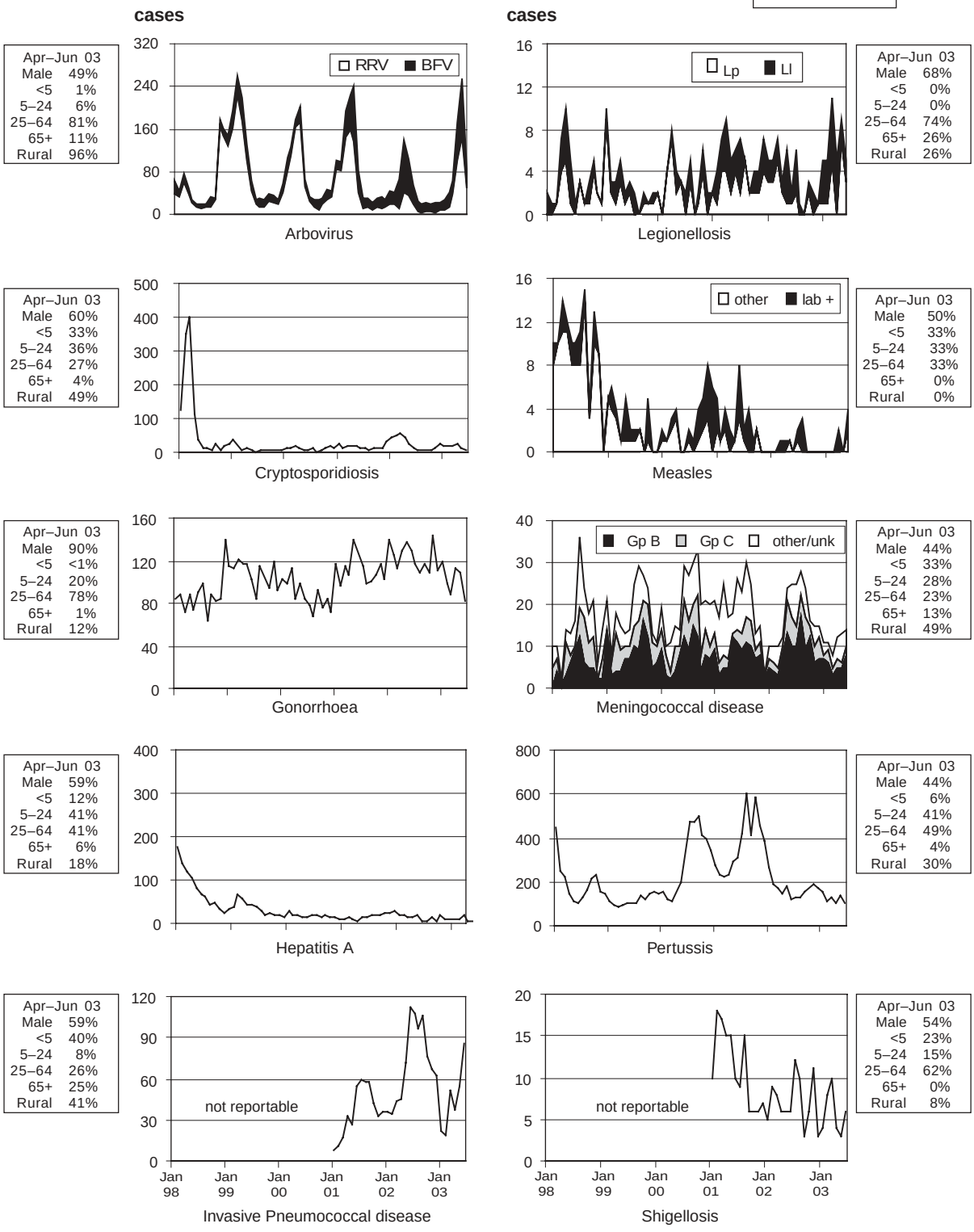

TABLE 2 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN JUNE 2003 BY AREA HEALTH SERVICES

Condition	Area Health Service																		Total	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	CHS	for June†	To date†
Blood-borne and sexually transmitted																				
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	58	49	51	25	41	13	50	24	95	28	15	23	15	18	2	36	12	-	562	3,426
Gonorrhoea*	18	16	7	1	5	1	1	-	38	6	3	2	-	-	-	-	1	-	103	669
Hepatitis B - acute viral*	-	-	-	-	2	-	-	-	-	-	-	-	1	-	-	-	-	-	3	40
Hepatitis B - other*	49	34	56	5	1	4	1	4	35	3	1	3	-	2	-	-	1	-	201	1,593
Hepatitis C - acute viral*	2	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	-	4	47
Hepatitis C - other*	59	32	52	19	3	36	40	25	71	32	32	9	6	14	1	11	7	-	453	3,236
Hepatitis D - unspecified*	-	1	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	2	5
Syphilis	15	9	4	2	12	2	1	2	18	5	1	5	2	-	1	-	-	-	80	445
Vector-borne																				
Barmah Forest virus*	1	-	1	-	-	1	1	-	-	49	20	1	-	1	2	-	-	-	77	334
Ross River virus*	1	-	-	-	-	2	4	-	1	63	29	1	-	1	4	-	-	-	106	414
Arboviral infection (Other)*	1	-	-	-	2	-	1	-	-	2	-	-	-	-	-	-	-	-	8	54
Malaria*	-	1	2	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	5	73
Zoonoses																				
Anthrax*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Leptospirosis*	-	-	-	-	-	-	1	-	-	-	-	1	-	-	-	-	-	-	2	29
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis*	-	-	-	-	-	-	2	-	-	-	1	1	-	-	-	-	-	-	4	23
Q fever*	1	-	-	-	1	-	2	1	-	2	-	5	2	-	-	-	1	-	15	186
Respiratory and other																				
Blood lead level*	-	5	-	1	2	1	9	1	1	-	-	3	1	-	4	-	-	-	28	265
Influenza*	-	2	-	-	-	-	1	1	3	-	-	-	-	-	-	-	-	-	9	108
Invasive pneumococcal infection*	10	11	13	4	12	10	12	2	3	1	1	-	-	2	-	2	2	-	85	264
Legionella longbeachae infection*	-	-	1	1	-	2	-	-	1	-	-	-	-	-	-	-	-	-	5	23
Legionella pneumophila infection*	1	-	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4	16
Legionnaires- disease (Other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Leprosy	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
Meningococcal infection (invasive)*	-	2	1	-	4	-	2	-	1	2	1	1	1	-	-	-	-	-	16	74
Tuberculosis	8	2	5	1	2	-	-	1	2	-	2	-	-	1	-	-	2	-	26	177
Vaccine-preventable																				
Adverse event after immunisation	-	-	-	1	-	2	2	-	-	1	-	-	-	4	-	1	1	1	13	97
H. *Influenzae b* infection (invasive)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4
Measles	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	4
Mumps*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	20
Pertussis	12	30	26	11	14	5	17	7	37	8	4	1	-	3	-	1	2	-	178	848
Rubella*	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	18
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Enteric																				
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cryptosporidiosis*	2	2	1	-	-	-	1	2	3	-	-	-	-	-	-	-	-	-	11	113
Giardiasis*	7	16	14	6	6	3	8	4	17	-	4	-	7	3	-	-	1	-	96	510
Haemolytic uraemic syndrome	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3
Hepatitis A*	1	-	1	1	-	-	-	-	2	-	-	-	-	1	-	-	-	-	6	56
Hepatitis E*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Listeriosis*	-	-	-	-	1	1	-	1	-	-	-	-	1	-	-	-	-	-	4	16
Salmonellosis (not otherwise specified)*	10	15	11	2	33	1	7	4	12	11	6	4	4	1	4	1	4	-	132	1,239
Shigellosis*	-	-	-	-	1	-	-	-	3	-	-	-	-	-	-	-	-	-	4	35
Typhoid and paratyphoid*	1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	17
Verotoxin producing *E. coli**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

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

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

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.