Environmental Health

The Journal of the Australian Institute of Environmental Health

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The Journal of the Australian Institute of Environmental Health

ABN 58 000 031 998

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ISSN 1444-5212

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The Australian Institute of Environmental Health gratefully acknowledges the financial assistance and support provided by the Commonwealth Department of Health and Aged Care in relation to the publication of *Environmental Health*. However, the opinions expressed in this Journal are those of the authors and do not necessarily represent the views of the Commonwealth.

Published by *Environmental Health*, The Journal of the Australian Institute of Environmental Health.

Correspondence to: Associate Professor Heather Gardner, Editor, P O Box 68 Kangaroo Ground, Victoria, 3097, Australia.

Cover Design by: Motiv Design, Stepney, South Australia

Typeset by: Mac-Nificent, Northcote, Victoria

Printed by: MatGraphics & Marketing, Notting Hill, Victoria

The Journal is printed on recycled paper.

ISSN 1444-5212

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Editorial

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We are proud to feature in this issue of *Environmental Health*, a paper that reviews the environmental and health effects of termiticides under Australian conditions. This comprehensive paper is destined to become a classic in the literature. It provides the basis for a large and continued research program. Indeed, this issue contains much valuable and topical information on risks to human health from ciguatera fish poisoning, through metal contamination of complementary medications to exposure to inorganic arsenic in drinking water, from Ross River virus infection to a possible invasion by the tropical bed bug, *Cimex hemipterus*, from the importance of Environment Improvement Plans in reducing significant site pollution to a new approach to food safety assessment to improve health.

National Conference in Hobart

The Conference lived up to expectations and some papers perhaps exceeded these, for example Lim Chin Moo's presentation on the Singapore experience of SARS provided important and useful information about dealing with an emergency but it was handled with such a light touch that it unexpectedly provided some humour. A successful Emergency Management Forum was held on the final day with Graham Burgess, Roscoe Taylor and Tim Strickland. Papers ranged from the macro to the micro, from the international to the national, to the local, from Martin Riddle on the Antarctic, to Andrew Langley on rural and remote water supplies and Australian Drinking Water Guidelines, to two local Tasmanian case studies, one on a waste water remediation program at Boat Harbour by Doug Doherty and another on the Tamar Estuary by Bill Wood. Derek Lightbody gave an inspired presentation on Copper Chrome Arsenate Treated Timber, and a particularly good session was on the issue of the competing needs of agricultural sustainability and safe food by Tom Ross, followed later in the day by Michaela Hobby on the AIEH's fresh approach to Food Safety Assessment (see also article by Hoyne and Isbester on this topic in this issue). Merle O'Donnell gave an inspiring paper on Indigenous Environmental Health, preceded by Jan Schmitzer and Zane Hughes' paper on developments in the education of Indigenous Environmental Health professionals.

Heather Gardner
Editor

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Call for Papers

The Journal is seeking papers for publication.

Environmental Health is a quarterly, international, peer-reviewed journal designed to publish articles on a range of issues influencing environmental health. The Journal aims to provide a link between the science and practice of environmental health, with a particular emphasis on Australia and the Asia-Pacific Region.

The Journal publishes articles on research and theory, policy reports and analyses, case studies of professional practice initiatives, changes in legislation and regulations and their implications, global influences in environmental health, and book reviews. Special Issues of Conference Proceedings or on themes of particular interest, and review articles will also be published.

The Journal recognises the diversity of issues addressed in the environmental health field, and seeks to provide a forum for scientists and practitioners from a range of disciplines. Environmental Health covers the interaction between the natural, built and social environment and human health, including ecosystem health and sustainable development, the identification, assessment and control of occupational hazards, communicable disease control and prevention, and the general risk assessment and management of environmental health hazards.

Aims

- To provide a link between the science and practice of environmental health, with a particular emphasis on Australia and the Asia-Pacific Region
- To promote the standing and visibility of environmental health
- To provide a forum for discussion and information exchange
- To support and inform critical discussion on environmental health in relation to Australia's diverse society
- To support and inform critical discussion on environmental health in relation to Australia's Aboriginal and Torres Strait Islander communities
- To promote quality improvement and best practice in all areas of environmental health
- To facilitate the continuing professional development of environmental health practitioners
- To encourage contributions from students

Papers can be published under any of the following content areas:

Guest Editorials

Guest Editorials address topics of current interest. These may include Reports on current research, policy or practice issues, or on Symposia or Conferences. Editorials should be approximately 700 words in length.

Research and Theory

Articles under Research and Theory should be 3000-5000 words in length and can include either quantitative or qualitative research and theoretical articles. Up to six key words should be included. Name/s and affiliation/s of author/s to be included at start of paper and contact details including email address at the end.

Practice, Policy and Law

Articles and reports should be approximately 3000 words in length and can include articles and reports on successful practice interventions, discussion of practice initiatives and applications, and case studies; changes in policy, analyses, and implications; changes in laws and regulations and their implications, and global influences in environmental health. Up to six key words should be included. Name/s and affiliation/s of author/s should be included at start of paper and contact details including email address at the end.

Reports and Reviews

Short reports of topical interest should be approximately 1500 words. Book reviews should be approximately 700 words and Review Articles should not exceed 3000 words in length.

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Ciguatera Fish Poisoning in Queensland: Epidemiological Features and a Case Definition for Routine Disease Surveillance

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Ciguatera Fish Poisoning (CFP) is the most widely reported form of foodborne illness involving finfish worldwide and it is endemic in the India-Pacific and Caribbean regions. CFP is intoxication caused by a range of different ciguatoxins that originate in the dinoflagellate, Gambierdiscus toxicus. *These toxins bioaccumulate up the food chain in predatory fish prior to human consumption. Diagnosis and under-reporting of CFP is complicated by the absence of a confirmatory diagnostic laboratory test. With a view to determining the indicative epidemiological features of CFP in Queensland, reported cases in Queensland over the 3.5-year period up to July 2001 were analysed. Of the 138 cases, almost 50% reported eating either mackerel or coral trout fish shortly before their illness. 94.5% of cases reported the onset of symptoms within 24 hours of consumption of the suspected fish and all but one case developed symptoms within 48 hours of consumption of the suspected fish. While many different symptoms were reported, 90.6% of cases reported peripheral and/or circumoral numbness and tingling or reversed temperature sensations. The results of this analysis have been used to develop a case definition that has been adopted by Queensland Health for future disease surveillance of CFP in Queensland.*

Key words: *Ciguatera Fish Poisoning; Tropical Finfish; Foodborne Illness; Public Health Surveillance*

Ciguatera Fish Poisoning (CFP) is intoxication caused by ciguatoxins that can accumulate in many different finfish found in tropical and subtropical regions of the world (Pearn 2001). Associated with both acute and chronic health effects, it is the most reported form of foodborne illness involving finfish worldwide (Lehane & Lewis 2000). CFP is endemic in the India-Pacific and Caribbean regions (Butera et al. 2000; Schnorf, Taurarii & Cundy 2002) and a number of different ciguatoxins have been characterised with variations across the endemic regions of the world (Lehane & Lewis 2000). In all cases, the toxin is bioaccumulated in larger predatory fish up the food chain from the origin of the toxin in the dinoflagellate, *Gambierdiscus toxicus* (Pearn 2001).

CFP is a notifiable disease in Queensland under the *Health Act 1937*. However, as many cases of CFP are mild, they may not seek medical attention and thus not be diagnosed or reported (Butera et al. 2000). The mis-diagnosis and under-reporting of cases is thought to be significant (Lehane & Lewis 2000; Miller, Pavia & Keary 1999), which affects the identification, investigation and control of outbreaks. Only an estimated 10-20% of cases in Australia are reported (Lehane & Lewis 2000). Further, the diagnosis and subsequent notification of CFP is complicated by the fact that CFP is a clinical diagnosis only (Gillespie et al. 1986) as a confirmatory

laboratory test is not available. Thus, there is the potential for variation in the clinical features that might be used by different medical practitioners to make the diagnosis of CFP in any particular case.

Case definitions have been developed for use in the investigation of specific outbreaks of CFP (Ng & Gregory 2000). However, there has not been a standardised case definition for the notification of CFP for routine surveillance purposes in Queensland to date. Similarly, there is not a standardised case definition in other parts of Australia for routine disease surveillance purposes. A literature search did not reveal a case definition in use in other countries that could be adopted or adapted for routine disease surveillance purposes in Queensland. With a view to enhancing the surveillance of CFP in Queensland, a project was undertaken by Queensland Health to develop a case definition that was consistent with the apparent epidemiological features of CFP in Queensland. This project was based on an analysis of all CFP cases that were reported to the Environmental Health Unit of Queensland Health between January 1998 and July 2001 inclusive.

Methods

Initially, 149 case reports were available for this analysis. This number of cases, over a continuous period of 3.5 years, was considered to be sufficient to determine the indicative epidemiological features of suspected CFP in Queensland. The case reports originated from the various Public Health Units in Queensland to which doctors had notified cases or to which cases had made direct reports, or from cases that had otherwise come to notice during the investigation of outbreaks of suspected CFP.

Key data from the case reports were entered into an Epi-Info database (Epi-Info Version 6.04d, January 2001). The data recorded included type of fish reportedly eaten, incubation period, first symptom noted, all reported symptoms and worst reported symptom.

On preliminary analysis of the case reports, it became apparent that not all cases had consumed a warm water ocean finfish (that is, a fish harvested in tropical or subtropical ocean waters). Specifically:

- In 130 (87.2%) cases, the person reported eating a type of fish that definitely could be associated with CFP (that is, a warm water ocean finfish).
- In 8 (5.4%) cases, the person was not able to identify specifically what fish had been eaten, but the consumption of a warm water ocean finfish could not be reasonably excluded.
- In the remaining 11 (7.4%) cases, the person reported eating a type of fish that could not be expected to be associated with CFP (that is, not a warm water ocean finfish).

Consequently, the 11 cases that reported eating a type of fish that could not be associated with CFP were excluded from any further analysis. This paper deals with the descriptive analysis of the remaining 138 cases that ate (130 cases) or probably ate (8 cases) a type of fish that could be expected to carry ciguatoxin.

Results

Table 1 details the types of fish that the cases reported consuming prior to the onset of their illness. As it was not possible to verify the type of fish eaten by each case, comment on the limitations of current fish nomenclature is necessary. The unequivocal identification of the type of fish that is implicated in individual cases of CFP will always be very difficult because specific fish names are not used consistently in the seafood industry or by recreational fishers. For example, the *Australian Seafood Handbook* (Last, Ward & Yearsley 1999) and *Grant's Guide to Fishe*s (Grant 1999) both list at least three different "coral trouts" and

Table 1: Reported type of fish consumed

Fish type	Cases (*n*)	Cases (%)
Mackerel	36	26.1
Coral Trout	31	22.5
Red Emperor	8	5.8
Leatherskin	6	4.3
Maori Cod	6	4.3
Reef Cod	6	4.3
Samson Fish	5	3.6
Queenfish	4	2.9
Barra Cod	3	2.2
Gold Spot Cod	3	2.2
Mixed Reef	3	2.2
Red Bass	3	2.2
Snapper	3	2.2
Sweet Lip	3	2.2
Rock Cod	2	1.4
Spotted Cod	2	1.4
Banded Trumpeter	1	0.7
Black Kingfish	1	0.7
Bream	1	0.7
Red Jew	1	0.7
Reef Fish	1	0.7
Trevally	1	0.7
Unknown warm water ocean finfish	8	5.8
TOTAL	138	

four different "mackerels". Thus, the use of general terms such as "coral trout" and "mackerel" does not reveal the actual species of fish implicated in a case of suspected CFP. Similarly, general labels such as "mixed reef", "reef cod" and "reef fish" are commonly used in the industry, but they typically comprise a range of different fish species mixed together. Thus, the use of such general terms makes it essentially impossible to identify the actual type of fish that was eaten and which presumably carried the ciguatoxin.

The incubation period for each case was calculated from the time of consumption of the suspect fish to the time of onset of the first symptom/s. Information was available for 127 (92%) cases (Table 2). The median incubation period was 7 hours, mean 11.2 hours, and range <1 ->100 hours. Only 1 (0.8%) case reported an incubation period in excess of 48 hours.

Table 2: Reported incubation periods

Incubation period	% (*n* = 127)
<12 hours	87 (68.5%)
<24 hours	120 (94.5%)
<48 hours	126 (99.2%)
≥48 hours	1 (0.8%)

Information on the first symptom reported was available for 121 (87.7%) cases (Table 3). Table 4 outlines the frequency of all the various symptoms reported by the 138 cases at any time in their illness up to the time of their interview by the Public Health Unit.

Table 3: First symptom reported by cases

Symptom	% (*n* = 121)
Diarrhoea	18.2
Nausea	13.2
Paraesthesia (numbness/tingling)	11.6
Muscle pain	9.9
Abdominal pain	8.3
Itch	8.3
Headache	8.3
Joint pain	5.0
Reversed temperature sensations	5.0
Pain (not specified)	2.5
Vomiting	2.5
Generalised weakness	1.7
Dizziness	1.7
Joint and muscle pain	0.8
Numbness/tingling and muscle pain	0.8
Chills	0.8
Flushing	0.8
Headache and nausea	0.8

Peripheral and/or circumoral numbness and tingling and reversed temperature sensations generally are recognised as the most typical symptoms of CFP (Butera et al. 2000). Indeed, these neurological symptoms are regarded widely as pathognomonic for CFP (Pearn 2001). In this series, 125 (90.6%) cases experienced one or more of these symptoms.

Table 4: Frequency of individual symptoms reported by cases

Symptom	% (*n* = 138)
Any combination of numbness/tingling of the hands, feet or around mouth	85.5
Generalised weakness	77.5
Numbness/tingling of hands	73.2
Reversed temperature sensations	71.7
Itch	70.3
Muscle pain	67.4
Headache	65.9
Numbness/tingling around mouth	65.2
Numbness/tingling of feet	61.6
Joint pain	60.9
Diarrhoea	60.9
Chills	50.0
Nausea	49.3
Dizziness	38.4
Abdominal pain	36.2
Sweating	36.2
Difficulty walking	33.3
Eye irritation	26.8
Tremor	24.6
Shortness of breath	23.9
Skin rash	23.9
Vomiting	23.2
Neck pain	22.5
Dental pain	19.6
Difficulty moving arms or legs	18.8
Pain on urination	13.8
Increased salivation	6.5

For the purposes of developing a robust case definition for the routine surveillance of CFP in Queensland and one that could be adopted Australia wide, various combinations or clusters of symptoms could be considered. However, given the typical association between CFP and peripheral and/or circumoral numbness and tingling and reversed temperature sensations, any such symptom clusters should include those neurological symptoms as a minimum. Table 5 shows the effect on the percentage of cases in this series that might be regarded as probable CFP, once a definition requiring numbness/tingling and/or reversed temperature sensation, and any one or more additional symptom/s is used. As would be expected, the percentage of cases that would satisfy the criteria for any particular symptom cluster decreases as the complexity of the symptom cluster increases.

Details of the worst symptom reported by the cases during their illness are outlined in Table 6. In a small number of cases, more than one symptom was reported as the worst symptom. For the purposes of Table 6, the first mentioned worst symptom reported by the case has been recorded as the worst symptom.

Table 5: Prevalence of various symptom clusters

Symptom cluster	Symptoms	Prevalence
Neurological only	Numbness/tingling and/or reversed temperature sensation	90.6%
Neurological and at least one other symptom	Numbness/tingling and/or reversed temperature sensation, plus at least one (1) of the following symptoms - nausea, vomiting, abdominal pain, diarrhoea, joint pain, muscle pain, itch and rash	89.9%
Neurological and gastrointestinal	Numbness/tingling and/or reversed temperature sensation, plus at least one (1) of the following gastrointestinal symptoms - nausea, vomiting, abdominal pain or diarrhoea	76.8%
Neurological and musculoskeletal	Numbness/tingling and/or reversed temperature sensation, plus at least one (1) of the following musculoskeletal symptoms - joint pain or muscle pain	75.4%
Neurological and skin	Numbness/tingling and/or reversed temperature sensation, plus at least one (1) of the following skin symptoms - itch or rash	68.1%
Neurological, gastrointestinal, musculoskeletal and skin	Numbness/tingling and/or reversed temperature sensation, plus one (1) or more symptom/s from each of the gastrointestinal, musculoskeletal and skin clusters	50.0%

Table 6: Worst symptom experienced during illness by cases

Symptom	% (*n* = 123)
Muscle pain	18.7
Itch	13.8
Numbness/tingling	13.0
Reversed temperature sensations	12.2
Generalised weakness	8.1
Joint pain	8.1
Headache	8.1
Diarrhoea	4.9
Abdominal pain	4.9
Any other symptom	8.1

Discussion

There are two particular limitations in this analysis that mean it can provide only indicative information on the epidemiology of CFP in Queensland. First, the absence of a case definition a priori for CFP is a significant limitation as different notifying doctors and even different Public Health Units probably used different criteria for the reporting and acceptance of a suspected case. This analysis is dependent on cases that passed any such reporting and acceptance thresholds. Second, the Environmental Health Unit of Queensland Health did not necessarily hold all case reports of suspected CFP that had been reported to the Public Health Units over the time period subject to this analysis. However, as it was the required practice within Queensland Health for completed questionnaires from suspected cases to be forwarded by the Public Health Units to the Environmental Health Unit, it is likely that there were few, if any, other case reports in the study period. Thus, it is considered that this factor has had little effect on the overall findings of this study. Despite both of these limitations, the information used in this analysis still remains the best available, at present, for the reporting of the key indicative features of suspected CFP in Queensland.

A diverse range of fish types was reportedly eaten among the cases prior to the onset of the clinical illness that was notified as CFP. However, two particular fish types, "mackerel" and "coral trout", accounted for almost half the cases at 48.6%. It is also possible that either or both of these fish types may have been included in the general categories of "mixed reef" and "reef fish" that accounted for a further 4 (2.9%) cases or the unknown warm water ocean finfish category that accounted for a further 8 (5.8%) cases. Assuming that the description of the type of fish reportedly eaten is generally accurate, Table 1 shows that there are many different fish that can be associated with CFP in Queensland. This is, of course, not an unexpected finding.

While fish types such as "mackerel" and "coral trout" might appear to pose a considerably higher risk, more detailed exposure information would be needed to more accurately calculate relative risks of developing CFP from specific fish types. Such information would include:

- the quantities or numbers of each fish consumed each year
- specific fish species consumed
- size and age of the fish eaten
- area where an implicated fish was caught
- the amount of fish eaten by the case immediately prior to onset of the illness
- which part of the fish was eaten, for example, flesh, viscera or head.

It is possible, for example, that "mackerel" and "coral trout" account for the greatest number of cases simply because far more of those fish types are eaten. Thus, if consumers were to change their consumption to other tropical or subtropical warm water ocean fish, their risk of contracting CFP may or may not alter. For the future risk management of CFP in Queensland, it is apparent that more information on cases

from an exposure perspective needs to be collected and analysed. As an outcome of this project, Queensland Health intends to collect and periodically report some additional exposure information from reported cases to Safe Food Production Queensland, which has regulatory oversight of the catching and processing sectors of the seafood industry in Queensland.

The incubation period data in this analysis are generally consistent with various publications cited in one review (Lehane 1999). Specifically,

> *Gillespie et al. (1986) gave the usual time of onset of symptoms as 1-6 hours, with onset in 90% of cases being within 12 h; and Lewis et al. (1988) reported 1-70 h, with a mean of 6.4 h. Glaziou and Legrand (1994) reported that symptoms of CFP appear 2-30 h after ingestion of toxic fish.*

It is apparent from this analysis and supported by other published literature that CFP typically commences within 1-24 hours of consumption of a ciguatoxic fish and that it is most unlikely for the onset of symptoms to be delayed beyond 48 hours. For the purposes of a case definition for routine surveillance of CFP in Queensland, this analysis supports the view that symptoms must commence within a maximum of 48 hours of consumption of a potentially ciguatoxic fish. A strong case could be mounted to limit the time period to 24 hours. However, with a view to not discounting potential cases that report incubation periods of 24-48 hours, the conservative approach for disease surveillance purposes is to adopt a maximum incubation period of 48 hours.

A diverse range of symptoms involving the neurological, gastrointestinal, musculoskeletal, skin, cardiac and other systems has been reported for CFP worldwide (Butera et al. 2000; Gillespie et al. 1986, Karalis et al. 2000; Miller, Pavia & Keary 1999; Pearn 2001). The published literature on CFP frequently refers to gastrointestinal symptoms being the first symptoms experienced (Lehane & Lewis 2000). However, the first symptom reported in this case series was:

- gastrointestinal (diarrhoea, nausea, abdominal pain or vomiting) in 43% of cases
- neurological (paraesthesia, reversed temperature sensation, itch, headache or dizziness) in 36% of cases
- musculoskeletal (muscle pain or joint pain) in 16% of cases.

These results suggest that the initial clinical picture in Queensland is not necessarily the same as is reported for CFP in other parts of the world. This could be explained by variations in the toxicology of the different ciguatoxins throughout the world, an artefact of the method of questioning of reported cases, or a toxin other than ciguatoxin might have accounted for some cases. From the perspective of developing a case definition for routine surveillance of CFP in Queensland, it is considered that these results do not provide an overwhelming pattern that supports the inclusion of the nature of the initial symptom as a key criterion in the case definition.

Another limiting factor for this analysis relates to the quality and consistency of the symptom data acquired at interview when the case was notified or otherwise reported to a Public Health Unit of Queensland Health. The reporting or non-reporting of a particular symptom might have been influenced by the way in which suspected cases were interviewed. For example, the asking of directly leading questions in regard to any particular symptom could have lead to quite different responses than if leading questions were avoided. The authors have no information on the specific nature and quality of the interviews of suspected cases and, for this reason, the data on reported symptoms must be regarded as indicative only. In order to control this source of

potential error, it has been identified that consistency of interviewing and, in particular, the avoidance of leading questions is necessary for the future surveillance of CFP in Queensland.

From Table 4, the following symptoms were reported at some time of their illness by over 70% of cases:

1. Any combination of numbness/ tingling of the hands, feet or around mouth
2. Generalised weakness
3. Numbness/tingling of hands
4. Reversed temperature sensations
5. Itch

The first, third and fourth of these symptoms are regarded as pathognomonic symptoms of CFP (Pearn 2001) and no further comment is indicated. While generalised weakness was very commonly reported (77.5% of cases), it is also a relatively nondescript and non-specific symptom that potentially is open to inconsistent interpretation by both the sufferer and the interviewer. Thus, it is not considered to be a symptom that is readily amenable for inclusion in a case definition as a primary key criterion.

In regard to skin itch, there are many possible causes of this symptom, including fish allergy and other fish-related toxins apart from ciguatoxin. In this case series, 93 (74.4%) of the cases that reported peripheral and/or circumoral numbness/tingling or reversed temperature sensations, also reported skin itch. On the other hand, four (4) cases (2.9%) reported itch as the only symptom they experienced. While each of these 4 cases had eaten a type of fish that could potentially carry ciguatoxin, the presence of skin itch alone is considered to be an inadequate clinical presentation to regard such cases as CFP with any reasonable confidence. Overall, skin itch is not considered to be a sufficiently sensitive symptom to be used as a stand-alone criterion for the routine surveillance of CFP in Queensland.

The primary purposes of routine disease surveillance are to observe trends in the pattern of the specific disease over time, to identify individual cases of certain diseases that demand immediate public health action to minimise the risk of further cases and to identify outbreaks rapidly such that effective short-term and long-term remedial action can be taken. The veracity of the surveillance data is related, in part, to the use of a suitably robust case definition. The key issues in regard to a case definition for any illness are ensuring adequate sensitivity and specificity. However, in the case of CFP, this is made essentially impossible by the absence of a confirmatory clinical laboratory test.

In regard to specific individuals diagnosed or suspected by their doctor of having CFP, their clinical management should not be dependent on the case definition used for routine surveillance purposes. Also, any particular personal follow-up action affected people may wish to take, for example litigation against the supplier of the putative food, is fundamentally distinct from the disease surveillance activities of a health department. Thus, in the absence of a gold standard against which the clinical diagnosis of CFP can be confirmed, the case definition for routine surveillance purposes can be defined only as that which is thought to be most suitable for the purposes of observing trends and identifying individual cases and outbreaks. It will be sufficient if it is likely, generally, to include those cases that could reasonably be thought to be probable CFP, while at the same time excluding those cases that could reasonably be thought to not be CFP. Provided the case definition is applied consistently and rigorously over time, the outcome data on reported cases that meet the case definition will show trends in disease incidence and outbreaks, which is the underlying intent of routine disease surveillance. To that end, it can be seen

from Table 5 that as the complexity of symptom clusters increases, the number of cases that would meet the clinical criteria for a case of CFP decreases. As an example:

- 90.6% of the cases in this series reported peripheral and/or circumoral numbness/tingling, and/or reversed temperature sensation

- 89.9% of the cases had at least one (1) of these neurological symptoms *plus* at least one (1) gastrointestinal, musculoskeletal or skin symptom

- 50% of the cases had at least one (1) of these neurological symptoms *plus* at least one (1) gastrointestinal, musculoskeletal *and* skin symptom

By using the classic neurological symptoms of numbness/tingling and/or reversed temperature sensation alone as the clinical criteria for CFP, only 13 (9.4%) cases in this series would not have been included. Using the symptom cluster requiring these classical symptoms plus just one (1) additional symptom would have reduced the number of cases in this series by only 1 (0.7%). On this basis, and especially from a disease surveillance perspective, it is considered unnecessarily restrictive to also require at least one other symptom from at least one other relevant bodily system, let alone from more than one other bodily system.

Being an intoxication, the severity of symptoms in CFP can be dependent on a number of factors, in particular the amount of ciguatoxin that is ingested and variations in individual susceptibility (Lehane & Lewis 2000). Among the people who eat part of the same toxic fish, there may be some who develop very obvious symptoms that clearly satisfy the clinical criteria for the proposed case definition, whereas there may be others who might experience vague or mild symptoms only. For the purposes of disease surveillance in Queensland, it became apparent during this analysis of the need for a secondary case definition relevant to those people who seemed to be likely to be suffering CFP on epidemiological rather than clinical grounds. In particular, this would relate to a person who ate part of the same fish as another person who clearly had clinical CFP, as defined above, but who experienced just gastrointestinal, musculoskeletal or skin symptoms in the absence of any numbness/tingling of the hands, feet or mouth, or reversed temperature sensations. It is considered appropriate to consider such a case as an epidemiologically linked case if it can be shown, or reasonably be expected, that they ate part of the same warm water ocean finfish as a clinical CFP case; their symptoms commenced within the proposed incubation period of 48 hours; and they experienced any diarrhoea, nausea, vomiting, abdominal pain, muscle pain, joint pain or skin rash.

Following the analysis of this case series, the authors consulted with all the Public Health Units in Queensland Health in regard to establishing a case definition for the routine surveillance of CFP in Queensland. Clinical and epidemiological case definitions were agreed and have been implemented for routine use on a statewide basis (Appendix A). Further, a standardised case report form has been approved and it includes the collection of data relevant to the case's likely source of ingested ciguatoxin. It is anticipated that this information will be helpful in the risk management approach by the seafood industry to the minimisation of cases of CFP in its Queensland and other markets.

Conclusion

CFP is a significant public health problem in many parts of the world as it has serious clinical and economic consequences. It rightfully deserves attention from public health authorities, but this paper highlights the difficulties that arise in routine disease surveillance activities when there is not a confirmatory test available for the disease in question. However, the responsibility of public health authorities is to implement procedures that provide the greatest opportunity for effective intervention, even

in the absence of a gold standard diagnosis. The outcomes of this project have led to significant enhancements to the routine surveillance of CFP in Queensland and it is anticipated that these will assist in the reduction of the incidence of CFP in Queensland in the future. It is hoped to be able to report on the successful implementation of these enhancements after a few years of use.

Acknowledgments

The authors acknowledge the assistance of the Public Health Units of Queensland Health for the data used for this paper. Appreciation is extended to members of the Queensland Health Ciguatera Project Team, Chris Towner, Terry Moore, Rod Miles and Dave Gould, and John Burke from Safe Food Production Queensland.

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Legislation

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Appendix A

The case definitions implemented by Queensland Health for its routine CFP disease surveillance activities are:

Clinical case of CFP

1. Consumption of warm water ocean finfish not more than 48 hours before onset of first symptom/s, and

2. Reporting of at least one (1) of the following symptoms:

    a. Paraesthesia (numbness and/or tingling), or

    b. Reversed temperature sensation

Epidemiologically linked case of CFP

1. Confirmed, or reasonably suspected, consumption of part of the same fish as a clinical case of CFP (as defined), and

2. Onset of one (1) or more of the following symptoms within 48 hours of consumption of the fish:

    a. Diarrhoea,

    b. Nausea,

    c. Vomiting,

    d. Abdominal pain,

    e. Muscle pain,

    f. Joint pain, or

    g. Skin rash

A Pilot Survey of the Presence of Undeclared Drugs and Health Risk Associated with Metal Contamination of Complementary Medications Offered for Sale in Queensland

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Reports in the literature indicate that elevated levels of metals in Traditional Chinese Medicines (TCMs) have been associated with poisoning of children and adults. Some TCM products available for sale in retail outlets in Queensland are not registered or listed on the Australian Register of Therapeutic Goods (ARTG) as required under the Therapeutic Goods Act 1989 *(Cmwlth). These products make therapeutic claims and, consequently, there is concern regarding their quality and safety. Queensland Health conducted a pilot study which investigated labelling, metal content and presence of scheduled substances in a sample of 40 complementary medicine products obtained from retail outlets in south east Queensland. In the absence of Australian guideline levels for metals in this type of product, results from this survey were compared to maximum levels for metals established by the health authority in Singapore. In addition, the risk to health that oral preparations of these products present was assessed. Non-compliance with Queensland legislation for labelling issues and therapeutic claims was found for a large number of the products of Asian origin. Nineteen of the 28 samples of Asian origin and six of the 12 samples of non-Asian origin contained detectable levels of at least one metal. Two of the products of Asian origin contained quantities of arsenic, antimony or lead, which exceeded Singapore guideline levels. Five of the Asian products contained undeclared scheduled substances. Further research is being undertaken better to characterise the risk that these products present to human health.*

Key words: *Health Risks; Labelling; Metals; Alternative Medicines; Chinese Medicines*

In Australia Traditional Chinese Medicines (TCM) are classified as therapeutic goods and are subject to State and Commonwealth Legislation. T*he Commonwealth Therapeutic Goods Act 1989* (Cmwlth) (CTGA) was designed to provide a national system of controls over the quality, safety, efficacy and timely availability of imported or locally manufactured therapeutic goods used in Australia and/or exported from Australia (Bensoussan & Myers 1996). In Queensland, the *Health Regulation 1996* and the *Health (Drugs and Poisons) Regulation 1996*, regulate the sale, distribution and labelling of scheduled[1] and non-scheduled therapeutic products.

A substance that is defined as a therapeutic good by the CTGA cannot be imported into, exported from, manufactured or supplied for use in humans in Australia unless it is registered or listed on the Australian Register of Therapeutic Goods (ARTG). Exceptions to this rule include where a substance is specifically exempted under the Act, or the substance is brought into the country for the personal treatment

of a specific person. Anecdotal evidence suggests that some of the medicines brought into Australia for personal use are subsequently sold in retail outlets. As all classes of therapeutic goods (either listed or registered) must comply with the Code of Good Manufacturing Practice (CGMP) and the composition of the goods also needs to be stated, there is some assurance as to the quality of therapeutic goods manufactured or distributed in accordance with CGMP. However, only limited post market surveillance is undertaken to determine whether products comply with the stated composition.

A number of studies indicate that certain complementary medicines, in particular Traditional Chinese Medicines (TCMs) and Proprietary Chinese Medicines (PCMs), may contain high levels of metals such as arsenic, mercury and lead and/or undeclared substances (Bateman, Chapman & Simpson 1998; Fratkin 1998; Huang, Wen & Hsiao 1997; Koh & Woo 2000). A recent systematic review of the literature examined 22 journal papers describing case reports, case series and epidemiological investigations where heavy metal poisoning by TCMs had occurred (Ernst & Coon 2001). The review found that poisonings were frequently related to elevated levels of lead and arsenic in medications and less frequently to elevated levels of mercury and cadmium. For adults and children (including infants) duration of TCM intake ranged from five days to 20-30 years. An extensive review of PCMs in Singapore found that of 2080 medicines screened between 1990 and 1997, 42 different medicines contained metals at concentrations above legal limits and 32 medicines contained 19 different undeclared drugs (Koh & Woo 2000).

In order to determine the presence of metals[2] and undeclared drugs in off-the-shelf medicines, which are offered for sale in Queensland, Queensland Health undertook a pilot survey. A primary focus of the survey was to examine products imported from Asia, as there was evidence to indicate that products manufactured in the Asian region were frequently contaminated and/or adulterated (Bensoussan & Myers 1996). Forty different products were collected, replicate samples were not taken, and sampling was not intended to identify inter- or intra-batch variability.

Methodology

Sample collection

A total of 40 samples were collected by Environmental Health Officers during September to November 1999 from three Asian style supermarkets, two herbalists and six health food shops. While a range of "off the shelf" complementary medicines were sampled, those manufactured in Asia were examined in detail.

Sampled products consisted of solids, powders, liquids, oils and cream preparations. Each sample contained at least 10g or 10mL of product. If a pack size was less than 10g or 10mL, additional packs of the same batch were also sampled. All samples remained in their original package for transport to the laboratory for analysis. Information supplied on packaging was used to determine the recommended dose.

Sample analysis

The Queensland Health Scientific Services (QHSS) laboratory performed metal analyses for antimony, arsenic, barium, chromium, lead, mercury and selenium. Samples for metal determinations were acid digested in accordance with QHSS method QSE-PPM-012. Levels in duplicate digests of the samples were determined using Inductively Coupled Plasma - Atomic Emission Spectroscopy (ICP-AES). Cadmium content was determined by Atomic Absorption Spectrophotometry (AAS). Methods used had detection limits of 3 mg/kg for arsenic and selenium, 2 mg/kg for antimony and lead, and 1 mg/kg for barium, chromium and mercury. All results reported are on a "total" basis. No speciation of metals was undertaken.

Methanolic extracts of the sampled medicines were examined for semi-volatile compounds using Gas Chromatography/Mass Spectrometry (GC/MS). Compounds tentatively identified using GC/MS were confirmed by running a standard of the compound under the same conditions.

The scheduling and labelling of products was examined to assess compliance with the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP) and Queensland legislation (Health Regulation 1996) for non-scheduled compounds.

Assessing the health risks

There are currently no metal contamination standards for TCMs in Australia. The Singapore Ministry of Health has specified maximum levels for some metals in CPMs. These include 5ppm for arsenic, 150ppm for copper, 0.5ppm for mercury and 20ppm for lead (Koh & Woo 2000). Singapore does not permit antimony in CPMs. Although no information was available on how these levels were derived, the maximum levels specified by the Singapore Ministry of Health were used as a basis to establish whether the levels were acceptable. No limits have been set by the Singapore Ministry of Health for barium, chromium and selenium.

Where metals were detected in oral preparations, the risk that these preparations present to consumers was assessed. Based on the metal level in these preparations and the recommended daily dose (as stated on the label) the amount of metal ingested per day was compared with an exposure level that over a lifetime would not be expected to produce an adverse health effect. As consumers of these products would be unlikely to consume the product on a regular basis over a lifetime the estimates of ingested daily intake using this approach would overestimate exposure to contaminants in the products. However, as no information was available on the duration of treatment or prescribing practices this conservative approach was considered reasonable.

For antimony, inorganic arsenic and lead the Tolerable Daily Intakes (TDI[3]) (µg/kg bw/day) as established by Food Standards Australia and New Zealand (FSANZ) were used. TDIs for these metals were 0.4 µg/kg bw/day for antimony, 3 µg/kg bw/day for inorganic arsenic and 3.6 µg/kg bw/day for lead (ANZFA 2001). For barium and chromium, chronic oral reference doses (RfD[4]) established by the United States Environmental Protection Agency (USEPA) were used. RfDs for barium and chromium (the more toxicologically relevant species: CrVI) were 70 µg/kg/day and 3 µg/kg/day, respectively (USEPA 2002a,c). The RfD for the less toxic, CrIII was 1500 µg/kg/day (USEPA 2002b). As no information was available on the chromium form of the samples analysed, a worse case approach was assumed and exposure estimates were compared with the CrVI RfD.

In assessing the risk to health, average body weights of 10 kg for children and 60 kg for adults were used and it was assumed that consumers complied with the recommended dose as stated on the label. Risk assessments using this methodology were only undertaken on oral preparations.

Results

Labelling

For the 28 products that originated in Asia (from China, Indonesia, Hong Kong, Korea, Singapore, Taiwan), 17 were oral preparations. Of the 12 samples of non-Asian origin, 9 were manufactured in Australia and 5 were oral preparations. No products of Asian origin were labelled as being registered or listed.

Compliance of products with the SUSDP was assessed based on their composition as provided on product labels and as determined by analytical analysis. All products manufactured in Australia complied with the SUSDP and the relevant

Queensland legislation, while labelling for 75% (21 of 28) of the products manufactured in Asia did not comply with relevant labelling requirements. Table 1 indicates that 20 products did not meet scheduling requirements based on stated composition of the product on the label.

Table 1: Compounds identified in products for which labelling did not comply with requirements under the SUSDP (n=20).

Compound identified	Number of products	Type of product	Labelling requirements under the SUSDP
Guaiphenesin	1	Liquid	S2
Chlorpheniramine	1	Liquid	S2/S3 (depending on presence of other ingredients - not determined)
Lead (> 10mg/kg)	1	Tablet	S4
Arsenic (>1mg/kg)	3	Tablet	
Camphor oil >2.5% (label claim)	1	Liquid	S6
Cineole >25% (label claim)	1	Liquid	
Eucalyptus >25% (label claim)	1	Liquid	
Barium (>10mg/kg)	10	Tablet	
Chromium (>10mg/kg)	1	Tablet	

In addition to the lack of first aid and other health related labelling requirements of the SUSDP, other aspects of labelling also did not meet requirements under the SUSDP. Product labels were often difficult to read as print was too small, some included little English, and information on net weight, batch numbers, expiry dates and dose and frequency of dose was often not in English or not provided.

Therapeutic claims on labels indicated that the medications could be used for a range of purposes such as headaches, general weakness, insomnia and dizziness, tonifying kidneys, enriching the liver, rheumatic conditions, backache, acute and chronic sinusitis, impotence, spinal problems, fever, weight loss and general health and vitality. Therapeutic claims made for 12 of the 28 products manufactured in Asia did not comply with Queensland legislative requirements. Some of the labels could not be assessed as they were not in English.

Composition

Five of the 12 samples (42%) of non-Asian origin and 20 of the 28 (71%) samples of Asian origin had detectable levels of at least one of the following metals: arsenic, antimony, barium, chromium or lead. Levels of cadmium, mercury and selenium were below detection limits for all of the samples.

Table 2: Summary of detectable metal concentrations (mg/kg) in samples from Asian manufactured and non-Asian manufactured medicines (n=40).

	Antimony	Arsenic	Barium	Total Chromium	Lead
Asian Manufactured (*n*=28)					
Minimum concentration	6	4.6	4	1	2.4
Maximum concentration	16	300	65	54	33
No. of samples where metal was detected	2	3	20	10	5
Non-Asian Manufactured (*n*=12)					
Minimum concentration			1	3	
Maximum concentration		4	7	4	
No. of samples where metal was detected	0	0	4	2	1
Singapore guideline	0	5	-	-	20
No. of samples exceeding the Singapore guideline		2			1

Table 2 shows the minimum and maximum concentration for each metal detected in samples and the number of samples that contained each metal. Barium and chromium were the most frequent metals detected in samples. Antimony, arsenic and lead were also detected in 2, 3 and 5 samples from Asian products respectively. Three Asian products

contained 4 metals. Eight of the Asian products and 7 of the non-Asian products contained no detectable metal concentrations. Generally, metals were present at higher levels in Asian manufactured products than non-Asian manufactured products (only 3 of which were of non-Australian origin).

Of samples from this survey that had detectable levels of arsenic or lead, three exceeded the Singaporean guidelines. A large number and variety of therapeutic claims for these products were noted on packaging. The claims ranged from sore throat, ear ache and headache to rheumatic arthritis, sciatica, mumps, children's fever, anorexia and sinusitis. Two of these three samples also contained antimony that according to the Singapore Guidelines is not permitted.

Table 3: The range of estimated daily intakes (µg/kg body weight/day) for both children and adults for the metals detected in oral preparations of products manufactured in Asia.

	Children (10kg)		Adults (60kg)		Tolerable dose criteria (µg/kg bw/day)	
	Range	Exceedance (x/y)*	Range	Exceedance (x/y)*	TDI	RfD
Antimony	0-14.5	2/2	0-2.4	2/2	0.4	
Arsenic	0-271.6	2/3	0-45.3	1/3	3 (inorganic)	
Barium	0.7-28.4	0/17	0.1-4.7	0/17		70
Total Chromium	0-10.6	1/9	0-1.8	0/9		3 (Cr VI)
Lead	0-24.7	2/4	0-4.1	1/4	3.6	

*(*number of exceedances (x) of the tolerable dose criteria, out of the number of non-zero exposures (y))*

As only metal concentrations in Asian manufactured products exceeded the Singapore guidelines, the risk to human health of ingestion of these oral preparations was assessed. The metal intake per day (calculated for children and adults) for these products is shown in Table 3. Daily doses for children and adults were assumed the same as no differentiation was made on labels for any product. For the purposes of exposure assessment, analytical values reported below the detection limit were treated as zero.

For the 17 samples for which daily intakes were determined for an adult, two contained a metal at a level that if taken in accordance with instructions on the label would exceed the TDI for that metal. One sample had elevated concentrations of both antimony and arsenic, while another had elevated concentrations of both antimony and lead.

In addition to the two samples which may pose a health risk to adults, metal concentrations in a further three sample medications exceed tolerable dose criteria.

Discussion

Contamination of complementary medicines by metals can occur in a number of ways. The ingredients may be contaminated with metals occurring in the environment, metals may be added to the preparation for a specific therapeutic purpose or preparations may have become inadvertently contaminated during processing (Ernst & Coon 2001).

Survey results indicate that 37% of the medicine products sampled for this study contain concentrations of barium, arsenic, antimony, chromium and lead above the levels of detection for these metals. Antimony, arsenic, barium, chromium and lead were found in Asian manufactured products, while barium, chromium and lead were found in a smaller number of non-Asian manufactured products. Barium was found in 50% of products sampled. This might be expected given that it is commonly found naturally in foods such as seaweed, fish and certain plants (ATDSR 1992).

Two of the samples of Asian origin contained levels of arsenic and lead that exceed Singapore guidelines. Consumption of two of these products as per daily recommended doses would result in the exceedance of TDIs for antimony, arsenic and lead for adults and a further three products could pose a health risk if consumed by small children.

Other dietary sources of metals for adults were estimated to be 0.01-0.34 µg/kg bw/day for antimony, 0.76 -0.98µg/kg bw/day for total

arsenic and 0.42-0.73 µg/kg bw/day for lead (ANZFA 2001). The estimated average dietary consumption for barium is 16.6 (g/kg bw/day (ATSDR 1992) and for chromium is 0.36-3.2 µg/kg bw/day (ATSDR 2000). If other dietary sources of metals are considered in addition to the intake from specific TCMs, further exceedances of the arsenic TDI and the chromium RfD would be expected.

Results of this survey indicate that lifetime consumption of certain TCMs could pose a health risk to consumers. As little information is available on usage patterns of TCMs it is difficult to determine the likely duration of use. However, the therapeutic claims made by the most contaminated products included chronic conditions such as anorexia, rheumatic arthritis and sciatica and more general claims of "keeping youthfulness", "good to blood and health" and "enriching liver, kidney, marrow and sperm". Therefore, long term use would be expected.

Many of the products sampled in this survey were not listed or registered with the TGA. As these products are available for sale in Queensland and are not subject to any regulatory action by the TGA, there is potential for the health of consumers to be affected. The survey also indicated that labelling of a large number of the products sampled did not meet the requirements of Queensland legislation with regards to the adequacy of labelling and therapeutic claims made.

As none of the TCMs sampled in this study were registered or listed, it appears that these products are entering Australia outside the existing regulatory requirements. Although, the extent of this problem is unknown, the appearance of such products on shelves is an issue that clearly needs addressing. Queensland Health will be advising retailers of TCMs for which therapeutic claims are made that they are required to list or register their products with the ARTG. On listing or registering their product, the manufacturers are required to list the ingredients of the product.

To ensure retailers and suppliers comply with the regulatory requirement under the CTGA, surveillance of these products is required to ensure that the products comply with the regulatory requirements of the CTGA. This surveillance would best be undertaken by the TGA in collaboration with State and Territory health agencies.

It is acknowledged that the small sample size limits generalisability of the survey's finding in terms of the potential for these products to present a risk to human health. Availability of such products across Queensland and Australia is also unknown, as the sampling area was limited to the South-East Queensland region.

To understand better the extent to which these products present a risk to human health, research by Queensland Health in collaboration with the National Research Centre for Environmental Toxicology and Griffith University is being undertaken. This study will determine the availability of unregulated TCM products across the State, the proportion of products which do not comply with Queensland legislation regarding therapeutic claims and labelling requirements, concentrations of metals and other ingredients in these products and whether these may present a risk to human health.

Conclusion

The pilot survey has indicated that five unregulated Traditional Chinese Medicines available for sale in South-East Queensland in 1999 contained levels of arsenic, lead or antimony that could pose a health risk to consumers (adults or children). Additionally, 20 of the product labels contained scheduled substances and/or made therapeutic claims that breach current regulations and were not, or were only partially, labelled in the English language. None of the TCM products of Asian origin were listed or registered with the Australian Register of Therapeutic Goods. Access by the general public to such products for which therapeutic claims are made is a

concern. Further studies are required to determine better the extent of contamination, the variability in contamination within and across batches, and the availability of such products to the general public.

Endnotes

1. A substance is scheduled if it is included in the schedules of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP), compiled by the National Drugs and Poisons Schedule Committee (Commonwealth of Australia, 2003).
2. Metals will be defined in this paper as a group of elements, which includes non-ferrous metals and toxic metalloids.
3. A tolerable daily intake (TDI) refers to the amount of a chemical that can be ingested daily over a lifetime without appreciable risk
4. A reference dose (RfD) is an estimate of a daily exposure to an agent that is assumed to be without an adverse health impact on the human population.

Acknowledgments

The authors would like to acknowledge the input of Queensland Health colleagues: Dr Gerard Neville, Dr Andrew Langley, and Chris Healey for advice in health risk assessment and regulation and staff of Public Health Units for collecting samples.

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Health Regulation 1996 (QLD)
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A Review of the Environmental and Health Effects of Termiticides under Australian Conditions

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There exists a major cost issue as regards termite damage to wooden structures. A factor in this cost has been the increasing trend towards slab-on-ground construction. Current literature has been reviewed in relation to concerns about the possible public/environmental health consequences of the repeated use of termiticides in large quantities. The previous, current and projected future use patterns of termiticides are reviewed in the context of techniques appropriate for termite control and treatment priorities. The phasing out of organochlorine termiticides in Australia was undertaken to minimise impact of these substances on the environment and to a lesser extent on public health. These persistent chemicals were replaced by substances with high activity but relatively low persistence in the soil. There has also been an increase in the use of alternative methods (e.g. physical barriers) for the control of termites. The transition away from organochlorine termiticides has led to a realisation that significant information gaps exist with regard to replacement chemicals and other technologies. Although relatively persistent, the organochlorine chemicals have a limited lifespan in soils. Their concentrations are gradually attenuated by processes such as transport away from the point of application and biodegradation. Wooden structures originally treated with these substances will, with the passing of time, be at risk of termite infestation. The only available option is re-treatment with chemicals currently registered for termite control. Thus, there are likely to be substantial future increases associated with the cost of re-treatment and repairs of older slab-on-ground dwellings. More information is required on Australian termite biology, taxonomy and ecology. The risks of termite infestation need to be evaluated, both locally and nationally so that susceptible or high risk areas, structures and building types can be identified and preventive measures taken in terms of design and construction. Building regulations and designs need to be able to reduce or eliminate high-risk housing; and eliminate or reduce conditions that are attractive to termites and/or facilitate termite infestation.

Key words: *Termite; Termiticide; Organochlorine; Organophosphate; Pesticides*

There are substantial on-going costs associated with the prevention and treatment of termite infestation in wooden structures. Reapplication of chemical barriers is required every two to ten years depending on the termiticide products used

and local conditions. Contamination of potable water with solvents following treatment of existing buildings with termiticides, and reports of fish kills due to pesticides contaminating waterways as a result of runoff from treatment sites and spillages have occurred. There are concerns about the environmental and public health consequences of the application and reapplication of (potentially large quantities of) toxic chemicals over time. For example, in the US, there has been a partial ban on the use of chlorpyrifos since 2000, with most home and outdoor use of the chemical stopped, including pre and post-construction application as a termite barrier. The ban is a result of public and United States Environmental Protection Agency (USEPA) concerns about pesticide exposure in children following the detection of developmental effects in rats (USEPA 2000).

Termites consume wood, including solid and decaying wood, bark and cellulose, and might feed on other plant material such as leaves, grass and fungi. They might also consume cellulose in the form of herbivore droppings, and they play an essential role in the maintenance of ecological systems. The wood in buildings and other structures such as wooden power-poles and bridges is also suitable food for termites and infestation can cause considerable damage. On a local level, the Queensland Department of Housing spent $410,000 managing termite infestations in public housing during the 1999-2000 financial year. In the Ipswich, Woodridge and Capalaba areas in southeast Queensland the estimated cost of repairs for termite damage ranged from $18,000 to $60,000 per property (Queensland Built Environment Research Unit January 2000, pers. com.). On average, termite infestations cost approximately $1500 in treatment, and repairs of $5000 for each building affected (Caulfield 2002). It is estimated that 10% of current Australian houses have had, or will have, termite infestations, with 65% in some areas, resulting in a per annum cost of approximately $4 billion (Caulfield 2002). Management of termites, and eradication of exotic species is costly, with a campaign to eradicate the exotic West Indian drywood termite *Cryptotermes brevis* (Walker) in Queensland estimated to have cost $4.2 million by 1998 (Peters & Fitzgerald 1998). Worldwide, damage caused by termites is estimated at US$22 billion per annum in terms of damage to wooden structures (La Fage et al. 1988). Treatment and prevention of termite damage is therefore essential but might give rise to unwanted side effects.

Until 1995, effective persistent chlorinated hydrocarbon (organochlorine) termiticide treatments were used to create barriers to termites in Australia. These pesticides have since been withdrawn and replaced with other, less persistent, chemicals and physical barriers. As a consequence, chemical termiticides need to be reapplied on a regular basis, averaging three to five years depending on local conditions. Currently, chlorpyrifos and bifenthrin are the termiticides in most common usage in Australia, with varying formulations of chlorpyrifos, permethrin, alpha-cypermethrin, bifenthrin, imidacloprid and fipronil registered for use in Australia by the Australian Pesticides and Veterinary Medicines Authority (APVMA). With the withdrawal of chlorpyrifos from use in a number of applications in the USA, the use of chlorpyrifos in Australia has reduced, with a corresponding increase in bifenthrin and imidacloprid use.

The major use of termiticides in Australia is for preventative treatments applied to buildings before and during construction, and to existing structures such as timber power-poles and bridges. Treatments of infestations in existing buildings, and remedial applications to other wooden structures are also significant.

This review broadly examines historical, current and future uses of termiticides in Australia and the migration and fate of currently used termiticides in soils. The following subject areas are examined:

- termite ecology
- historical and current termite management in Australia
- migration, behaviour, efficacy and persistence of currently used termiticides in soils
- toxicology and risks to human health
- potential effects of termiticides on non-target species
- biological control and alternative treatments
- future directions.

The focus is on identifying risks to the environment and human health and identifying knowledge gaps, research priorities and industry concerns. Where detailed information or reviews are available elsewhere, either electronically or in print, users are directed to those.

Termite Ecology

Australia's termite fauna is diverse, represented by five families (Mastotermitidae, Termopsidae, Kalotermitidae, Rhinotermitidae and Termitidae). Within these families there are 40 known genera and more than 266 described species. Termites may be grouped as dampwood, drywood or subterranean, depending on their foraging habits, biology and ecology.

Dampwood termites (e.g. *Neotermes insularis* (Walker), *Glyptotermes brevicornis* (Froggatt), and *Ceratokalotermes spoliator* (Hill) live in rotten wood, particularly logs or damp sections of trees. They sometimes inhabit rotten wood in structures, but are generally not considered to be of economic concern. *G. brevicornis* and *C. spoliator* can cause damage to structures such as power-poles. Drywood termites have no contact with the ground, and obtain their moisture requirements from the wood in which they live. Drywood termites are considered to be of economic importance, with *C. brevis* identified as the most destructive species (Peters & Fitzgerald 1998).

Subterranean termites of the genera, *Reticulitermes*, *Schedorhinotermes*, *Heterotermes*, *Nasutitermes*, *Mastotermes* and *Coptotermes*; are those that require contact with the ground or moisture, and construct tunnels or galleries. They are responsible for major damage to timber in buildings, utilities (e.g. power-poles, bridges) and in trees. Subterranean termites forage for food by means of covered runways (galleries), which extend from the central nest to food sources above or below ground. They communicate by secreting pheromones. Colonies consist of distinct castes, each performing a specialised task within the colony. Workers provide food for the colony, feed the other caste members and excavate galleries, while soldier termites defend the colony and tend to be equipped with mandibles or a proboscis (depending on the species) for defence. Reproductive castes are winged, and tend to swarm after summer rains to establish new colonies. On returning to the ground they shed their wings and search for food and moisture in the soil. After digging a chamber near a food source, the pairs mate and a colony is begun. Supplemental reproductives can be formed in some species within 3 to 4 months after separation from the founding colony (Pawson & Gold 1996), making it important not to fractionate the colony during management procedures.

Termites play a key role in the nutrient cycles of ecosystems in which they occur, serving an ecological function by converting dead trees and grasses into organic matter, and they are important in the recycling of nutrients and the enrichment of soils. Whitford (1991) recorded long-term negative effects on soils, vegetation and organic material following removal of termites from desert rangelands in New Mexico. Soils without termites had lower infiltration and water content and higher

nitrogen levels. The changes in soil characteristics resulted in different plant species composition and changed vegetation productivity (Whitford 1991).

In Australia, the majority of pest termites are native (the exception is *C. brevis*), which means they are well adapted to local conditions and may be quite resistant to treatments when compared to situations in the US where the major pest species, *Coptotermes formosanus* (Shiraki), is exotic, restricted in range and habitat and therefore potentially easier to control. The majority of baseline studies into the effectiveness of chemical treatments has been conducted in North America on *C. formosanus* and may not be transferable to Australian conditions and species. Of the termites that exist in Australia, relatively few are considered to be pests of sound timber, with the most economically important being *Coptotermes acinaciformis* (Froggatt), *Coptotermes acinaciformis raffrayi* (Wasmann), *Coptotermes michaelseni* (Silvestri), *Mastotermes darwiniensis* (Froggatt), *Nasutitermes exitiosus* (Hill), and *Schedorhinotermes reticulatus* (Froggatt).

The management of social insects differs from that of other pests. The complex social interactions of termites (grooming, chemical communication, caste differentiation) need to be taken into account, and can be exploited when treating colonies. For example, worker castes gather food and return it to the colony, so by providing poison baits or dusting workers with a slow-acting toxin, termiticides can be transported to, and spread within, the nest. Grooming behaviours by termites within the nest can result in the toxic dusts that coat returning workers being spread quickly to other colony members. There is some evidence that social interactions might help termite colonies overcome physiological stresses such as starvation and disease, and possibly the effects of poisons. For example, DeSouza et al. (2001) exposed groups of *Cornitermes cumulans* (Kollar) workers to chlorpyrifos in order to evaluate effects of group size on tolerance to poisoning. Survival of poisoned and control termite groups depended on the numbers of individuals in the group, with termites in groups surviving longer than those held alone, up to a certain density. The significance of this is that there might be some socially induced ability to overcome physiological stress (DeSouza et al. 2001). Social functions might enhance mechanisms that can help in the detoxification of termites exposed to poisons. The mechanisms by which this occurs are not understood and require further investigation. There is much variation in the breeding strategies of termite species, which in turn might influence the effectiveness of chemical treatments.

Tunnelling behaviours of termites through soil treated with pesticides needs to be understood. The ability of a termite colony to tunnel through pesticide-treated soil may be affected by population size. For example, Jones (1990) found that *C. formosanus* at high population levels were able to construct more and longer tunnels and therefore were potentially able to cross termiticidal barriers. Other factors may influence tunnelling, for example in *C. formosanus* the size, length and complexity of search tunnels excavated by termites is dependent upon available food sources (Hedlund & Henderson 1999). When food size (the amount of food available in the feeding chamber) is large, feeding activity and survival are increased, but search tunnel volume is reduced.

Termite foraging activity appears to be influenced by seasonal variation in temperature. Evans and Gleeson (2001) found that in artificial feeding stations *Coptotermes lacteus* (Froggatt) foraged further from their mounds in summer, whereas in winter they were more likely to be found clustered close to mounds. Variation in foraging activity occurred according to air and soil temperature changes, demonstrating that seasonal and daily changes influence activity and foraging

behaviour (Evans & Gleeson 2001). For *C. lacteus* and *C. formosanus* at least, termites adjust search activity in response to available food supply and environmental conditions. An understanding of these behaviours might be useful for increasing the efficacy of baiting for termite management as bait and monitoring systems rely on termites finding and feeding on the baits.

Termite Management in Australia

In Australia prior to 1962 arsenic dusting was the most common means of small-scale termite management. Nests were located and dusted directly with arsenic trioxide powder. Organochlorines came into common usage after 1962, and provided long-term protection against termites. Organochlorines used under slabs offered protection from termites for up to 30 years, and pre-treated wood for up to 10 years. For some species, organochlorine baits were used with success e.g. Mirex packed into baits of *Eucalyptus regnans* and agar gel was effectively used against *M. darwiniensis* in the Northern Territory. The Northern Territory and Western Australia received an extension to its use of organochlorines in the form of Mirex and Mirant baits for *M. darwiniensis* management only. Mirex (Product No. 42539) and Mirant (Product No. 42410) are currently registered until June 2004. These products are largely used for prevention of termite infestation of fruit trees in orchards.

Large scale termite management prior to 1995 was based on the use of highly persistent organochlorine insecticides (Peters & Fitzgerald 1998), such as aldrin, dieldrin, chlordane and heptachlor (known collectively as cyclodienes, because of their particular chemical structure), which were well suited to slab-on-ground housing construction. Because of their chemical stability, they were extremely effective, and had no immediate adverse health effects at the levels of exposure arising from the approved use. Due to increasing environmental and public health concerns, associated primarily with their persistence in the environment and their tendency to accumulate in the fat of animals and humans, these chemicals were voluntarily withdrawn from the market by 1995 and alternative strategies for termite management developed. Some states (e.g. Western Australia) had prohibited the use of organochlorines for post-construction before 1995.

Organophosphates and pyrethroids have since been offered as alternatives to the organochlorines. However, they are effective against termites for a much shorter time, have shorter lifetimes, and break down relatively quickly compared to the organochlorine termiticides, and thus do not provide effective long-term protection. The need for more regular applications of the newer, less-persistent chemicals results in an increased chance that householders and pest control operators will be more frequently exposed to the chemicals.

Current Chemical Termite Management

The withdrawal of the organochlorines as termiticides in Australia has of necessity encouraged the introduction and investigation of new products and methods. Previously, because the organochlorines were so effective there was apparently little commercial incentive to develop new chemicals or barrier controls. Chemical control can be divided broadly into preventative and curative. New buildings are treated with a chemical barrier beneath and surrounding their slabs. Where access to slabs is not practical for under-slab termiticide application, reticulation systems can be installed pre-construction, enabling repeated application of termiticides over time. New buildings must comply with the Building Code of Australia, which includes Australian Standards for the prevention of termite infestations (AS 3660.1. 2000, 'Termite management - Part 1: New building work'). The standard describes the use of

barriers and resistant materials in construction. There are also guidelines for the prevention of infestations in existing buildings (AS 3660.2. 2000, 'Termite management - Part 2: In and around existing buildings and structures - Guidelines'). Where a termite infestation has occurred, chemicals may be applied as spot treatments, sprays, baits or gases.

There are a range of termiticides registered for use in Australia as varying formulations of synthetic pyrethroids (e.g. permethrin and bifenthrin), organophosphates (chlorpyrifos), the chloronicotinyls (imidacloprid), arsenic trioxide (as a dust for spot treatments), and recently a phenylpyrazole (fipronil), along with wood preservatives such as boron, copper, fluorine and creosote that are used alone or in combination to prevent termite attack.

The organophosphate termiticides

Chlorpyrifos is a widely used organophosphate insecticide, which is active against a range of insect pests. The toxicity of chlorpyrifos results from the action of the metabolite chlorpyrifos oxon, which inactivates acetylcholinesterase (AChE), an enzyme intimately involved with transmission at neural junctions. Overstimulation of the peripheral nervous system then results in death. Chlorpyrifos acts on insects primarily as a contact poison, and has some action as a stomach poison. The solvents used in formulations are usually hexane or xylene. The environmental fate and effects of chlorpyrifos, including comprehensive solubility, sorption and degradation data, has been extensively reviewed by Racke (1993) and in the USEPA's chlorpyrifos registration eligibility documents (1999).

Chlorpyrifos has low solubility in water and partitions readily from aqueous to organic phases (Racke 1993). Because it is tightly adsorbed by soil particles, it is not expected to leach significantly and movement through soil is limited. Volatilisation from the soil surface due to its intermediate vapour pressure can contribute to loss. Cleavage of the phosphorothioate ester bond to form 3,5,6-trichloro-2pyridinol (TCP) is the major path to degradation. Once this occurs TCP is degraded by photolysis. The vapour phase of chlorpyrifos can be destroyed by hydroxyl radicals. Alternatively, it might be adsorbed by airborne particles, and might be reactivated and taken up by local biota. High soil temperature, low organic content and low acidity increases the degradation of chlorpyrifos.

Chlorpyrifos has a half-life in soils ranging from 2 weeks to over 1 year, depending on the soil type, climate, and other conditions. As documented by Racke (1993), depletion rates for chlorpyrifos in soil increase with temperature, with each 10oC rise in temperature approximately doubling the depletion rate (Murray et al. 2001). Chlorpyrifos dissipates more rapidly from water than from soil (Racke 1993), and volatilisation is the primary route of loss of chlorpyrifos from water. However, because it also partitions from the water column to sediments, desorption from sediments can cause long term residual contamination of the water column.

The natural pyrethrin and synthetic pyrethroid termiticides

Pyrethrins are natural insecticides derived from the flowers of certain species of *Chrysanthemum* plants. The plant extract (pyrethrum) contains pyrethrins (pyrethrin I and II). Pyrethroids are synthetic analogues of pyrethrins. They are used to form chemical barriers to repel and/or kill termites and are used in the control of many insect pests. Pyrethroids act by inhibiting the nervous system of insects inhibiting ATPase enzyme production, and affecting sodium ion channels, and in some cases affecting gamma-aminobutyric acid (GABA) action.

Permethrin is the active ingredient in a number of broad-spectrum insecticides,

including termiticides. In most cases permethrin (25:75 CIS: TRANS) is mixed with hydrocarbon solvents. Permethrin is currently available in Australia as a dust, or liquid used to treat active infestations. Elsewhere, it is used in wood preservatives to prevent termite attack, for example in Rurply industrial plywood protectant (Bayer Cropscience Pty Ltd), the active ingredient is permethrin combined with benzalkonium chloride.

Bifenthrin is used in Australia as a barrier termiticide as well as for treatment of active infestations. Bifenthrin is also used as a wood preservative in Australia. Because of its physicochemical properties bifenthrin is likely to be immobile in soil and relatively insoluble in water. However, because it is attached to colloidal particles, it may be transported with them into waterways and groundwater. Bifenthrin will exist in the atmosphere in particulate and vapour phases, where vapour phase bifenthrin is degraded by hydroxyl radicals and ozone. Because pyrethroids are readily degraded by microorganisms, it is likely that microbial degradation of bifenthrin will occur.

Alpha-cypermethrin is a racemic mixture of two of the four cis isomers - the (1R, cis)S and (1S, cis)R isomers of cypermethrin. It is registered in Australia for use as a chemical barrier. Deltamethrin is used in impregnated barrier products. In Australia deltamethrin is sometimes, but not often, used in termiticides.

The chloronicotinyls (nicotinoids)

The nicotinoids are a relatively new class of insecticide, modelled on natural nicotine. Imidacloprid is a systemic, chloro-nicotinyl that works by interfering with the transmission of stimuli in the insect nervous system. It causes blockage of the nicotinergic neuronal pathway, leading to acetylcholine accumulation, resulting in insect paralysis, and/or death. Imidacloprid-based insecticide formulations are available as dustable powder, granular formulations, seed dressing (flowable slurry concentrate), soluble concentrate, suspension concentrate, and wettable powder. It is currently registered in Australia for post-construction applications only.

Hydrolysis and aerobic soil metabolism data indicate that imidacloprid is persistent and mobile with a tendency to leach. Under some circumstances contamination of groundwater is possible (Cox 2001). Imidacloprid can be translocated via plant roots. When used to form termiticide barriers, consideration should be given to the risk of product losses due to plant uptake, and possible ingestion by grazing animals.

The phenyl pyrazoles (fiproles)

Fipronil is a phenyl pyrazole, a new class of chemical. Fipronil has been under consideration by the APVMA for registration in Australia as termiticide and gained registration for protection of structures from subterranean termite attack in October 2002. It was previously used only for topical flea and tick control on dogs, and as an agricultural pesticide. Fipronil is a disruptor of the insect central nervous system via the GABA channel, acting with contact and stomach action. It blocks the GABA-gated chloride channels of neurons in the central nervous system, resulting in neural excitation and death of the insect.

Growth inhibitors

Growth inhibitors or insect growth regulators work by interrupting or inhibiting the life insect cycle. These chemicals inhibit chitin production, and thereby prevent the juvenile termites from molting and growing to the next life stage. Hexaflumuron is a benzoylurea insecticide, which is a growth (chitin synthesis) inhibitor, used in bait stations. Similar are Triflumuron and Chlorfluazuron chitin synthesis inhibitors, also used in bait stations (Dow Agrosciences Recruit II MSDS).

Other chemicals

Arsenic trioxide is still used for termite control, in bait and switch management

programs, and for direct dusting onto nests. The arsenic content in these dusts ranges from 379g/kg to 500g/kg.

Methyl bromide gas is used to fumigate drywood termite infestations. It is pumped into sealed buildings or wooden articles.

Boron complexes for wood protection are being developed with some success by Commonwealth Scientific and Industrial Research Organisation (CSIRO) and the Centre for Green Chemistry at Monash University. With leach-resistant boron binding to wood as the ultimate aim, high levels of mortality for C. acinaciformis have been recorded for particular boron formulations (Humphrey et al. 2002). Boron impregnated rods are inserted into wooden railway sleepers as a slow release preservative and termite repellent.

Impregnated wood preservatives include mixtures of copper, arsenic and other chemicals used for protection against termite and borer attack. Generally, these are used in pressure impregnation of new wood. Combinations of disodium octaborate tetrahydrate and benzalkonium chloride are also used in formulations of insecticidal wood preservative.

Creosote is an external timber preserving oil that is painted onto external timber as a means of preservation and a termite deterrent. Previously creosote was typically used in combination with aldrin.

Mirex and Mirant (Mirex 2.0 g/kg) baits are the only organochlorine cyclo-compounds termiticide currently used for termite control in Australia. They are registered only for use in the Northern Territory and Western Australia for *M. darwiniensis* control (registered currently to June 2004), primarily for use in the prevention of termite infestations of fruit trees under cultivation.

Migration and Behaviour of Currently Used Termiticides in Soils

Environmental variables are important for the efficacy, transport, degradation and volatilisation of termiticides. The period for efficacy depends on termite activity, climate, soil type, disturbances and landscaping practices. Following application, termiticides can be lost from the original application site via lateral and vertical movement into surrounding soil and groundwater. It is then possible that chemicals will be transferred to local biota and animals.

The movement of pesticides through soil largely depends on the physical properties of individual chemical actives, the presence of water and biota and organic material, as well as the solvent/s that have been used in the pesticide formulation. Soil characteristics, pH and the presence of organic matter play major roles in determining the persistence and efficacy of chemicals. The half-life of a chemical in the soil will depend on a combination of these factors. Sandy soils with low biota/low organic content and wet conditions generally result in increased transport, whereas those with high clay and organic content tend not to allow chemical migration as readily. The mineral content of the soil will influence degradation, by affecting adsorption rates or catalysing decomposition. Termiticides, in particular the organophosphates, can be lost from the soil through movement of volatiles into the air due to their significant vapour pressures at ambient temperatures.

Soil properties, including organic content, silt and clay content, pH and cation exchange capacity might affect the bioavailability and dispersal of pesticides. In comparisons of different soil types treated with imidacloprid, pesticide effects on *Reticulitermes flavipes* (Kollar) were greatest in sand and reduced in silty clay loam soils (Ramakrishnan et al. 2000). Testing of four soil types with six termiticide formulations indicate that soils have a significant influence on the efficacy of the chemicals (Forschler & Townsend 1996). Of the termiticides tested (including chlorpyrifos, fenvalerate, cypermethrin and permethrin), all had concentrations lethal to termites that were at least seven times lower in sandy

soils than in sandy loam or sandy clay loam (Forschler & Townsend 1996). Murray et al. (2001) examined the stability of chlorpyrifos in six Australian soil types and found that soil pH had no effect on the rate of degradation. In contrast, acidic soils with low clay and organic content in Texas were found to be the most stable in terms of remaining bioavailability of pesticides, while alkaline soils with high clay content and organic compositions higher than 1% were least effective in retaining termiticide residuals over time (Gold, Howell & Jordan 1996).

The Efficacy and Persistence of Currently Used Termiticides

The physicochemical properties of termiticides directly influence their behaviour, persistence and bioavailability after application. Australian evaluations of the organophosphates in soils suggest an effective life for chlorpyrifos of seven to 12 years if covered, to as little as four years when exposed to the elements (Lenz, Watson & Watson 1988), and suggest that in tropical Australia, the chemical be reapplied at three year intervals. A field study of leaching and degradation of pesticides (including chlorpyrifos, chlorthal dimethyl, fenamiphos, fenamiphos plus metabolites, linuron, metalaxyl, metribuzin, prometryne, propyzamide and simazine) in coastal sandy soils (pH 5.3) in Western Australia demonstrated that degradation rates vary widely between pesticides (Kookana, Di & Aylmore 1995). In this study, chlorpyrifos degraded significantly, but did not move considerably. It was concluded that it was unlikely to reach groundwater (Kookana, Di & Aylmore 1995). A study of six termiticides (bifenthrin, chlorpyrifos, cypermethrin, fenvalerate, permethrin and isofenphos) applied to different soil types in Texas, demonstrated significant differences in effectiveness (as measured by termite activity), bioavailabilty and residue. The most stable over 5 years were permethrin and fenvalerate. Isofenphos was the least stable with significant loss of activity within 24 months after application (Gold, Howell & Jordan 1996).

Barrier efficacy tests of a range of termiticides (Dursban and Equity [chlorpyrifos]; Dragnet [permethrin]; Prevail and Demon [cypermethrin]; Biflex [bifenthrin]; Pryfon [isophenphos]; PP321 [lambda-cyhalothrin pyrethroid] and Sumithion [Fenitrothion]) in the US indicate that while all formulations provide equal protection against *R. flavipes*, *C. formosanus* was able to tunnel deeper into sand treated with organophosphates than in sand treated with pyrethroids (Su, Ban & Scheffrahn 1993). Isofenphos lost effectiveness within 1 year, and rapidly degraded - probably as a result of alkaline sand (pH 8.1), microbe activity and high rainfall (Su, Ban & Scheffrahn 1993).

Pyrethroids (bifenthrin, cypermethrin, lambda-cyhalothrin and permethrin) appear to provide longer protection than organophosphates (chlorpyrifos, fenitrothion and isofenphos). According to 5 year field trials in Florida against *R. flavipes* (Su, Ban & Scheffrahn 1999), permethrin had the longest half-life (21.9 months) of the pesticides examined. Microencapsulated formulations generally result in longer persistence.

Summary of efficacy and longevity information

The physicochemical properties of each active ingredient and its formulation will influence potential impacts on the environment. For example, imidacloprid is less toxic to fish and aquatic invertebrates than are chlorpyrifos and bifenthrin (Table 1), but its physicochemical properties suggest that it has a high leaching potential (Health Canada 2001). Imidacloprid is very soluble in water and therefore newly applied imidacloprid is subject to runoff (Health Canada 2001). However, it becomes strongly bound to soils in which it is allowed to age for 4 to 8 weeks (Health Canada

2001). In comparison, both bifenthrin and chlorpyrifos have low solubility in water and are therefore not likely to leach or move from soil particles. They can be involved in runoff if the colloidal particles to which they are attached are washed into waterways, which could result in contaminated sediments.

Reapplication of termiticides is required at intervals of between 2 and 10 years depending on the climate, soil type and termite species and activity in particular situations. In all cases annual inspections for termite infestations are advised. Reapplication of chlorpyrifos and bifenthrin is required every 2 to 10 years depending on the situation (according to Dursban and Biflex label information). Sub-floor applications will remain effective for considerably longer than exposed areas. For imidacloprid, the interval may be as short as 1 to 2 years (Bayer Cropscience Premise SC MSDS 2002). Fipronil has low mobility in soil, but reapplication may be required after 2 to 5 years depending on the situation (BASF Termidor label information). Regular reapplication increases the risk of accidental or occupational exposures and the possibility of environmental contamination through runoff or spillage.

Solvents

Solvents are used in termiticides to dissolve or mix the active ingredients into a liquid, slurry or foam for application. Solvents and adjuvants used may include a range of petroleum distillates, isopropanol, methanol, toluene, xylene, penetrants, stickers (film extenders), surfactants, detergents, dusts, emulsifiers and anticaking agents. The solvents used to formulate commercially available termiticides are of interest, since alone or in combination with other chemicals they may have significant effects on the active ingredients and their environmental fate. In some cases, solvents may cause toxic effects. Often, the solvents used in pesticide formulations are treated as inerts, and not listed in detail on pesticide labels. It is often difficult to obtain information about the exact mix of adjuvants and solvents used because of commercial in confidence issues. Because particular combinations of solvents and adjuvants used may give companies a competitive advantage, their inclusion in labelling is often sparse. Also, continuous subtle changes to the formulations of products are made to suit particular purposes, resulting in several non-identical products sharing the one original name. Because of the varied requirements for termiticides, solvents may not always be suited to all applications and boutique formulations might be needed for specific uses. For example, around power-poles, a formulation is required to remain in a restricted area and not spread away from the pole when applied, whereas under the slabs of houses it is desirable that the termiticide be distributed evenly underneath the slab.

There might be interactions leading to additive or reduced-level effects between pesticides and other components. For example, Axelrad, Howard and McLean (2002) found synergism between chlorpyrifos and regular spirit solvent and also with chlorpyrifos and pyrethrum with combinations significantly more toxic than the individual compounds. They also found additive effects (in terms of neurological toxicity) in other combinations of chlorpyrifos and pyrethrum with individual components of a commercial formulation. Their data suggest that exposure to multiple pesticide formulations can result in synergistic neurotoxicity. Therefore the hazard associated with a particular pesticide might be amplified in the presence of another pesticide.

There is some evidence that solvents alone might affect termites. For example, sand treated with solvents alone might be toxic to termites, with 24 to 86% of *Reticulitermes hesperus* (Banks) killed after 24 hours of exposure to the air-dried solvent treated sand (Rust & Smith 1993).

Toxicology and Risks to Human Health from Termiticide Usage

Termiticides must be approved and

registered by the APVMA prior to registration for use in Australia A proposed pesticide must undergo a rigorous assessment process, which includes assessment for possible effects on health and on the environment. Toxicity and potential public health issues are assessed by the Australian Department of Health and Ageing, and the National Occupational Health and Safety Commission (NOHSC) assesses occupational safety issues. Environment Australia assesses environmental safety, and the APVMA assesses chemistry and efficacy. Toxicological data for termiticides are derived largely from animal studies, and involve doses of active ingredients that are generally much higher than those for likely human exposures. However, humans might be more or less sensitive to particular chemicals than the animals used, and so animal experiments do not always translate well to human risk. Toxicity tests identify "no observable effect levels" (NOELs) in animals, and these are used to establish acceptable limits for exposure in humans at which no adverse health effects would be expected - the Acute Reference Dose (ARD) and Acceptable Daily Intake (ADI). Safety factors, usually as a factor of 100 are derived from NOELs to determine a safe level of exposure for humans that is 100-fold lower than that for animals. This is to account for uncertainties in applying animal data to human exposure. Testing is carried out on the active ingredients, and in many cases also on the available formulations. Where testing only includes the active ingredient, effects due to carriers, additives, solvents, adjuvants and mode of application might not be fully understood. Because formulations are designed to enhance environmental persistence, it is highly desirable that long-term effects on environmental and public health under Australian conditions, using Australian species, are investigated, with emphasis on long-term sublethal effects that are not assessed in acute studies.

When applied according to safety guidelines and manufacturers' recommendations, the risk of poisoning or exposure to high levels of termiticides is minimal. Trace residues nonetheless can sometimes be found in water, and spillages might occur. Indoor air pollution caused by pesticides can be of concern in buildings with poor ventilation, resulting in long-term exposure to low levels of chemicals. For example, chlorpyrifos was recorded in indoor air in Japan, and did not decrease 5 years after application for termite control (Yoshida, Taguchi & Fukushima 2000). Japanese investigators found that levels of chlorpyrifos and S-421 in indoor air ranged up to 0.258μg/m^3 and 0.174μg/m^3 respectively in houses where chlorpyrifos had been applied to building timbers for termite control (Katsura et al. 1996). In Western Australia, 19 of 22 houses monitored for pesticide levels had detectable levels of pesticides in indoor air; in order of frequency: heptachlor; dieldrin; chlordane; aldrin and chlorpyrifos. Although chlorpyrifos was least frequently detected, the mean concentration of 2.23μg/m^3 was significantly higher than that of other pesticides (Dingle et al. 1999), and substantially higher than the NOHSC occupational guideline of 0.2μg/m^3.

Chlorpyrifos is moderately toxic to humans, but highly toxic to fish and aquatic organisms (refer to Table 1 for LC_{50}/LD_{50} values in animals). Poisoning from chlorpyrifos can affect the central nervous system, the cardiovascular system, and the respiratory system. It is a skin and eye irritant. While some organophosphates are readily absorbed through the skin, studies suggest that skin absorption of chlorpyrifos is limited in humans. Symptoms of acute exposure to organophosphate (and other cholinesterase-inhibiting compounds) may include the following: numbness, tingling sensations, loss of coordination, headache, dizziness, tremor, nausea, abdominal cramps, sweating, blurred vision, difficulty breathing or respiratory depression, and slow heartbeat (EXTOXNET 1996 Chlorpyrifos Pesticide

Information Profile). Very high doses may result in unconsciousness, incontinence, convulsions and death.

Some organophosphates, may cause delayed symptoms beginning one to four weeks after an acute exposure. In such cases, numbness, tingling, weakness, and cramping may appear in the lower limbs and progress to in-coordination and paralysis. Improvement may occur over months or years, and in some cases residual impairment will remain. Plasma cholinesterase activity levels may be inhibited when chlorpyrifos particles are inhaled. The USEPA (2000) analysis found exposure to Dursban (chlorpyrifos) on the skin, in food, or by inhalation could be harmful to human health. The USEPA said it had a particular concern with Dursban poisoning cases reported to federal officials. Repeated or prolonged exposure to organophosphates might result in the same effects as acute exposure, including delayed symptoms.

Chlorpyrifos is toxic by oral, dermal and inhalation routes. It is readily absorbed into the bloodstream through the gastrointestinal tract if ingested, through the lungs if it is inhaled, or possibly through the skin if there is dermal exposure. In humans, chlorpyrifos and its principal metabolites are eliminated rapidly. Chlorpyrifos primarily affects the nervous system through inhibition of cholinesterase. Animal studies suggest that chlorpyrifos is rapidly absorbed and metabolised to TCP, and the parent compound and metabolite are rapidly excreted in urine. In orally dosed pregnant rats (at 14 to 18 days gestation) AChE and BuChE activities were inhibited significantly in both maternal and foetal brains one hour after ingestion (Ashry et al. 2002). The dose used here was 50 mg/kg, which is 61% of the oral LD_{50} for female rats (refer to Table 1 for LD_{50} and LC_{50} values for selected animal species, and Table 2 for selected chronic toxicity data). In addition, neonatal rats orally administered much lower rates of chlorpyrifos, at 1mg/kg or 5 mg/kg, demonstrated deficiencies in cataechloaminergic synaptic function persisting into adulthood (Slotkin et al. 2002).

Available evidence suggests that chlorpyrifos is not teratogenic except at high doses or mutagenic (Table 3). It does not appear to have reproductive effects at levels of 1 mg/kg per day (Table 3). Chlorpyrifos is not considered to be a carcinogen (Table 4). There is no available evidence that it is mutagenic. However, Rahman et al. (2002) report dose-dependent DNA damage detectable in mice 24 hours after being fed chlorpyrifos at doses of 0.28 to 8.96 mg/kg body weight. Detailed toxicity assessments can be found in the USEPA's chlorpyrifos registration eligibility documents, toxicology chapter (1999).

Assessment of neurological function in a group of pesticide applicators exposed to chlorpyrifos (n=191) compared to non-exposed controls (n=189) revealed that there was no significant difference in clinical examinations. However, the exposed group under-performed in pegboard turning and postural sway tests and reported significantly more symptoms (memory problems, fatigue, loss of muscle strength). Eight exposed subjects who had reported prior chlorpyrifos poisoning had low performance on a number of tests (Steenland et al. 2000). If ingested, chlorpyrifos is eliminated primarily through the kidneys, but can accumulate in fat (Table 5) (WHO & FAO 1975).

The pyrethroids are generally not considered toxic to humans, but exposure can cause skin, eye and respiratory irritation, and sometimes cause allergic reactions. Tingling sensations in the hands and face might follow external exposure. Bifenthrin and alpha-cypermethrin poisoning can cause nerve damage, resulting in tremors, difficulty walking, agitation and abnormal gait. Bifenthrin is absorbed through intact skin if applied topically. If ingested bifenthrin is moderately toxic to mammals, and large doses may cause in-coordination, tremor, salivation, vomiting, diarrhoea, and

Table 1: Experimental LD50 and LC50 values for selected animal species

	Chlorpyrifos	Bifenthrin	Permethrin	Deltamethrin	*Alpha-cypermethrin	Imidacloprid	Fipronil
Rats Oral LD_{50}	155 mg/kg (WHO & FAO 1975); 95 - 270 mg/kg (EXTOXNET 1996).	54-70 mg/kg (EXTOXNET 1995).	430 to 4000 mg/kg (WHO & FAO 1984).	52->5000 mg/kg (EXTOXNET 1984).	63-98 mg/kg (WHO/IPCS 1992)	450 mg/kg (EXTOXNET undated).	97 mg/kg (NRA 1996).
Rats Dermal LD_{50}	>2000 mg/kg (EXTOXNET 1996); 135 - 202 mg/kg (WHO & FAO 1975).	>2000 mg/kg (FMC 2000).	4000 mg/kg (WHO & FAO 1984).		>2000 mg/kg (WHO/IPCS 1992).	>5000 mg/kg (EXTOXNET undated).	>2000 mg/kg (NRA 1996).
Rats Acute inhalation LC_{50}	>0.2 mg/L (4-hr inhalation) (EXTOXNET 1996).	0.61 mg/L/1 hr (FMC 2000).	0.685 mg/L (WHO & FAO 1984).		0.32-1.42 mg/L (4-hr air) (WHO/IPCS 1992).	>0.069 mg/L (aerosol); >532 mg/L (dust) (EXTOXNET undated).	0.682 mg/L (NRA 1996).
Mallard ducks Oral LD_{50}	112 mg/kg (EXTOXNET 1996).	2150 mg/kg (8-d dietary LC_{50}) (EXTOXNET 1995).	9900 mg/kg (EXTOXNET 1996).	>4640 mg/kg (EXTOXNET 1985).	>10000 mg/kg (WHO/IPCS 1992).	283 mg/kg (EXTOXNET undated).	
Japanese quail Oral LD_{50}			15500 mg/kg (EXTOXNET 1996)	>10000 mg/kg (EXTOXNET 1984).		31 mg/kg	
Rainbow trout LC_{50}	0.009 mg/L (96-hour LC_{50}) (EXTOXNET 1996).	0.00015 mg/L (96-hour LC_{50}) (EXTOXNET 1995).	0.0054 mg/L (48-hour LC_{50}) EXTOXNET 1996).	0.00059-0.00197 mg/L (WHO/IPCS 1990)	0.0028 mg/L (WHO/IPCS 1992).	211 mg/L (96-hr LC_{50}) (EXTOXNET undated).	approx. 0.246 mg/L (USEPA 1996).
Bluegill sunfish LC_{50}	0.01 mg/L (96-hour LC50) (EXTOXNET 1996).	0.00035 mg/L (96-hour LC_{50}) (EXTOXNET 1995).	0.0018 mg/L (48-hour LC50) (EXTOXNET 1996).	0.0012 µg/L (WHO/IPCS 1990)			approx. 0.083 mg/L USEPA 1996).
Daphnia LC_{50}	*Ceriodaphnia* 60ng/L (EXTOXNET 1996)	*C. dubia* approx. 0.00007 mg/L (Mokroy & Hoagland 1989); Daphnia magna 0.0016 mg/L (96 hr LC_{50}) (EXTOXNET 1995).		*Daphnia* 0.005 mg/L (48-h LC_{50}) (Stevenson et al. 1978 in WHO/IPCS 1990).	*Daphnia magna* 0.0001-0.0003 mg/L (WHO/IPCS 1992)	*Daphnia magna* 1.8 mg/L NOEC; 85 mg/L (48-h LC_{50}) (EXTOXNET undated).	0.190 mg/L (USEPA 1996).
Honey bees Apis mellifera LD_{50}	0.114 µg/bee (WHO & FAO 1975)		0.11 µg/bee (contact) (Stevenson et al. 1978 in WHO 1990).	0.079 µg/bee (oral); 0.051 µg/bee (contact) (Stevenson et al. 1978 in WHO/IPCS 1990).	0.13 µg/bee (oral) 0.11 µg/bee (contact) (Murray 1985 in WHO/IPCS 1992).	0.008µg/bee (EXTOXNET undated).	0.00417 µg/bee (oral); 0.00593 µg/bee (contact) (NRA 1996).

*Note: Results may vary according to carrier and conditions. *variation in toxicity depends on the ratio of cis/trans- isomers present in cypermethrin and alpha-cypermethrin.This may reflect different mixtures of isomers.*

Table 2: Summary of selected chronic toxicity data

	Chlorpyrifos	Bifenthrin	Permethrin	Deltamethrin	Alpha-cypermethrin	Imidacloprid	Fipronil
Chronic Toxity	Oral: dogs 3.0 mg/kg/day for 2 years: increased liver weight; signs of cholinesterase inhibition occurred at 1 mg/kg/day. Rats and mice fed technical chlorpyrifos for 104 weeks showed only cholinesterase inhibition. 2-year doses of 1 and 3 mg/kg/day in rats and dogs showed moderate depression of cholinesterase. Measurable change in plasma and red blood cell cholinesterase levels seen in workers exposed to chlorpyrifos spray. Human volunteers ingesting 0.1 mg/kg/day chlorpyrifos for 4 weeks showed significant plasma cholinesterase inhibition.	Oral: dogs fed up to 2500 ppm for 1-year resulted in a NOEL of 41 mg/kg/day resulted in increased cholesterol levels in blood, some stress to the liver (elevated liver cytochrome p-450 levels). Adverse effects in rats fed 300 ppm: increased thyroid lesions in males. At 900 ppm: decreased body weight gain in females.	Oral: dogs fed 5 mg/kg/day for 90 days, no adverse effects. Rats fed 150 mg/kg/day for 6 months showed slight increase in liver weights. Levels of (0.1 ppm for 3 to 6 weeks after hatching) in the diet of chickens (0.1 ppm for 3 to 6 reportedly suppress immune system activity.	Oral: mice fed 12 mg/kg NOEL Rats fed >10 mg/kg/day NOEL	Oral: Dogs fed 270 mg/kg/day for 13 weeks showed signs of intoxication, were otherwise unaffected. NOEL of 90 mg/kg/day (WHO/IPCS 1992). Rats: 200 mg/kg/day for 5 weeks, no toxic effects (WHO/IPCS 1992).	Oral: NOEL in dogs 41 mg/kg/day for 52 weeks. NOEL in male rats 5.7 mg/kg/day, 7.6 mg/kg/day in females. In rats: 16.9 mg/kg/day caused increased thyroid lesions in males and decreased weight gain in females. In rats: 51.3 mg/kg/day in males and 73 mg/kg/day in females - weight change in liver, kidney, lung, brain, spleen, adrenal and gonads (NPIC 1997).	Oral: Dogs fed 10 mg/kg/day for 13 wks - weight loss, and tremors. Fed 2mg/kg/day - reduction in appetite. (NRA Fipronil Evaluation). NOEL in fingerlings in 90 day life stage 0.01 mg/L^{-1} (NRA Fipronil Evaluation).

Note: Compiled from EXTOXNET Pesticide Information Profiles and WHO/FAO Pesticide Data Sheets unless otherwise referenced.

irritability to sound and touch (EXTOXNET 1995 Bifenthrin Pesticide Information Profile). Permethrin affects the central and peripheral nervous system, and exposure might cause tremors, salivation, hyper-excitability and paralysis. Deltamethrin poisoning results in different symptoms than those of other pyrethroids - mammals undergo salivation and writhing and rolling convulsions, and other type II motor symptoms. Symptoms of deltamethrin poisoning in humans include convulsions, ataxia, tremors and vomiting. The pyrethroids are generally quickly broken down in mammals and excreted in the urine and faeces, with the remainder accumulating in fatty tissues (Table 5) (EXTOXNET 1995 Bifenthrin Pesticide Information Profile).

Imidacloprid poisoning is likely to cause similar signs to nicotinic poisoning such as fatigue, twitching, cramps, difficulty breathing and muscle weakness including the muscles necessary for breathing. Toxic effects are listed as reduced muscle tone, tremors, apathy and muscle cramps with associated difficulty breathing in severe cases (Therapeutic Goods Administration Australia [TGA] 2001). Imidacloprid is not irritating to the skin and does not cause skin

Table 3: Summary of selected health assessment data - reproductive and teratogenic effects

	Chlorpyrifos	Bifenthrin	Permethrin	Deltamethrin	Alpha-Cypermethrin	Imidacloprid	Fipronil
Reproductive Effects	No effects on reproduction in a three-generation study in rats fed dietary doses up to 1 mg/kg/day. In rats were fed 1 mg/kg/day for two generations, a slight increase in the number of deaths of newborn offspring was recorded.	Maternal toxicity NOEL: 1 mg/kg/day for rats; 2.67 mg/kg/day for rabbits. Developmental toxicity NOEL: 1 mg/kg/day for rats, >8 mg/kg/day for rabbits.	250 mg/kg/day of permethrin fed to female rats during the 6th-15th day of pregnancy affected fertility.	3-generation NOEL in rats fed >2.5 ng/kg/day. Slightly embryotoxic in a 3-generation rat study, but no adverse affect on reproduction. Significant maternal and perinatal pup weight losses, which reversed upon cessation of treatment (WHO/IPCS 1990).		NOEL of 8 mg/kg/day in rats.	Daphnids NOEL 21 day reproductive testing at about 0.01 mg/L^{-1} (NRA Fipronil Evaluation). Rats fed 15mg/kg/day:reduced bodyweight of F1 offspring and reduced mating performance (NRA Fipronil Evaluation).
Teratogenic Effects	No effects in pregnant rats fed doses up to 15 mg/kg/day for 10 days. At 25 mg/kg/day for 10 days, minor skeletal variations and a decrease in foetal length. No birth defects in offspring of male and female rats fed 1 mg/kg/day over a three-generation reproduction and fertility study.	No teratogenic effects at the highest levels tested (100 ppm, ~5.5 mg/kg/day) in a two-generational study in rats.	Not embryotoxic nor teratogenic in a mouse study at dosage levels of 5-150 mg/kg from days 7-12 of gestation. No adverse effects on weaning efficiency.	No activity observed in mice, rats or rabbits even at toxic doses in the pregnant females (WHO/IPCS 1990).		NOEL of 30 mg/kg/day in rats, based on skeletal abnormalities observed at the next highest dose tested of 100 ppm. In rabbits: days 6-19 of gestation, NOEL of 24 mg/kg/day; abnormalities were observed at 72 mg/kg/day (highest dose tested).	

Note: compiled from EXTOXNET Pesticide Information Profiles and WHO/FAO Pesticide Data Sheets unless otherwise referenced.

sensitisation. Evidence of liver toxicity, tremors and weight loss were seen in animals fed high doses (TGA 2001).

Hexaflumuron has a relatively high LD_{50} in rats at >5000 mg/kg, indicating that it is not particularly toxic to mammals. Toxicity tests conducted in animals indicate that hexaflumuron is an irritant to eyes (in rabbits), and skin. The ingredients are not listed as carcinogenic, and it is not recorded to have caused reproductive damage in animals (National Pesticide Information Center [NPIC] 2000). Triflumuron has similar toxicological characteristics (TGA 2001). The chitin synthesis inhibitors have the advantage of low levels of toxicity to mammals but are very toxic to aquatic animals, and should therefore not be used in situations where it might enter waterways or groundwater.

Fipronil is irritating to the skin and eyes. It is toxic by oral, dermal and inhalation routes. It can cause skin irritation in cats and dogs, and may cause reproductive effects such as decreased mating, reduced litter size and decreased postnatal survival. Fipronil is not mutagenic (FAO/WHO Joint Meeting on Pesticide Residues [JMPR] 1997; JMPR 2000; Pesticides Action Network [PAN] 2000). The photodegradate MB46513 has a higher acute toxicity to mammals than fipronil itself by a factor of approximately 10

Table 4: Summary of selected health assessment data - mutagenic and carcinogenic effects

	Chlorpyrifos	Bifenthrin	Permethrin	Deltamethrin	Alpha-Cypermethrin	Imidacloprid	Fipronil
Mutagenic Effects	No evidence that chlorpyrifos is mutagenic.	Evidence inconclusive. Studies of mouse white blood cells inconclusive, but positive for gene mutation. However, other tests of bifenthrin's mutagenic effects, including the Ames test and studies in live rat bone marrow cells, were negative.	No mutagenic activity reported.	No mutagenic activity reported.	Not mutagenic based on tests with Salmonella, E. coli and Saccharomyces data and in-vivo and in-vitro tests with rat liver cells (WHO/IPCS 1992).	2/23 laboratory mutagenicity assays tested positive for mutagenic effects. Positive for causing changes in chromosomes in human lymphocytes, and for genotoxicity in Chinese hamster ovary cells at toxic concentrations.	
Carcinogenic Effects	No increase in incidence of tumours in rats fed 10 mg/kg/day for 104 weeks, or mice fed 2.25 mg/kg/day for 105 weeks.	No evidence of cancer in 2-year study of rats fed 10 mg/kg/day. However, 87-92 week feeding study of mice with doses of 7, 29, 71, and 86 mg/kg showed significantly higher, dose-related trend of increased tumour incidence in the male urinary bladder. Incidence significantly increased at 86 mg/kg/day.	Inconclusive	No evidence in rat, mouse or dog long-term diet studies, supported by microbial, mammalian cells and in vivo mammalian mutagenicity studies suggests no carcinogenic potential (WHO/IPCS 1990).		No carcinogenic effects in a 2-year carcinogenicity study.	Carcinogenic to rats at doses of 300 ppm, causing thyroid cancer related to disruption in the thyroid-pituitary status (PAN 2000).

Note: compiled from EXTOXNET Pesticide Information Profiles and WHO/FAO Pesticide Data Sheets unless otherwise referenced.

Table 5: Summary of health assessment data - fate in humans and animals

	Chlorpyrifos	Bifenthrin	Permethrin	Deltamethrin	Alpha-Cypermethrin	Imidacloprid	Fipronil
Fate in Humans and Animals	In rats, eliminated primarily through kidneys: 90% via urine, 10% via faeces. Accumulates in fat. Half-life of about 62 hours in humans. In cows, unchanged pesticide in faeces, not in urine or milk. Detected in milk (0.304 ppm) for 4 days after spray dipping with 0.15% emulsion	Rapidly broken down and excreted in mammals. Rats treated with 4-5 mg/kg, excreted 70% in the urine, 20% in faeces within 7 days. After 7 days, remainder found accumulated in fatty tissues.	Metabolised by mammals in the livers. Metabolites," of permethrin are quickly excreted Permethrin may persist in fatty tissues, with half-lives of 4 to 5 days in brain and body.	In rats: Eliminated in 2-4 days; half life in the brain of 1-2 days; half life in body fat 5 days.	Humans: urinary excretion of metabolites complete 48 hours after the last of five doses of 1.5 mg/kg/day. Rats: rapidly metabolised by hydroxylation and cleavage. Remaining 1% stored in body fat and eliminated slowly.	Absorbed from the gastrointestinal tract, eliminated via urine and faeces, 70-80% and 20-30%, respectively, of the 96% of the parent compound administered within 48 hours.	Excreted in rats via faeces (45%-75%) and urine (5-25%) (NPIC 1997)

Note: compiled from EXTOXNET Pesticide Information Profiles and WHO/FAO Pesticide Data Sheets unless otherwise referenced.

(USEPA Federal Register Environmental Documents 1998). Fipronil and its breakdown products are neurotoxic to mammals. Although there is no evidence of fipronil causing birth defects, it may cause delayed development at high doses. Fipronil is carcinogenic to rats at doses of 300 ppm, causing thyroid cancer related to disruption in thyroid-pituitary status (PAN 2000). However, it is considered to be non-carcinogenic (JMPR 1997, 2000). Studies have shown that there is potential for bioaccumulation of the photodegradate MB46513 in fatty tissues (PAN 2000).

Arsenic trioxide is highly toxic and a carcinogen, but because it is used in bait stations or applied as a dust directly to nests, potential for non-occupational exposure is limited. Signs of poisoning include stomach cramping, vomiting and diarrhoea, and long-term effects of exposure include increased incidence of cancers of the liver, lung, skin, bladder and kidneys (TGA 2001). Arsenic has also been identified as genotoxic and teratogenic.

Potential Effects of Termiticides on Non-Target Species

In most cases, termiticides are unlikely to cause significant environmental effects, providing that they are applied according to recommended application rates and methods and the recommended safety precautions are taken. Contamination of water surfaces and runoff should be avoided in all cases. Termiticides might present hazards to wildlife if they are absorbed or ingested. Unfortunately, as insecticides, termiticides are toxic to beneficial insects such as bees. The greatest hazards are posed to aquatic organisms through contamination of waterways via runoff or spills. Soil organisms, such as earthworms, which play a significant role in soil ecosystems are often exposed to termiticides, which may have lethal or sub-lethal effects depending on levels of exposure.

Current guidelines for Australian organisms are largely derived from overseas data, sometimes with correction factors applied to suit local conditions. However, the differences in persistence between soils in Australia and elsewhere indicate that experimental evidence needs to be gathered. There are significant gaps in knowledge regarding the long-term effects of termiticides in the Australian environment. Although considerable data have been accumulated for overseas species (see Table 1 for examples), these are not easily related to local conditions.

Chlorpyrifos is highly toxic to freshwater fish, aquatic invertebrates and estuarine and marine organisms. Because of its persistence in aquatic sediments, chlorpyrifos might represent a hazard to sediment feeders. Fish species are differentially affected by chlorpyrifos, with some species (e.g. *Gambusia*) surviving contaminations that killed several other species. Such differences in species susceptibility are likely to be the result of species differences in the sensitivity of brain acetylcholinesterase to inhibition by chlorpyrifos-oxon (Carr, Ho & Chambers 1997). The ecotoxicology of chlorpyrifos, including comprehensive LC_{50} data for many species, has been extensively reviewed by Barron and Woodburn (1995). Experimental LC_{50} and LD_{50} levels for some species are given in Table 1.

Aquatic invertebrates are susceptible to toxic effects should these pesticides enter waterways. Cladocera, *Ceriodaphnia dubia* (Richard 1894), exposed to binary and tertiary mixtures of chlorpyrifos, profenofos and endosulfan were increasingly impaired reproductively than when exposed to single pesticides (Woods, Kumar & Correl 2001). In a mixed Australian zooplanktonic community, chlorpyrifos at single, and multiple doses in laboratory tests, decreased cladoceran densities, but copepod densities increased commensurately, while phytoplankton density did not change in response to chlorpyrifos. Continuous, low doses of chlorpyrifos had no measurable effect (Simon & Helliwell 1997).

Sarneckis and Kumar (2001) tested two

commercial formulations of chlorpyrifos, Lorsban and a wettable powder, on embryos and tadpoles of *Littoria ewingii*, the brown tree frog, and *Xenopus laevis*, the South African clawed frog. Lorsban was found to be more toxic than the wettable powder, and *X. laevis* was more sensitive than L. ewingii to both formulations. Embryos exposed to these pesticides developed abnormalities varying from blistering, spinal curvature, reduced growth and oedema. The authors found that there was a dose-response relationship between AChE inhibition, malformations and chlorpyrifos exposure in tadpoles - 96h LC_{50}/EC_{50} corresponded with 80-90% AChE inhibition. Mixture toxicity tests using chlorpyrifos plus profenfos/or endosulfan resulted in synergistic or additive effects when tested on *X. laevis* (Woods & Kumar 2001). Such mixtures might occur when aquatic systems become polluted, and thus mixture toxicity tests reflect actual pollution of aquatic systems in a more realistic way than do experiments using individual toxicants (Woods & Kumar 2001).

Cholinesterase activity in earthworms, and the stability of the lysosomal membrane, are sensitive to organophosphate exposures (Booth, Heppelthwaite & O'Halloran 2000). For earthworms, chlorpyrifos in soils was toxic at LC_{50} of 104 to 1174 mg/kg of soil (Ma & Bodt 1993), however, there was wide variation in toxicity.

The pyrethroids are relatively non-toxic to birds, but toxic to mammals if swallowed. They are highly toxic to invertebrates, fish and aquatic organisms (Table 1). The pyrethroids are much more toxic to aquatic than terrestrial organisms because they inhibit ATPase enzymes, which are essential for maintaining the ionic concentration gradients required by cells for osmoregulation and the maintenance of ionic balances in an aquatic environment. Fish and other gilled animals are particularly at risk because of the large exposed surface areas gills provide. Bifenthrin has a high bioconcentration factor in bluegill sunfish (FMC Bifenthrin MSDS), Bifenthrin's Log Kow of >6, and its persistence might lead to exposure risks in apex species, particularly those that feed on sediment dwelling organisms. The octanol water partition coefficient (Log K_{ow}) describes the ability of a chemical to partition between an aqueous and a lipid phase. It is a measure of a chemical's potential for bioconcentration. A Log K_{ow} value of greater than 3 indicates a propensity for a chemical to bioaccumulate in fat. The pyrethroids are also extremely toxic to bees. Experimental LD_{50} levels for some species are given in Table 1.

Imidacloprid is highly toxic to some species of birds, but acute toxicity in birds varies widely between species. It has caused eggshell thinning, decreased weight and reduced egg production and hatching success in some bird species (Cox 2001). Imidacloprid is highly toxic to fish and other aquatic organisms, with LC_{50} levels for mysid shrimp of 37 ppb and behaviour effects noted in surviving shrimp (EPA evaluation data in Cox 2001). Imidacloprid is acutely toxic to earthworms, with LC_{50} levels of 2-4 ppm in soils for *Eisenia foetida* (Zang et al. 2000). Imidacloprid's ability to move through soil relatively easily poses concerns for potential contamination of waterways and groundwater. Imidacloprid is absorbed by plants from the soil through roots (van Iersel, Oetting & Hall 2000) and therefore there may be a risk of exposure to herbivores. Experimental LD_{50} levels for some species are given in Table 1.

Hexaflumuron has low levels of toxicity to mammals (oral LD_{50} in rats of >5000 mg/kg), but is toxic to aquatic animals (NPIC 1997) and to bees (TGA 2001). Fipronil is highly toxic to some game birds, fish and aquatic invertebrates, and moderately toxic to some waterfowl and mammals (Tables 1 and 2). The metabolites of fipronil are much more toxic. The metabolite MB461 is more toxic to birds, and the metabolites MB46136 and MB45950 are more toxic to freshwater invertebrates than fipronil itself (PAN,

2000).

Summary of toxicity

A considerable amount of toxicity data is available for termiticides, some of which is summarised in Tables 1 to 4. Table 1 shows the relative toxicity to terrestrial versus aquatic species: each of the commonly used termiticides is, not surprisingly, highly toxic to insects. Consequently, beneficial species such as honeybees are susceptible if they come into contact with these chemicals. Termiticides are also toxic to fish and aquatic invertebrates, and so they should not be allowed to enter waterways. All of the termiticides are considerably less toxic to birds and mammals than to fish and invertebrates.

The data in Table 2 demonstrate that mammals might be affected if they ingest or contact termiticides in large quantities or over an extended time. Chlorpyrifos causes cholinesterase inhibition in a number of mammals including humans, rats, mice and dogs exposed repeatedly. Bifenthrin and permethrin ingestion cause some changes in the livers of dogs and rats, and tumours. Weight loss has been reported in dogs fed fipronil in long-term trials.

Reproductive and teratogenic effects are summarised in Table 3. Very slight reproductive and teratogenic effects were seen in rats fed chlorpyrifos. Only at the higher doses were adverse effects seen in rats from a range of termiticides. Table 4 summarises the mutagenic and carcinogenic effects that were seen in rats fed high doses of Fipronil. An increase in tumours was recorded for rats fed high doses of bifenthrin over a long time period. In most cases, adverse affects in mammals occur only after exposure to large quantities of termiticides, or to small amounts repeatedly over a long time period.

Application of termiticides to the soil around houses risks exposure by humans and animals, or runoff and seepage to watercourses. The use of termiticides in accordance with manufacturers' specifications and following industry guidelines greatly minimises the possibility of exposure and risk to human and environmental health.

Incidents Associated with Termiticide Use in Southeast Queensland

Chlorpyrifos has been implicated in chemical related urban fish kills, as has bifenthrin. The increasing use of bifenthrin has resulted in an increasing number of incidents involving bifenthrin. Identifying the source of the chemical is often difficult. The following are incidents reported in the media during 2001 and 2002:

- January 2001: Swan Lake canal estate, Gold Coast. A pest control company driver washed spillage into a stormwater drain. Over 1000 fish were killed. Chlorpyrifos was subsequently identified at 100 times the lethal dose for fish in the canal water (Queensland Environmental Protection Agency press release February 2001).

- February 2001: Bifenthrin (0.125 mg/kg) was found in oysters from Redcliffe waterways (Keys & Mortimer 2001), at well above Food Standards Australia and New Zealand standards code for maximum residue levels (0.05 mg/kg). The source of this contamination is not known, but it is possibly as a result of runoff from nearby canal estate building sites.

- February 2001: Loders Creek, Southport. A fish kill was followed by tests revealing high concentrations of chlorpyrifos. The source of contamination is not known, but it was probably as a result of runoff from nearby canal estate building sites (Qld EPA press release February 2001).

- March 2001: Oxley Creek tributary, Brisbane. Chemicals leaked through storm drains to the creek in runoff following a fire at a nearby industrial area at Rocklea. Chemicals included insecticides and herbicides, requiring water to be pumped from the creek by the Qld EPA, to prevent contaminated water reaching Oxley Creek (Qld EPA press release March 2001).

- March 2002: Broadbeach Canal, Gold Coast. Fish kill thought to be due to the spraying of a new bridge with pesticide for termites. Chlorpyrifos was found in samples sent for analysis (Qld EPA press release No. 09/02).

- March 2002: Loders Creek, Southport. Fish kill thought to be as a result of Chlorpyrifos runoff from canal estate building sites (Qld EPA press release No. 07/02).

In addition, during 2001/2002 a number of complaints were received from Gold Coast residents about the taste and odour of tap water following termiticide treatments. Permeation through plastic water pipes by solvents is suspected as the cause. Analysis of water samples indicated the presence of volatile organics, C_3 and C_4 substituted benzenes and aromatic hydrocarbons. The termiticides chlorpyrifos and bifenthrin were identified but not quantified (Steven Begg pers. com.; Marshall & Begg 2002).

Contamination of local Brisbane waterways has also occurred. A reported decline in abundant species at the Boondall wetlands prompted an examination of local organisms for chemical contamination in 1995 (Mortimer, Shaw & Liess 1997). As well as evidence of contamination by heavy metals, in some sites bifenthrin and chlorpyrifos were found in sediments around Brisbane waterways. In the Redcliffe area north of Brisbane, oysters have previously tested positive for the presence of chlorpyrifos at every site examined, although the latest results were negative for bifenthrin (Keys, Mortimer & Webb 2002).

In 1999, consumer confidence in the pest control industry was shaken when more than 5000 new homes were allegedly treated with watered-down termiticides instead of the required treatment (*The Courier Mail*, 18 December, 1999). The Building Services Authority launched a prosecution against the company involved for improper termite control. In most cases, incidents involving termiticides have been as a result of inappropriate use and application, or of spills.

Biological Control and Alternative Treatments

The protected underground nature of termite colonies makes them poor candidates for biological control. A few parasitoids are known but they appear to have limited potential for controlling, preventing or treating termite populations. Viruses, bacteria, protozoa, nematodes, bacteria and most fungi have shown little promise in termite management (Culliney & Grace 2000). Biological control attempts using bacteria, fungus, *Metarhizium anisopliae*, and nematodes (*Heterorhabdis* spp. and *Steinernema* spp.) have had some limited success in experimental trials (Peters & Fitzgerald 1998).

Ant species have been proposed as possible biological control agents for termites under certain conditions. Kenne et al. (2000) proposed the use of the generalist ant *Myrmicaria opaciventris* (Emery) as a biological control agent for termites in sugarcane plantations. Semiochemicals from *Ochetellus* (previously *Iridomyrmex*) *glaber* (Mayr)worker ants strongly repel *C. formosanus* termite workers, and dichloromethane extracts taken from extracts of whole ants used to treat sand, resulted in a barrier that was not penetrated by termites (Cornelius & Grace 1994). It is possible that ant semiochemicals could provide a source of alternatives for future

barrier control. Monoterpenoids are potentially useful for the development of new insecticides because they have low mammal toxicity. Cornelius, Grace and Yates (1997) found that monoterpenoid alcohols, particularly eugenol, were effective as repellents against *C. formosanus*, and that termites would not tunnel through eugenol or geraniol treated sand barriers for five days.

The fungus *Metarhizium*, penetrates the termite cuticle causing mortality. *Metarhizium anisopliae* has been isolated from diseased nests of *R. flavipes* in Canada. Subsequent bioassays have shown that the fungus is spread rapidly to healthy termites through contact, and is able to cause mortality within 24 hours (Zoberi 1995). In laboratory experiments with *C. formosanus*, where strains of *Beauvaria bassiana* (Balsamo) and *M. anisopliae* were exposed to foraging termites, termites did not avoid the fungal baits. Exposure to *M. anisopliae* strains resulted in rapid termite mortality while the *B. bassiana* strain resulted in slower but increasing mortality (Delate, Grace & Tome 1995; Jones, Grace & Tamashiro 1996). *Cryptotermes brevis* is susceptible to *M. anisopliae*, with mortality four weeks after exposure at 93 to 100% (Nasr & Moein 1997). In Australia, some strains of *Metarhizium* have been identified by CSIRO as potentially able to control termites (Media release 99/128 1999).

It might be possible to infect forestry trees with fungi in order to control the termites that attack them. Suzuki et al. (1996) discuss field application of fungi, which resulted in no clear effects on the majority of trees (*Pinus luchuensis*). *Aspergillus niger* and *Paecilomyces fumoroseus* as well as *B. bassiana* and *M. anisopliae* show promise for control of termites (Suzuki et al. 1996). Entomopathogenic fungi hold promise as baits for termite management. Grooming and social interactions have potential to spread fungus infections throughout colonies. However, termites might avoid fungus conidia and remove and bury fungus-killed individuals, which along with defensive secretions might limit the spread of the fungus (Rath 2000). Direct application to nests has resulted in complete mortality, however, feeding sites and bait stations have not shown similar success in the treatment of buildings and timber structures (Rath 2000).

Several protozoicides have been investigated for use as baits. Waller (1996) investigated urea, ampicillin and tetracycline fed to *R. flavipes* and *R. viginica* (Banks). The most effective was urea, which was palatable and decreased termite survivorship by killing the symbiotic protozoa that help termites digest cellulose.

Alternative Treatments and Management Tools

Soil insecticide barriers have been the single most important means of controlling termites over the last 50 years, however, limitations of current soil termiticides and the popularity of slab-on-ground housing, along with increased awareness of potential environmental and health effects has led to a search for alternatives, which include baiting programs, spot dusting and a range of other options. If termite colonies are accessible, they can be directly dusted using arsenic trioxide or permethrin dust, or alternatively aggregated individuals can be dusted so that they return the poison dust to the colony. This is the favoured method of spot treatment of wooden railway bridges and isolated and obvious nests (Peter Langford, Queensland Rail pers. com.).

Termite resistant wood is used in some cases. For example, Black Cypress Pine - *Callitris endlicheri* (and its latex) is resistant to termite attack. It grows south of Kingaroy and west of the Great Dividing Range in Queensland. It is durable, and resistant to fungi, however, it is unpopular with builders because it is difficult to work with and is often knotty. Long-term use will depend on the timber being grown in commercial plantations. Some other Australian natives are described as "termite resistant", with AS3360.2 (2000) listing 37 eucalypt, 9 non-eucalypt and 5 softwood species as suitable

for minor construction purposes, such as fencing and landscaping.

Monitoring and baiting stations are used to detect termite activity. When it is detected, the monitoring station baits may be replaced with slow-acting poisons such as hexaflumuron. The principle of baiting techniques is to have a susceptible substance in an aggregation device (bait station) on which the termites aggregate and continue to feed once they have found the bait stations. Bait stations can be placed in in-ground and aboveground situations. A bait toxicant in timber or a cellulose matrix can be placed in the station, or dusting the visiting termites might indirectly destroy the colony.

In Australia the main problem with baiting against *Coptotermes* species has been the inconsistency of termites finding and accepting baits (Peters & Fitzgerald 1998). Baiting is most beneficial when used as part of an integrated pest-management strategy. Bait stations buried in the soil, might be found by foraging termites. Alternatively, above-ground bait stations are placed in direct contact with infested timbers, and aggregation stations have been used successfully when placed in the hollow centres of infested trees or stumps. The advantages of using baiting as part of an integrated pest-management strategy are that they do not lead to widespread contamination of the soil, and require only a small amount of toxicant as opposed to treatment systems, which employ a saturation approach. Baits can be used via a "bait and switch" technique in which the original non-toxic bait used to aggregate termites is replaced by toxic bait. Alternatively, the aggregated termites can be poisoned by topical application of a pesticide (arsenic dust which is then carried back to the nest) or the baits may be toxic or contain metabolic, growth, moult or chitin inhibitors. In all cases, the toxin must be slow acting to allow termites to feed and then move away from the bait and spread through the colony, as the presence of dead individuals can have a repellent effect.

Heat applied to infested surfaces has been used with some success. In laboratory studies, increasing wood core temperatures to 46°C and 49°C resulted in 100% mortality of C. brevis nymphs (Woodrow & Grace 1998). This was in small wooden blocks (13.5 x 13.5 cm). In larger wooden blocks where temperatures took longer to rise, termites were better able to tolerate the conditions, and it has been hypothesised that slow rates of thermal increase may lead to heat acclimation of termites. Cumulative effects of sublethal stresses due to gradual heating might increase mortality. Heat treatments can be used in whole-of building treatments by using a propane heating unit to blow hot air into the building which is sealed within a tent of tarpaulins. Hot air is blown in and around the building to heat interior and exterior walls and temperatures must be maintained at 45°C to 50°C for 50 minutes to an hour. This practice is rarely used. Freezing can be used for specific areas such as verandas and/or furniture. Liquid nitrogen is pumped into the sealed area to cool the area to -20°C. This is a method that is obviously not practical for infestations of buildings or large areas.

Electricity has been used to spot-treat infested wood using an electrogun to deliver a low current and high voltage, which kills the termites in their path, and heat generated by microwaves can be used to kill termites. Microwave generators are mounted against the infested wall. Microwaves can also be used to treat wood as a preventative measure. By passing wood through microwaves a change in structure might result. The palatability of the wood to termites can be changed. The addition of pesticides and/or preservatives in the early stages of the procedure is possible.

Techniques developed for tracing the movements of subterranean termites by feeding them radioisotopes could potentially be used to exterminate colonies. Isotopes fed to termites via insertion of a wooden dowel

with an agar mixture of inert radioisotopes La-140 or Sc-45 were spread through the colony over 24 hours (Airey & Charlton 1994), thus demonstrating the effectiveness of such a method. This technique obviously poses technical and safety issues that render it unsuitable for general use.

Carbon dioxide might be a viable alternative to conventional pesticides in some situations, such as vault fumigation, and possibly for eradication of pests in structures. Significant mortality has been recorded for *C. formosanus* exposed to 95% or greater carbon dioxide atmospheres, however, 60 hours of exposure were required to produce 100% mortality (Delate, Grace & Tome 1995).

Sand, (12-grit) layered around the foundations of a house may act as a barrier against termites (Ebling & Forbes 1988). Basalt barriers with 50% or more of the particles with diameters of 1.7-2.8mm was not penetrated by formosan subterranean termites in laboratory and field trials (Tamashiro et al. 1991). Similarly, granite barriers were effective for some species. Verkerk (1990), states that gravel barriers are an effective barrier against termites. A double barrier system, with a plastic membrane separating two different size gravels would have to be used North of the Tropic of Capricorn due to the presence of *M. darwiniensis*. Deltamethrin impregnated blankets, stainless steel mesh, or aluminium termite barriers, flashing and damp-poof coursing can provide effective barriers against infestation, but any cracks, deformities or degeneration in these materials might allow access.

For subterranean cables, a number of barriers are used, or have been investigated, to prevent termite damage. Welded stainless steel tubes are used for high voltage cables as an effective barrier. The stainless steel tubes are corrugated so that they are able to flex and bend to some degree without cracking. Power and utility cables can be taped with stainless steel, brass or copper tapes, overlapping to form a seal that is impervious to termites; these cables are then usually finished with a PVC coating for extra protection. High Density Polyethylene (HDPE) cables are resistant to attack because they are too hard to penetrate, are smooth and therefore difficult for termites to grasp with their mandibles (Olex cables technical informative PT-I 107-5, 2000). Nylon can also be an effective termite barrier; used extensively to protect telecommunication cables. As with HDPE, the effectiveness of nylon is as a result of its smooth impermeable surface (Olex cables technical informative PT-I 107-5, 2000).

Future Directions

The future of termite management lies within an integrated approach that is able to take into account preventative and retrospective treatments for termites that is not only effective, but is also economical, safe, and environmentally friendly. Such treatments are likely to consist of physical and chemical barriers, used in combination with resistant or preserved wood or steel framing, and according to the requirements of individual sites.

For an integrated approach to be effective it must involve industry, government and consumer groups, and combine treatment and prevention programs with education, monitoring and collating data, and research into new and better measures for termite management. There is a widely perceived need for improvements in building design in order to prevent or reduce termite infestation along with increased interest and demand for physical barrier systems. New building techniques and designs, along with novel barrier systems to reduce exposure to termites are warranted, and further research on termite foraging behaviour is required to facilitate the development of more effective bait and monitoring technology.

Currently in Australia, we may be enjoying a period of overlap where older buildings are still being protected by the organochlorines while new structures are treated with organophosphates. Houses built

before 1995 may still be protected to some extent by the organochlorine termiticides. However, their effectiveness will have been reducing over time, and older houses might be at risk of termite infestation. The potential costs of re-treatment and repairs are likely to increase in the future as older slab-on-ground dwellings become susceptible due to the eventual failure of the organochlorine termiticides.

The success of the organochlorines over long periods of time has added to the popularity of slab-on-ground housing in Australia; buildings which are much more susceptible to infestation by termites than older style above-ground housing. This means that the potential for termite damage is increased. The result of Australia's previous reliance on the persistent organochlorines is that modern housing is susceptible to attack by termites, with termites entering buildings through weepholes, expansion joints, service ducts and cracks in brickwork which often extend several courses below ground. Simple alterations in building and property maintenance practices, such as providing exposed slab edges above ground and extending aprons of slab around slab-on-ground dwellings will allow termite incursions to be reduced and detected, providing that regular inspections take place.

The efficiency of different pesticide formulations needs investigating. Therein lies the potential for chemical companies to examine their formulations to suit specialised purposes. Different management techniques can be investigated, the electricity supply industry being an example. In Australia, there are approximately eight million wooden power-poles, which are inspected (typically at five-year intervals) for termite infestation and rot. These poles exist in a wide range of conditions, in urban and rural environments, in a range of soil types and climates, some adjacent to waterways or drainage areas. However, despite substantial differences, all are treated by similar processes, using commercial formulations where termiticides are applied. Biflex (bifenthrin) is most commonly used on wooden poles today. Previous treatment regimes included chemicals such as aldrin, creosote and chlorpyrifos treatments. Many wooden poles are fluoride treated to prevent rot and most wooden poles are now CCA pre-treated. Issues with current treatment include concerns about the uncertain potential of the "chemical cocktail" of past treatments that might surround power-poles.

In addition, there are 54.5 km of timber railway bridges in Queensland alone and approximately 9,500 km of rail corridor. Each bridge is susceptible to termite attack and undergoes inspection every four to eight years. Arsenic dusting into nests and application of chlorpyrifos or bifenthrin to soil is applied on discovery of termite activity. Fipronil and imidacloprid are sometimes used. In the last 10 years, 40 km of timber bridges have been replaced with concrete structures, particularly in tropical regions. However, many timber bridges remain. Construction of concrete footing to keep timber supports from contacting the ground is being considered to reduce termite damage.

There is potential for clients to work with termite managers and chemical companies to review their formulations to suit specialised purposes, identify termite management strategies appropriate to the location of infrastructure, and to develop chemical-free physical barriers and/or more sustainable termite management practices. Future approaches based on integrated pest management principles require the cooperation of stakeholders in order to gather information and perform comprehensive research.

Several requirements are apparent. More information is required on Australian termite biology, taxonomy and ecology. Ideally, an understanding of the way in which termites forage, how they locate food sources, what specifically attracts and repels them, and an understanding of the mode and speed of infestations needs to be gained. The risks of termite infestation need to be evaluated, both locally and nationally so

that susceptible or high risk areas, structures and building types can be identified and preventive measures taken in terms of design and construction. Building regulations and designs need to be able to reduce or eliminate high-risk housing and conditions that are attractive to termites and/or facilitate termite infestation.

Further education is required for homeowners, builders, designers, legislators and landscape designers so that they can reduce the risk of infestation through the avoidance of practices in landscaping and design that inadvertently favour termites. This would mean ensuring under-floor ventilation, which discourages termite activity, not stacking timber or building up soil against buildings, reducing timber use where inspection for termites is difficult, and not building wooden in-ground structures (e.g. untreated timber retaining walls) close to houses.

There needs to be a specific focus on the creation of alternative barriers for the range of wooden structures that need protection. The focus needs to take account of the current limitations of physical barriers and monitoring stations, which may be avoided by termites and to overcome the loss of activity associated with short-term response chemicals, which have replaced the more hazardous organochlorine compounds. An innovative approach to barrier design is needed that seeks to incorporate the specific features of slow release chemicals, based on natural products which are more acceptable to the environment and public health.

Since most termite-related damage to timber occurs from subterranean termites, preventative measures rely heavily on site housekeeping and the establishment of physical or chemical barriers to stop termites getting into the premises or timber from the underlying soil. Once termites have been found in wooden structures, a range of physical and chemical techniques are available to treat and eliminate (or control) the infestation. There is an obvious need for a reliable long term, maintenance free method of preventing termite infestations that poses little or no risk to human and environmental health. Research is required into the behaviour of termiticides, current and potential, in a wide range of Australian soils and under Australian conditions using rigorous, repeatable science, and open and collaborative communication between stakeholders needs to be maintained so that the concerns of each group are addressed.

Acknowledgments

The authors wish to thank the following people for their contributions, assistance and criticisms: John Cheadle (retired, formerly of Arrest-a-Pest), John Field (formerly of Amalgamated Pest Control), Shaun Hale (Amalgamated Pest Control), Michael Ball (Queensland Department of Public Works), Roland Cruice and Bill Wiersma (Queensland Department of Public Works and Housing), Peter Dobson and John Martinkovic (Brisbane City Council), Brenton Peters and John Fitzgerald (DPI Forestry), John French (University of the Sunshine Coast), John Mott (The University of Queensland), Ian Marshall and Steven Begg (Queensland Health).

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Environment Improvement Plans: The Way of the Future Report on an Assessment of Two Industrial Site-specific Environment Improvement Plans (EIPs) and an Assessment of the Application of EIPs within a Local Neighbourhood Context

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This paper describes an assessment of two site-specific Environment Improvement Plans (EIPs) significant to the residents of the City of Hobsons Bay, an inner western municipality of Melbourne comprising the suburbs of Spotswood, Newport, Williamstown, Altona, Brooklyn, Seabrooke and Laverton. The Environment Protection Authority (EPA) initiated environment Improvement Plans (EIPs) in 1989. There is limited available published material associated with the workings of the EIP development process. The project explored the initial experiences of participants in the development of an EIP to identify key issues critical for the success of the process. Some of the important functional issues covered included logistics, committee support, representation, trust and group dynamics. Success may depend upon how the key participants feel about the process. The two EIPs, which formed the basis of the project, were the Peerless Holdings P/L (Pridham) EIP located at Merino St, Laverton, Victoria, and the Mobil Refinery EIP located at Altona, Victoria. Overall, the project showed that the EIP process is a successful tool for industry to use in minimising environmental pollution. Prior to the implementation of EIPs both sites were experiencing significant environmental pollution problems. These reduced dramatically after the introduction of an EIP. The project confirmed that complacency from all stakeholders, and in particular industry, looms as the largest threat to undermining the success of the EIP process. The project has revealed that local residents need to be better represented and supported and that strategies need to be developed to increase community interest in the process. The project also assessed the application of EIPs within a local neighbourhood context, which confirmed that the issues around developing a proactive approach require further investigation.

Key words: *Industry Environment Improvement; Environment Improvement Plan (EIP)*

The City of Hobsons Bay supported the research into the assessment of the Environment Improvement Plan (EIP) process as part of Council's Public Health Coordinator completing the Sir Gustav Nossal International Fellowship Leadership in Health Reform Program. The goal of the program was to build capacity for equitable health reform.

The Environment Protection Authority (EPA) initiated environment Improvement Plans (EIPs) in 1989. This innovative concept is unique to Victoria, Australia. There is limited available published material associated with the workings of the EIP development process. This project explored the initial experiences of participants in the development of an EIP to identify key issues critical for the success of the process. Some of the important functional issues covered include logistics, committee support, representation, trust and group dynamics. There is limited evidence as to the effectiveness of EIPs and their success may

depend upon how the key stakeholders feel about the process. The project also assessed the application of EIPs within a local neighbourhood context.

The project focused upon two industrial site-specific EIPs: Peerless Holdings P/L (Pridham) located at Merino St, Laverton, Victoria, and the Mobil Refinery site located at Altona, Victoria, which were significant to the residents of the City of Hobsons Bay, an inner western municipality of Melbourne, comprised of the suburbs of Spotswood, Newport, Williamstown, Altona, Brooklyn, Seabrooke, and Laverton. The objectives of the project were to:

- Examine the mechanisms, workings and group dynamics associated with the development of an EIP
- Explore the initial experiences of stakeholders in order to identify key issues critical for the success of an EIP
- Assess the knowledge gained from the EIP process for potential use in the development of the Neighbourhood EIP concept within a local government context.

Site Specific Environment Improvement Plans

The EIP program was described by the author Unglik as a "radical initiative", which was adopted with "extreme trepidation" by EPA senior management (Unglik 1996, p. 86). The Chairman of the EPA at the time persuaded a senior executive of Mobil Australia to develop such a plan for a Petrochemical Refinery located at Altona, Victoria.

The EPA's Information Bulletin (EPA 1993) describes an EIP as a public commitment by a company to enhance its environmental performance. The plan outlines areas in which a company can improve its site-specific operations and is usually negotiated in conjunction with the local residents, local government, EPA and other relevant government authorities. Normally an EIP will contain clear timelines for completion of improvements (action plans) and details about ongoing monitoring of the plan. Improvements may include new works or equipment or changes in operating process. They may also include organisational cultural change.

The EIP process is about bringing the local residents, industry, local government and the EPA together to develop a plan for the benefit of all concerned through improving a particular site's environmental performance. This may include a range of measures to reduce air, noise or ground water pollution issues.

Garbutt has described the EIP process as being based on the concept of the local community's "right to know" and to participate in decisions that may potentially have an impact on their environment (Garbutt 2000). It should not be assumed that the community members participating in the process represent the wider community and it is important to recognise that a truly representative group is unlikely. Meek described it as very important that the wider community is regularly informed of progress, thus providing opportunity for input (Meek 1996).

A company may voluntarily initiate an EIP or the EPA can direct a company to develop such a plan. There are approximately 55 EIPs currently operating in Victoria and the EPA has directed only two of these plans. The level of voluntary action is a strong measure of the success of EIP's from the perspective of both industry and the broader community (Garbutt 2000).

The development of an EIP can benefit the local community and industry in many ways, especially through a greater understanding of the way industry operates and the subsequent environmental impact. It can give people in affected communities feelings of being involved in, and having a

degree of control over, decisions about industrial sites directly impacting upon their local neighbourhood (Wills & Fritschy 2000). The EPA's Information Bulletin (EPA 1993) outlines how the EPA benefits through responsible industry with open reporting and its own well developed monitoring program, which allows the EPA to concentrate on poor performers. Wills and Fritschy describe the EPA's need to balance the benefits of local industry and community participation in the EIP process against its own responsibilities for enforcing the law in the interests of the general community (Wills & Fritschy 2000).

The development of an EIP needs to be considered as a dynamic process with ongoing review and continuous improvement.

Method

A Reference Group was established to oversee and provide guidance for the project. A mentor from the Sir Gustav Nossal International Leadership in Health Reform Program provided valuable input. A process for the investigation and exploration of issues associated with the development of site specific EIPs was established through semi-structured telephone interviews of local resident advocates, industry representatives, and Environment Protection Authority (EPA) representatives. Questions focused on the EIP development process. Some targeted questions as to "how the EIP has made a difference" were also included.

Findings

The project results indicate that the following factors are critical to the success of the EIP process:

- Commitment by the company to solve problems
- Good communication between the company and local residents
- Willingness by local residents and the company to listen to opinions
- Preparedness to compromise from both sides
- Readiness of the company to finance environmental improvement actions
- Local residents' understanding of company needs and budget constraints
- EPA threat of legal action
- Trust between participants
- Local government involvement
- Recognition by industry of the benefits that might result from the EIP process

An essential ingredient is that local resident advocates participating in the development of an EIP must provide effective and assertive input into the process. The key to the success of the process from the viewpoint of the local residents interviewed is the threat of legal action from the controlling authority (EPA). The research showed that it is the combination of voluntary participation in such a process, combined with the ever-present threat of legal action for offences in connection with poor environmental performance that yields success.

The results also highlight important resident participation recruitment issues. Recommendations for further studies of this nature include addressing issues such as:

- Developing strategies to attract and generate community interest in order to increase and broaden community participation
- Investigating the possibility of local resident advocates

receiving an honorarium for participation in the process.

- Developing a training package for local resident advocates
- Improving community awareness about general environmental policy
- Developing guidelines for local resident EIP participation
- Developing strategies for communicating with local residents about their views (especially residents that have not complained) of the success or failure of the EIP process.

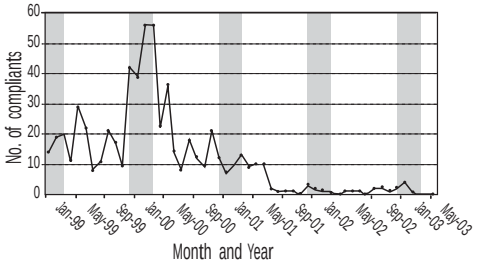
The results suggest that roles and responsibilities for key stakeholders need to be clearly defined. The results also highlight the importance of appointing an independent and neutral chairperson and minute taker.

The project confirms that complacency from all stakeholders, and in particular industry, looms as the largest threat to undermining the success of the EIP process. Strategies are required to ensure that companies undergo continuous improvement.

The results of the project highlight that the EIP process must focus on dialogue and not necessarily on agreement. Most of the interviewed participants were satisfied with the process. Environment Improvement Plans are about groups of people often with divergent views coming together to resolve a common problem.

The results suggest that an EIP will lead a company on the path to achieving increased economic performance, environmental pollution reduction, and improved corporate social responsibility. This assists a company towards reaching the goal of sustainable development. Figure 1 illustrates the reduction in the number of environmental odour complaints received by the EPA from 1999-2002 for Pridham.

Monthly odour complaints against Pridham received by EPA (1999-2003)



Data provided by Environment Protection Authority

Implications for Further Action

There are opportunities for Councils to undertake a number of actions. These actions are:

- Actively participate in the development of local EIPs
- Review statistical data to determine the level of resident concern in relation to environmental pollution from local industry and actively promote EIPs to identified businesses
- Nominate an appropriate Council Officer
- Develop appropriate policies and procedures in relation to site specific EIPs and Neighbourhood EIPs support for local resident advocates on key issues
- Provide knowledge and expertise to EIP committees
- Celebrate the achievements of successful EIPs
- Recognise and award outstanding local resident advocates

Neighbourhood Environment Improvement Plans

The *Environment Protection Act 1970* was amended in May 2001 to include the

Environment Protection (Liveable Neighbourhoods) Act. This legislation introduced the concept of Neighbourhood EIPs, which build upon the success of site-specific environment improvement plans, such as those of Pridham and Mobil. Plans for these sites have all been successful in reducing environmental pollution.

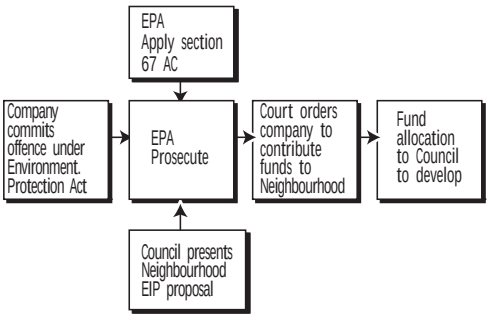
Neighbourhood EIPs provide a statutory mechanism for those affected by pollution in their local area to come together in a constructive forum. Residents, industry and the local council can agree on the environmental priority issues for a particular neighbourhood.

The findings indicate that the establishment of focus groups to identify neighbourhood problems and potential neighbourhood sites will be useful. However, results indicate that the implementation of Neighbourhood EIPs will be more difficult to achieve because a number of pollution sources might be involved and the sense of ownership would be reduced. Conversely, local residents might feel a greater sense of identity with a local neighbourhood issue and therefore might be more willing to participate. Increased community awareness about the EIP process might also overcome this problem.

The project leads to the conclusion that the critical success factors identified for site specific EIPs are also relevant to Neighbourhood EIPs. Inevitably, local councils become the major drivers of Neighbourhood EIPs. It is therefore important that councils are fully aware of the implications of their involvement in NEIPs. Appropriate funds will need to be allocated to resource the development and implementation of NEIPs and a potential source lies with the EPA in directing revenue from fines for environment protection breaches to local council Neighbourhood EIP initiatives.

Under section 67AC of the Environment Protection Act DATE a court may order an offender to take specified actions, which include "to carry out a specified project for the restoration or enhancement of the environment in a public place or for the public benefit (even if the project is unrelated to the offence)". The EPA has advised that if an offence occurs in a particular municipality then it may advocate in court for this section to be enacted and for revenue from fines for environment protection breaches to be forwarded to a local council for the development of a Neighbourhood EIP. Revenue from this source may be utilised for purposes other than the development of EIPs. To take advantage of this potential source of revenue Councils will need to have identified potential Neighbourhood EIPs and have documented proposal/s for presentation in court.

Figure 2: Funding source for Neighbourhood EIPs



Conclusion

Overall, the project indicates that the EIP process is a successful tool for industry to use in minimising environmental pollution around a site. Prior to the implementation of EIPs both sites were experiencing significant environmental pollution problems. These reduced dramatically after the introduction of an EIP. The project has revealed that local residents need to be better represented and supported and that strategies need to be developed to increase community interest in the process.

Acknowledgments

The following are thanked for their support and assistance in the undertaking of this project: Hobsons Bay City Council, Members of the Reference Group that provided guidance for the project, which included, Simon Barraclough, La Trobe University, Toni Meek, Environment Protection Authority (EPA), Nessie Hardy, Community Representative, Michael O'Hanlon, Hobsons Bay City Council, Robert Brunton, Australian Institute of Environmental Health, Margaret de Bono, Statistical compilation, and the various industry, community and EPA representatives interviewed.

Endnote

Mr Bruckner presented a paper on Environment Improvement Plans to the 2003, Victorian State Conference of the Australian Institute of Environmental Health.

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Legislation

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An Intervention Trial on Short Term Exposure to Inorganic Arsenic in Drinking Water

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There is still considerable debate surrounding the proposed reduction of current drinking water guidelines for arsenic to 10µL and how low is low enough. One of the main issues is the estimation of potential health impacts of low doses of arsenic and the ability to assess exposure at these lower arsenic levels in drinking water. There has been little investigation of whether reducing the concentration of arsenic in drinking water at low arsenic concentrations in drinking water (greater than the current WHO drinking water guideline of 10µg/L, but less than 50µg/L), to levels below the guidelines, is associated with a significant reduction in urinary arsenic concentrations. We investigated the effect of removing a contaminated drinking water source and replacing it with uncontaminated bottled water on the urinary inorganic arsenic concentrations of 23 exposed individuals. The average drinking water arsenic concentration was 17.5µg/L (range 2.7-27.0µg/L). The results of this study showed that changing from usual drinking water to bottled water decreased urinary inorganic arsenic concentrations, but not significantly. The trial had low power and a larger sample size would be required in addition to more information on other routes of exposure. The results of this small study suggest that factors other than drinking water arsenic concentrations may play an important role in short term exposure to drinking water with below 20µg/L inorganic arsenic.

Key words: *Arsenic, Drinking Water, Exposure, Intervention, Urine*

Many countries have drinking water concentrations above 50µg/L (Frey et al. 1998; Frumkin & Thun 2001; Reid 1994; USEPA 2000). Recently, the United States Environmental Protection Agency (USEPA) revised its arsenic enforceable maximum concentration for arsenic to 10µg/L with a Maximum contaminant goal of zero (USEPA 2001). The current WHO drinking water guidelines is 10µg/L (WHO 1996). Because of the large number of communities in many countries with drinking water arsenic concentrations in excess of 10µg/L, the cost implications of such proposed reductions are high. There has been limited investigation of whether reducing arsenic in drinking water from relatively low concentrations to levels below proposed guideline levels, reduces body burden. Elevated urinary arsenic concentrations have been found in participants consuming water with >100µg/L arsenic (Hopenhayn-Rich et al. 1995; Hopenhayn-Rich et al. 1996; Kurttio

et al. 1998; Valentine et al. 1979). There are limited data available to indicate whether urinary arsenic concentrations are elevated following consumption of drinking water with arsenic at or slightly above current guideline values, compared with consumption of uncontaminated drinking water. Further, there are other sources of arsenic, which may contribute to urinary arsenic concentrations such as contaminated soil, dust, food and cigarette/tobacco smoke. These factors are likely to become more important to overall exposure at lower drinking water arsenic concentrations (Kalman et al. 1990; Polissar et al. 1990; Vahter & Lind 1986).

To investigate the contribution of lower arsenic concentrations in drinking water to urinary inorganic arsenic concentrations, an intervention trial was conducted to replace contaminated drinking water as a source of arsenic exposure and to determine the effect of this intervention on urinary inorganic arsenic concentrations after a washout period. When ingested, inorganic arsenic is methylated to monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA) (Apostoli et al. 1997). Consumption of seafood may also contribute to DMA concentration via methylation of organic arsenic (Buchet et al. 1996). Inorganic arsenic can be detected in the body several hours after ingestion and concentrations peak at approximately ten hours, where MMA and DMA predominate some 24 hours later and may be present for some time (Apostoli et al. 1997). In this study, with the focus on exposure, inorganic arsenic in urine was used as the outcome variable to reduce the possible influence of diet on the measure.

Materials and Methods

A cross-over intervention trial was conducted in a small subgroup of a study population recruited for a previous study of predictors of short and long term measures of arsenic exposure. To be eligible, residents needed to consume drinking water from the tap with measured concentrations of inorganic arsenic >10µg/L. A small rural town was selected as the study area due to the usually consistently high concentrations of arsenic in the water supply (average 45µg/L) and low arsenic concentrations in soil (Hinwood et al. 1998, 2002). In this intervention trial, the drinking water arsenic concentration was approximately half previously measured levels. The population of the town was 219 in 1996 (Australian Bureau of Statistics 1996).

A sample size calculation was conducted and was based on the results of a prior study of urinary inorganic arsenic concentrations where an average difference of 2.0 µg/L + 2.31 µg/L arsenic in urine was observed between residents consuming contaminated water and a control population (Hinwood et al. 2002). Twenty-eight individuals were required, to achieve 90% power to detect a change of 2.0µg/L as significant at the 5% level. Each person was to be their own control, which was expected to reduce the expected standard deviation to 1.4µg/L.

Participants were recruited by telephone. Telephone calls were made during the day and when there was no answer, calling was done in the evening. Advertisements were also placed in local papers in an attempt to recruit additional participants. All participants were given an information sheet outlining the purpose of the study and study requirements.

Households were randomly assigned to the intervention groups using the random number generator function in SPSS for Windows (SPSS 1997). Households were used rather than individual participants due to the potential difficulties of different family members in the same house having to use different water sources at the same time.

A three-week data collection schedule was designed to enable measurement of the effect of the intervention on urinary inorganic arsenic concentrations as well as determining the "background" variability in urinary inorganic arsenic concentrations when consuming the usual drinking water

supply. First morning void urine samples were collected in two weeks where usual tap water was consumed and a week where bottled water was consumed. The crossover allowed for identification of any potential "carryover" effect in either direction.

The washout period selected was 5 days and participants were requested to consume bottled water in the 5 days prior to the bottled water consumption week and subsequent collection of samples. Approximately 65% of the absorbed dose of inorganic arsenic may be excreted in urine and 30% of the dose is excreted within 24-48 hours, with the remaining 35% excreted within a week (Apostoli et al. 1997; Moyer 1995; USEPA 1994).

Participants in one group started the study consuming tap water for the first week of sampling, followed by the washout period with bottled water, bottled water consumption for the next week, then tap water for the third week. In the three weeks, samples were taken on the same days each week. For the second group the sequence of the intervention was tap water for the first week, tap water for the second week, a washout period, then use of bottled water for the third week. A third group was introduced due to participant availability and logistical problems with a sequence of a washout period followed by bottled water consumption for the first week and tap water for the second and third weeks.

Data collection

Participants were asked to provide two spot urine samples over the same two consecutive days during each week of the trial. For each participant, the first morning void sample was collected in a 1L new polyethylene bottle. Participants were advised to empty their bladder into the 1L bottle first thing in the morning and record the time. Each first morning void urine sample was sub-sampled by placing four 50-60mL aliquots into new 60mL polycarbonate containers and sample details were recorded in a sample log with a chain of custody form. All samples were frozen within 12 hours and transported to the analytical laboratory for storage and analysis.

Participants were requested to document their fluid (including drinking water and alcohol), smoking, and fish intake using an intake diary during each 2-day collection. A brief questionnaire asking for details about residence, age and gender was administered. Participants were also asked to use the bottled water for drinking tea and coffee, preparation of food and drinks.

Drinking water samples were provided for each household every week in 500mL acid washed polyethylene containers. Eight bottled water samples were sent for analysis to ensure the absence of arsenic in the intervention drinking water.

Chemical analysis

Samples were analysed for inorganic arsenic using continuous flow sodium borohydride generation of arsine on a hydride generator and detection using an Atomic Absorption Spectrophotometer "GBC Scientific 906". The method measured inorganic arsenic with a detection limit of 2µg/L for urine samples and 1µg/L for water samples. The difference in detection limits was due to the differences between urine and water matrices.

Every assay included quality controls to determine assay performance (Hinwood et al. 2002). The precision and accuracy of the inorganic arsenic method for this study was acceptable with a coefficient of variation of 14 and 16% for the low quality control and high quality control samples respectively, based on the accepted coefficient of 20% specified in the Australian standard method (Standards Australia 1987).

Statistical analysis

The data were highly censored with 32% of the data below the analytical limit of detection. Non-detects were assigned a concentration of 1µg/L (half the analytical limit of detection) (Liu et al. 1997). Urinary inorganic arsenic concentrations were log

transformed for most analyses. Correlations were performed on untransformed data. The non-parametric Kruskal Wallis test was performed to test for differences between groups. Non-parametric Wilcoxon test was used for paired analysis. A linear regression random effects model was performed on log transformed data using STATA (StataCorp 1995).

Results

The demographic characteristics of the intervention groups were investigated. Intervention Group 3 with only 4 participants was different from the other groups with a higher mean age and no men in the group (Table 1). Recorded water intake for each group was similar. Group 2 had a higher number of smokers and Group 1 had a higher alcohol intake. Information on occupations, and activities such as pesticide use were collected, however, no participants recorded occupations or pesticide use that might increase exposure to arsenic.

Table 1: Demographic characteristics of the population

		All	Group 1	Group 2	Group 3
n		23	8	11	4
Age (years)	Mean	46	44.5	45.7	50.5
	Range	5-68	5-68	16-65	34-60
Sex (%)	Male	34.8	37.7	45.5	0
Recorded Water Intake glasses*/day					
	Median	5.6	6.5	4.8	6.0
	Range	1.2-11.1	1.1-8.4	2.3-11.1	4.3-11.0
Current Smoker (%)		30.0	25	36.4	25
Recorded Alcohol Intake glasses*/day					
	Median	0.1	0.1	0	0
	Range	0-7.7	0-7.7	0 - 1.3	0 - 1.4

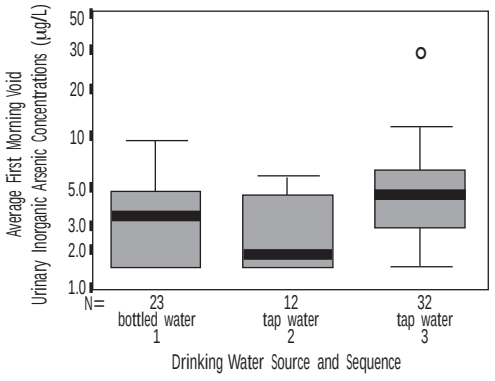
** 250mL*

The geometric means and ranges for averaged first morning void samples (the average of the first morning voids were taken for non-intervention and intervention time periods) were lower when participants consumed bottled water rather than tap water (Table 2). The differences were not statistically significant.

It was expected that the difference between urinary inorganic arsenic concentrations when participants were consuming bottled water compared with tap water, would be greater than the difference between the two time periods where tap water was consumed. This was not observed and a paired analysis, which tested the mean difference in urinary arsenic concentrations with corresponding water arsenic concentrations showed no statistically significant difference indicating the intervention had no significant effect.

To investigate these results further, the sequence of administering the intervention was examined for its influence on urinary inorganic arsenic concentrations. Figure 1 shows the influence of sequence on urinary arsenic concentrations using a box plot and whiskers presentation. It is clear from the figure that median urinary inorganic arsenic concentrations were higher when participants consumed tap water. When participants consumed bottled water following a week of tap water and despite a 5-day washout period, the median was lower,

Figure 1: Urinary inorganic arsenic concentrations for each study sequence of consumption of bottled water and tap water



Legend:

1 Samples taken while participants drinking bottled water following 5day washout with bottled water after consuming tap water

2 Samples taken while participants drinking tap water following consumption of bottled water

3 Samples taken while participants drinking tap water after consuming tap water for previous week.

Table 2: Urinary inorganic arsenic concentrations for individual and averaged first morning void urine samples with and without intervention

Intervention Group	Tap Water Urinary Arsenic (μg/L)	Water Arsenic Concentration (μg/L)	Intervention Urinary Arsenic (μg/L)	Intervention Water Arsenic (μg/L)
Spot 1	GM (Range)	GM (Range)	GM (Range)	
All	2.89 (<DL-27.8) (*n*=46)	17.46 (2.68 - 27.0)	2.44 (<DL-15.5) (*n*=21)	<1
1	3.25 (<DL-27.8) (*n*=16)	19.39 (14.8- 26.7)	2.34 (<DL-7.0) (*n*=7)	<1
2	2.75 (<DL-13.2) (*n*=23)	15.49 (2.7 - 23.4)	2.59 (<DL-15.5) (*n*=?)	<1
3	2.69 (<DL-18.3) (*n*=7)	19.96 (13.9 - 27.0)	2.32 (<DL-6.2) (*n*=4)	<1
Spot 2				
All	2.80 (<DL-34.6)	17.46 (2.68 - 27.0)	2.23 (<DL-9.2) (*n*=22)	<1
1	3.25 (<DL- 34.6)(*n*=16)	19.39 (14.8- 26.7)	2.64 (<DL-9.2) (*n*=8)	<1
2	2.44 (<DL- 16.6)(*n*=22)	15.49 (2.7 - 23.4)	2.39 (<DL-7.2) (*n*=10)	<1
3	2.94 (<DL-12.9) (*n*=8)	20.29 (13.9 - 27.0)	1.65 (<DL-3.4) (*n*=4)	<1
Averaged Data				
All	3.25 (<DL-31.20)(*n*=46)	17.46 (2.68 - 27.0)	2.25 (<DL-30.97) (*n*= 22)	<1
1	3.42 (<DL-31.20)(*n*=15)	19.11 (14.8- 26.7)	2.56 (<DL-6.0) (*n*=8)	<1
2	2.97 (<DL-12.7) (*n*=22)	15.49 (2.7 - 23.4)	3.32 (<DL-11.7) (*n*=10)	<1
3	3.67 (<DL-12.9) (*n*=8)	20.28 (13.9 - 27.0)	2.20 (<DL-4.05)(*n*=4)	<1

but not significantly. The lowest median urinary inorganic arsenic concentration was recorded for participants consuming tap water following bottled water consumption.

These results indicate that sequence is very important to the intervention and is suggestive of a carryover effect. The range of inorganic urinary arsenic concentrations is similar irrespective of whether bottled water or tap water was consumed (Figure 1). A range of factors collected from questionnaire was examined for their potential influence on the results of the intervention trial using a random effects regression model. Factors such as water intake, fluid intake including alcohol, fish intake, smoking (Y/N) had no influence in inorganic urinary arsenic concentrations.

The intervention alone (Table 3) only explained 6.2% of the urinary inorganic arsenic concentrations recorded. When other factors such as drinking water arsenic concentrations, the intervention, sequence of intervention, age, sex and intervention group, were included in the random effects regression analysis model to explore their effects, these factors could explain 31% of the variation. Sequence and age were shown to be more significant factors (Table 3).

Information on other factors such as the use of tap water for washing, bathing and cooking was also collected. All participants recorded using tap water for washing, bathing and cooking for 100% of the time, which may have introduced further sources of arsenic outside water consumption. It was not possible to determine the relative contribution to exposure from these sources.

Table 3: Random effects linear regression on averaged spot urinary arsenic concentrations

Model Number	Model		Regression coefficient	95%CI	Chi ^{2}	Overall R^2
1	Intervention (Y/N)		0.33**	0.01, 0.65	4.2	6.2
2	Intervention (Y/N)		0.46	-0.19, 1.1	25.5	31.3
	Intervention Group	1	0.10	-0.80, 0.29		
		2	-0.43	-0.66, 0.74		
	Drinking Water		-0.41	-0.26, 0.18		
	Age		0.009**	0.0002, 0.02		
	Sex		0.20	-0.12, 0.52		
	Fluid Intake		0.03	-0.02, 0.09		
	Sequence	tw-bw	0.04	-0.66, 0.74		
		bw-tw	0.76**	0.11, 1.41		

*** p <0.05*

Discussion

The results of this study showed that changing from tap to bottled water decreased urinary arsenic concentrations, but the decrease was not statistically significant. The only factors, which were shown to be important, were the sequence of the intervention and age. There are a number of factors, which may explain the findings.

The tap water arsenic concentrations were significantly lower than those previously recorded for the study area and changing from a drinking water arsenic concentration of 17.5µg/L to 1µg/L may not have been sufficient to detect a significant difference in corresponding urinary inorganic arsenic concentrations. Another finding was that during the intervention, the urinary inorganic arsenic results did not drop to the level observed in previously recorded in control populations (Hinwood et al. 2002).

The trial was small with only 23 participants and was unlikely to have had sufficient power to detect a difference in inorganic urinary arsenic concentrations if one existed. This was confirmed when a re-calculation of the sample size to detect a statistically significant difference (<0.05) for the lower than expected urinary inorganic arsenic concentrations observed in this study, at 80% power was 141 subjects. This demonstrates the need to include a measure of intraperson variation in the sample size calculation rather than relying on population means.

It may also be argued that insufficient time was allowed for the washout period. This study did not exclude participants from washing, bathing and cooking in tap water, although participants were requested to use bottled water for preparation of drinks and food. These sources of exposure may also contribute to short term exposure, leading to higher than anticipated urinary inorganic arsenic concentration when a given participant was consuming bottled water.

Another major factor may have been non-compliance in both directions. That is, there may have been carryover of the use of bottled water in the week where tap water was to be used and alternatively not using the bottled water at all times bottled water was to be consumed. This non-compliance would have the effect of reducing the difference in urinary arsenic concentrations between different weeks. The observation of an increasing difference in urinary arsenic concentrations when drinking tap water versus bottled water with an increasing average urinary arsenic concentrations for the study period for each individual supports this contention.

The individual variation in urinary arsenic concentrations was very high and this combined with a coefficient of variation (CV) for the analytical method of 16% may have contributed to the lack of statistical significance in the findings even though the CV was in the acceptable limits of the standard method.

Conclusion

This preliminary work presents important information for the debate surrounding the proposed reduction of current guidelines values for arsenic in drinking water. This is more pertinent to those supplies which may not be in compliance with proposed guideline values but which are not substantially above them.

The results of this study indicate that for people living in areas with drinking water arsenic concentrations near proposed and current guideline values (up to 20µg/L), simply providing bottled water instead of town water supply is unlikely to cause a large reduction in urinary arsenic concentrations. Further, the influence of other potential contributors to exposure and lifestyle factors need to be included, as they will assume greater importance as drinking water concentrations are reduced.

To confirm these results, an intervention study with a larger sample size is required

and other routes of exposure such as exposure from bathing and cooking need to be more fully explored. In addition, the significance of reductions at different drinking water arsenic concentrations also needs to be explored and estimated costs of arsenic removal and benefits of improved health examined.

Acknowledgments

The authors would like to thank the CRC for Water Quality and Treatment for funding this trial and Topsy Baulch for her assistance in collecting samples.

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Climate Variations and the Transmission of Ross River Virus Infection in Coastal and Inland Region of Queensland: An Analysis from Townsville and Toowoomba

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To determine the different impact of climate variability on the transmission of Ross River (RR) virus infection between coastal and inland regions of Queensland, historic data analysis was conducted in Townsville and Toowoomba over the period 1985-96. The results show that temperatures, rainfall and high tides are possible contributors to the transmission of RR virus infection in the coastal region of Queensland, with a lagged effect of zero to four months, while temperatures were the main potential risk factor for the transmission of RR virus infections in inland regions of Queensland. These different climatic risk factors in coastal and inland regions seem to have their influence through the different distributions of the vectors of the diseases in the two regions. This study suggests that the transmission of RR virus infection is related to climate variations and attention should be paid to this, given global warming and its consequent impacts.

Key words: *Ross River Virus; Climate Change; Queensland; GLS Regression Analysis*

Arboviral diseases, including Ross River (RR) virus infection, are among the most sensitive of all diseases to climate change (Lindsay & Mackenzie 1996). Various studies have shown that the transmission of RR virus infection is related to weather and climate (Lindsay & Mackenzie 1996; Tong et al. 2001). Temperatures, rainfall, relative humidity, and high tides can affect the development of mosquitoes, the vector of the disease, and hence the transmission of the RR virus infection.

RR virus infection, or epidemic polyarthritis, is a mosquito-borne disease caused by an alphavirus, Ross River virus. It is a debilitating and frequently persistent disease characterised by arthritis, fever, rash, and fatigue (Mackenzie, Lindsay & Coelen 1994; Curran et al. 1997). RR virus infection is the highest incidence vector-borne disease in the Australasian region, with thousands of cases occurring annually in Australia (Mackenzie, Lindsay & Coelen 1994). For example, the national notified incidence in 1996 was 42.7/100,000 and a total of 53,347 laboratory-confirmed cases were reported to the Commonwealth Department of Health over the period 1991-2000. Queensland had more than 60% of all the cases (Commonwealth Department of Health and Ageing 2001; Curran et al. 1997).

The virus has been isolated from 38 species of mosquitoes in Australia (Mackenzie, Lindsay & Coelen 1994). The disease has different mosquito vectors in different regions. On the northern coasts of Australia, it is *Aedes vigilax*. On the south and southwest coasts of Australia, *Aedes camptorhynchus* is thought to be the main vector. Both species of mosquitoes are dependent on tides. *Culex annulirostris*, which breeds in vegetated semi-permanent and permanent fresh water, is the major vector in the inland tropics and temperate inland regions of New South Wales and Queensland

that are subject to flooding or irrigation during summer (Lindsay, Mackenzie & Condon 1993). *Aedes notoscriptus* may be important in semi-rural and urban areas. These freshwater mosquitoes are closely associated with human habitations (Dale & Morris 1996). There are three possible drivers of RR virus infections: rainfall and its effect on salt-marsh-breeding mosquito population dynamics, rainfall and its effect on fresh water-breeding mosquito population dynamics, and tidal inundation of saltmarsh and its effect on mosquito population. All of these, together with other potential risk factors, could lead to the transmission of the disease.

We have reported that there was a spatial shifting for RR virus infections in Queensland over the period 1985-96, which could be due to the impact of climate variations (Tong et al. 2001). We also found that temperatures, rainfall and high tides are possible contributors to the transmission of RR virus infection in the coastal region of Queensland (Bi & Parton 2002). However, the differences in climatic factors affecting the transmission of the disease between coastal and inland regions remain unclear. Therefore, in the present study a historic data analysis was conducted for Townsville and Toowoomba, a coastal and an inland town in Queensland, respectively, using data covering the period 1985-96.

Materials and Methods

Study sites and populations

Situated in the tropics of North Queensland, Townsville is located approximately 1,380 kilometres north of Brisbane, and 350 kilometres south of Cairns. With a population exceeding 130,000 in 1996, it is the largest population centre in northern Queensland. With incidence per 100,000 being the main variable of interest, all annual residents of Townsville over the period 1985-96 were treated as the study population (as the denominator). Locally notified RR virus infections during this period were treated as the numerator.

Toowoomba from the Darling Downs and Granite Belt was chosen to represent the inland region of Queensland in this study, given its population and incidence of the disease. As Australia's second largest inland city after Canberra, Toowoomba is located 700 metres above sea level at the eastern edge of the Great Dividing Range. With a population of nearly 90,000 in 1996, the city sits at the gateway to the inland western region from the coastal corridor of Queensland.

Data collection

The Queensland Department of Health provided notified cases of RR virus infection. Population data for Townsville and Toowoomba were provided by the Australian Bureau of Statistics. Climate data were retrieved from the Australian Bureau of Meteorology.

Data analysis

Data analysis was conducted using the Statistical Package for the Social Sciences (SPSS) (SPSS 2001). The monthly incidence of RR virus infections in each town was treated as the dependent variable, and climatic variables such as monthly mean maximum and minimum temperatures, relative humidity, monthly total amount of precipitation and monthly mean high tide as independent variables. Spearman's correlation analyses were conducted between monthly climatic variables and the incidence of the disease. For consideration of lagged effect, the analyses were conducted between the incidence of the disease and climatic variables in the current month, and previous one, two, three and four months. Since there might be auto-correlations among both dependent and independent variables, AutoRegressive Integrated Moving Average (ARIMA) and Generalised Least Square (GLS) regression analyses were performed to control for this. A model was developed

after the effect of auto-correlation had been removed by the ARIMA procedures, and the GLS regression analysis was conducted to assess the independent effects of each climatic variable thereby inferring unique variance aspects (Box & Jenkins 1976).

Results

The monthly mean values of dependent and independent variables at the study sites

Table 1 describes the mean, standard deviation, and the minimum and maximum values of the monthly incidence of RR virus infection and climatic variables in Townsville and Toowoomba over the study period. It shows that the monthly mean incidence of the disease was 11.75/100,000 in Townsville, a coastal town. This was much higher than 3.72/100,000 in Toowoomba, an inland town. The information about the climatic variables in these towns also shows differences in temperatures, rainfall and high tides, in which Townsville has higher temperature and more rainfall.

Table 1: Monthly incidence of RR virus infection and climatic variables in Townsville and Toowoomba, Australia, 1985-96*

	Mean	Std. Deviation	Minimum	Maximum
Incidence	11.75^	2.69^^	0.00	119.46
(1/100,000)	3.72^	1.82^^	0.00	22.67
MaxT °C	28.96	2.22	23.60	34.30
	22.50	3.36	14.50	30.50
MinT °C	19.90	2.37	12.40	25.70
	11.90	2.95	4.80	18.80
3pmRH (%)	55.80	6.47	41.00	74.00
	51.20	9.84	25.00	76.00
9amRH (%)	64.80	6.20	52.00	85.00
	72.30	8.08	46.00	89.00
High Tide (cm)	278.7	9.40	258.50	309.20
	#	#	#	#
Rainfall (mm)	19.95^	5.89^^	0.10	865.40
	11.22^	3.02^^	0.10	421.20
SOI**	-3.10	9.94	-25.40	21.00

** Upper line: the data in Townsville; lower line: the data in Toowoomba; ^ the geometric means of incidence of the disease and rainfall; ^^geometric standard deviation; # data inapplicable because Toowoomba is not a coastal town*

*** Southern Oscillation Index*

Correlation between the monthly incidence of RR virus infection and climatic variables at the two study sites, 1985-96

Table 2 shows that, with the exception of the SOI, there were significant correlations between the monthly incidence of RR virus infection and climatic variables, both in Townsville and Toowoomba. The lagged effect lasts from zero to two months in Townsville and zero to four months in Toowoomba. It seems that at both locations the correlation between monthly mean minimum temperature and the incidence of the disease was marginally stronger than that between monthly mean maximum temperatures and the incidence of the disease.

Table 2: Correlation between monthly incidences of RR virus infection and climatic variables in Townsville and Toowoomba, 1985-96

	Townsville		Toowoomba	
	Coefficient	*P*	Coefficient	*P*
MaxT (°C)	0.432 (2)	0.000	0.482 (4)	0.000
MinT (°C)	0.454 (2)	0.000	0.496 (3)	0.000
Rainfall (mm)	0.387 (2)	0.000	0.327 (4)	0.000
SOI	-0.156		0.089	0.150
	0.102			
3pmRH (%)	0.342 (1)	0.000	0.306 (1)	0.000
9amRH (%)	0.292 (1)	0.000	0.329 (1)	0.000
HT (cm)	0.410	0.000	#	

The number in the bracket is the amount of lagged months; # data inapplicable

Inter-correlations among climatic variables in the two locations were also examined (Table 3). This shows that there were strong correlations between monthly mean minimum and maximum temperatures, and between 3.0pm relative humidity and 9.0am relative humidity, both in Townsville and Toowoomba. In the assessment of the relationship between climate variability and the transmission of RR virus infection using regression analysis, these correlations indicated where attention needed to be given to potential problems of multicollinearity. Therefore, they were put

into different regression models as shown in Tables 4 and 5.

Table 3: Inter-correlations among climatic variables in Townsville and Toowoomba, 1985-96^

	MaxT	MinT	3pmRH	9amRH	Rain	HT	SOI
MaxT	1.00						
	1.00						
MinT	0.93	1.00					
	0.92	1.00					
3pmRH	0.41	0.65	1.00				
	-0.20	0.17	1.00				
9amRH	0.14	0.36	0.79	1.00			
	-0.16	0.19	0.92	1.00			
Rain	0.29	0.44	0.67	0.60	1.00		
	0.25	0.45	0.52	0.43	1.00	1.00	
HT	0.39	0.42	0.48	0.32	0.38	1.00	
SOI	-0.10	0.06	0.35	0.30	0.19	0.11	1.00
	-0.04	-0.002	0.18	0.13	0.21	#	1.00

^ The upper line is the results of Townsville and lower one is the results of Toowoomba; # data inapplicable

Regression analyses between the monthly incidence of RR virus infection and climatic variables, 1985-96

The results for Townsville (Table 4) show that monthly mean minimum temperature, high tide and rainfall or the monthly mean maximum temperature and high tides were significant climatic variables associated with the transmission of RR virus. It seems that the monthly mean minimum temperature plays a more important role than the monthly mean maximum temperature because Model 1 has higher R^2 values than Model 2, indicating larger extent of variance explained. These results tend to confirm previous observations that particular temperatures and rainfall are necessary for the developments of the mosquitoes and the virus within the mosquitoes (Lindsay, Mackenzie & Condon, 1993; Tong et al. 2001). Further, *Aedes vigilax* the main species in northern Queensland is sensitive to high tides.

The regression analysis for Toowoomba (Table 5) shows that temperatures affect the occurrence of RR virus infection in the city. As for Townsville, it seemed that the role of mean minimum temperatures is possibly more important than that of mean maximum temperatures.

Table 4: Climatic variables and monthly incidence disease in Townsville, 1985-96

Explanatory variables	B		*P* value
Model 1			
MinTP2 (°C)	0.0618		0.011
HT (cm)	0.0098		0.046
RainP2 (mm)	0.0006		0.036
Constant	-8.708		0.000
		R^2=0.37	
Model 2			
MaxTP2 (°C)	0.0388		0.018
HT (cm)	0.0095		0.047
RainP2 (mm)	0.0005		0.121
Constant	-7.6118		0.000
		R^2=0.32	

Table 5: Climatic variables and monthly incidence disease in Toowoomba, 1985-96

Explanatory variables	B		*P* value
Model 1			
MinTP3 (°C)	0.0395		0.020
9amRHP1 (%)	0.0091		0.114
Constant	-5.9560		0.000
		R^2=0.32	
Model 2			
MaxTP4 (°C)	0.0310		0.039
9amRHP1 (%)	0.0099		0.085
Constant	-6.2415		0.000
		R^2=0.30	

Discussion

RR virus infection is fairly common in Australia. In southeast Australia, cases occur primarily between January and April; in south coastal Victoria and southwest Australia, they are between October and December (Kay & Aaskov 1988; Mackenzie, Lindsay & Coelen, 1994; Mackenzie et al. 1998). The distinctive seasonal pattern is related to the life cycle and habitat of the vector. Climate variability might impact on the incidence, temporal and spatial distributions of the disease via its influence on the vectors.

Rainfall is important in the transmission of RR virus infection. Mosquitoes have

aquatic larval and pupal stages and therefore require water for breeding. Sufficient amounts of precipitation will assist in maintaining the mosquito's breeding habitats further into the summer months, which is particularly important for fresh water breeding mosquitoes. Outbreaks of RR virus infection in Western Australia are predominantly rainfall associated; a feature of the outbreaks in the arid north and interior of the state is the short interval between the occurrence of heavy rains and notification of the first human case (Lindsay, Mackenzie & Condon 1993).

Timing of rainfall is as important as the amount. Major outbreaks of RR virus infections in southwestern Australia, for example, usually follow heavy late spring or summer rain, but not heavy winter rain. The outbreaks in the arid Pilbara region of Western Australia usually follow heavy autumn and winter rain (Lindsay & Mackenzie 1996). The pattern of rainfall is also important in the transmission of RR virus infection. Too much rain at once might flush dormant mosquito eggs away from breeding sites. More frequent, lighter rains might replenish existing breeding sites and maintain higher levels of humidity, which assists in dispersal and survival of adult mosquitoes. Also above average winter and spring rains are thought to increase the survival rate of juvenile western grey kangaroos (Lindsay & Mackenzie 1996; McMichael 1996).

Temperature has a dramatic effect on the length and efficiency of the extrinsic incubation period (EIP) of RR virus in its vectors. Mosquitoes exposed to higher temperatures after ingestion of RR virus become "infective" more rapidly than mosquitoes of the same species exposed to lower temperatures (Lindsay & Mackenzie 1996). Transmission of RR virus may therefore be enhanced under warmer conditions because more mosquitoes become infectious within their often-short life span. Temperatures, especially minimum temperatures, may also play an important role in maintaining the survival of mosquito larvae in winter and have a significant impact on the development of adult mosquitoes. But too high temperatures in summer might speed the death of adult mosquitoes (Lindsay & Mackenzie 1996). Also, the high temperatures might force people to stay in their houses and thus reduce their contact with mosquitoes.

Over 90% of cases of RR virus infection in southwestern Australia occur between November and March, the warmest months of the year. This may, in part, be due to a faster development of the mosquito and a much shorter EIP of RR virus in *Aedes camptorhynchus* at warmer temperatures. However, the outbreaks of RR virus infection in arid regions of Western Australia rarely occur during the hottest months of the year. Combined with temperatures, it was found that heavy autumn and winter rains are far more likely to result in outbreaks of RR virus infection than summer rains (Lindsay & Mackenzie 1996). This is probably because temperatures in summer in Western Australia are too high and reduce the growth and development of the mosquitoes. High temperatures also affect the reproduction of the vertebrate host.

The study at Port Hedland and Exmouth on the arid Pilbara coast of Western Australia showed that most RR virus infections occurred during the months of highest relative humidity in both towns. This might reflect the timing of rainfall at the two towns to some extent. However, the fact that rain sometimes falls at other, less humid times of the year indicates that humidity should be considered as another possible contributing factor to outbreaks of arboviral diseases, particularly in normally arid regions (Lindsay & Mackenzie 1996).

Tidal inundation of salt marshes is a major source of water for breeding of the important arbovirus vectors *Aedes vigilax* and *Aedes camptorhynchus*. Adult females of both species lay their eggs on soil, mud substrate and at the base of plants around the margins

of their breeding sites. Large populations of adult mosquitoes can emerge as little as eight days after a series of spring tides, depending on temperature. A rise in sea level might lead to more frequent and widespread inundation of coastal saltmarshes in the region, as a consequence extending the breeding grounds of these two species of mosquito. This, in turn, could give rise to a much larger summer population of mosquitoes and consequent exposure of large numbers of urban dwellers on the eastern seaboard to infection. This is particularly important to the transmission of RR virus infections in the coastal region, especially the northern coastal district of Queensland, including Townsville, because the main mosquito species there is *Aedes vigilax* (Lindsay, Mackenzie & Condon 1993; Russell 1995). Perhaps a principal cause of the outbreak of RR virus infections in 1988-1989 in Western Australia was the rise in sea level and an accompanying increase in high tides. It was found that for the last eight months of 1988 and the first four months of 1989, the mean sea level was 5.5 cm above the long-term mean. Thus, large areas of saltmarsh that are normally dry during the summer months were regularly inundated with water through the summer of 1988-1989. This provided ideal breeding sites for *Aedes camptorhynchus* during late spring and summer and *Aedes vigilax* in summer. Of particular importance was the fact that the regular inundation of the breeding sites enabled *Aedes camptorhynchus* to persist through the summer of 1988-1989 (Lindsay, Mackenzie & Condon 1993).

Conclusion

This study in Queensland showed that there was an association between climate variability and the transmission of RR virus infection in Townsville, a coastal region in northern Queensland, with 30% to 37% of variance explained and significant though low power of association. The rise in temperatures (minimum and maximum), precipitation and sea levels might have an important impact on the epidemic potential in northern Queensland in future. This is because these climatic variations will act on *Aedes vigilax*, the main vector in this area and thus on the transmission of the disease. This could take two months, which is consistent with the two month lagged effect from this study. If global warming results in a significant sea level rise, an increase in temperatures and more irregular rainfall as predicted by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) (CSIRO 1996), the distribution of breeding sites for saltwater mosquitoes will also change locally along low lying parts of the Queensland coast leading to an expansion of the epidemic focus and a higher potential for epidemics.

In Toowoomba, an inland region of Queensland, the results showed that there were correlations between various climatic variables and the monthly incidence of RR virus infections. However, in the regression analysis, only temperatures (minimum and maximum temperatures) were significantly associated with the transmission of RR virus infection. However, the reason why "precipitation" was not significant in the transmission of the disease remained unclear. Obviously, arboviral transmission in an area is complicated and involves many parameters, such as vertebrate host identity and availability, vector identity, breeding conditions and sites, vegetation/herbage for vector protection during sunlight hours; as well as weather conditions, and physical requirements (tides, saltmarsh, rivers, lakes, or dams). In this study in Toowoomba, only weather conditions were taken into account in the analysis, therefore, multi-disciplinary study is needed in future to have a better understanding of RR virus transmission in this area.

Besides other biological and physical parameters mentioned above, the various distributions of different vectors between coastal and inland regions of Queensland and their various sensitivities to climate effects could be one of the main reasons for

the higher incidence in the coastal region than in the inland region. These need to be addressed in further studies. However, results from this study should be applicable to other districts in Australia and even in the Australasian region generally, and be helpful to policy makers in developing preventive strategies.

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REPORTS AND REVIEWS

Has the Tropical Bed Bug, *Cimex hemipterus* (Hemiptera: Cimicidae), Invaded Australia?

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This article provides the first published record of the tropical bed bug, C. hemipterus *in Australia and describes a dual infestation of bed bugs involving two species in a backpackers' hostel. The implications of the introduction of this pest into Australia are discussed.*

Key words: *Tropical Bed Bug; Australia*

Bed bugs are haematophagus arthropods known to attack humans. Reactions to their bites vary; some individuals do not notice the bite whereas others report intense local pain, which might disrupt sleeping patterns. Following the bite, large wheals may be produced accompanied by itching and inflammation, swelling, and occasionally blistering of the skin (Tharakaram 1999). Bed bugs have cryptic habits; they usually feed during the night and hide on and under mattresses, bed frames and various crevices in walls and room furnishings during the day. Often infestations are not readily apparent and the bites may be attributed to other blood sucking arthropods, with inappropriate control attempts ensuing.

Bed bugs as a group have a wide distribution. The human pest species are the common bed bug (*Cimex lectularius*) and the tropical bed bug (*Cimex hemipterus*). In Australia, scientific literature regarding the incidence and distribution of bed bugs is notably absent, although their presence is well known among pest controllers, and the accommodation industry who often find themselves with this uninvited guest. The occurrence of *C. lectularis* has long been recognised in Australia (Lee 1975; van Dyck 1995). This article provides the first published record of *C. hemipterus* in Australia and describes a dual infestation of bed bugs involving both species in a backpackers' hostel. The implications of the introduction of this pest into Australia are discussed.

In May 1998, the Department of Medical Entomology at ICPMR received samples of bed bugs from the Australian Quarantine and Inspection Service for species confirmation. The identity of these bed bugs was confirmed as *C. hemipterus*, with differential diagnosis being based on the lack of an upturned lateral flange on the thorax in *C. hemipterus*, which is present in *C. lectularius*, as well as other taxonomic features (Ghauri 1973). This was believed to be the first record of the tropical bed bug in Australia. The bed bugs were collected from a cafe in Bundaberg, following complaints from patrons who reported being bitten while dining. Upon inspection, bed bugs were found in wooden slats within the chairs and behind nearby skirting boards. Several chemical treatments were required before the bed bugs were fully exterminated. The origin of the Bundaberg infestation remains unknown; however, the cafe is situated in close proximity to backpacker hostels and inspections of these revealed several with

active bed bug infestations (although the species of *Cimex* was not determined). As mentioned, it is not known how the bed bugs came into the country, but it is speculated that they might have been transported to the cafe from one or more of the hostels via backpacks, as travellers were observed placing them beside and on the chairs at the cafe. If this is in fact what happened, then the potential for the bugs to be widely dispersed within Australia should be recognised.

In early July this year, a pest inspection of a backpackers' hostel on the Queensland Sunshine Coast (for confidentiality, the town is not given), conducted by one of the authors as part of a preventative maintenance program, revealed an infestation of bed bugs. Live bed bugs were found in several rooms and mainly on, or in close association with, bed mattresses. Following submission to Medical Entomology, ICPMR, identification of the samples revealed a mixed infestation of both *C. lectularius* and *C. hemipterus*. Samples from each room were not kept separate, and so it was impossible to ascertain if both species were cohabiting but the presence of both bed bug species within the same premises is the first report of a dual infestation for Australia. As with the previous case, the source of the infestation was not identified, nor could the duration of the infestation be determined. Advice on how to recognise and control the infestation was provided to the owner.

Overseas, *C. hemipterus*, as its common name suggests, has a tropical distribution but it is also found in the subtropics, whereas *C. lectularius* occurs mainly in temperate regions and the species overlap towards the edge of their temperature preference (Burgess 1990). As both species have now been identified in Australia, the risk of potential infestation must cover a greater geographical area than previously assumed. In areas where the two species are sympatric, cohabitation is known to occur (Newberry et al. 1987). However, the behaviour and habits of the two species appear not to differ so that control strategies do not have to be specifically tailored. This may change in light of recent reports from overseas of chemical resistance to the synthetic pyrethroids in *C. hemipterus* (Myamba et al. 2002). As the main chemical of choice for control in Australia is permethrin (a synthetic pyrethroid), control failures may well occur. This would be especially confounding in those locations where both species coexist if identification to the species level was not attempted.

The introduction of the tropical bed bug, *C. hemipterus*, into Australia is perhaps not unexpected, as there appears to be a current resurgence of bed bugs worldwide (Krueger 2000; Paul & Bates 2000). This has been largely attributed to an increase in international travel, with the bed bugs being carried in clothing, luggage and bedding, and the movements of infested furniture and furnishings. Changes in the pest control industry are also thought to have inadvertently favoured bed bugs, as control for cockroaches has moved away from traditional methods of chemical application towards specific baits and insect growth regulators, which do not impact on bed bugs as former methods have done (Koehler & Harlan 2001). It is quite possible that *C. hemipterus* has been introduced into Australia previously, but as most pest controllers do not attempt to determine bed bugs to the species level in an infestation prior to treatment, the identity of *C. hemipterus* would have gone undetected. There also exists the likelihood that the *C. hemipterus* infestations described in this report have arisen not via travellers from overseas, but through local spread of the pest. The possibility that the species is now established in Australia cannot be excluded.

Traditionally in developed nations, backpacker hostels have been at the greatest risk of infestation, although recent trends have also included more "up-market" hotels and even urban homes (Krueger 2000). It is unlikely that bed bugs will reach the high

level of infestations seen in the past. However, a lack of recognition of the pest and its status in Australia among health managers and pest controllers, along with an increase in international tourism, will ensure that bed bugs will be an ongoing problem, especially for the accommodation industry. This identification and reporting of a new bed bug species that might have become established in Australia will it is hoped raise the profile of these nuisance pest species.

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A Fresh Approach to Food Safety Assessment: Introducing the AIEH Food Safety Standard of Practice and Australian Food Safety Assessment

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The recent implementation of nationally uniform Food Safety Standards demonstrated a real need for an approach to food safety assessment that is nationally uniform and consistent with the legislative standards. The Food Safety Standard of Practice (FSSP) and Australian Food Safety Assessment (AFSA) were developed with the aim of providing both an assessment tool and a standard of practice for Environmental Health Practitioners (EHPs) undertaking food safety assessments. AFSA is a comprehensive assessment tool, which can be used to determine risks associated with food handling operations and to adjust assessment frequency accordingly. The Food Safety Standard of Practice is professional guidance document aimed at promoting consistency and accountability in the work practices of environmental health practitioners. The development of the FSSP and AFSA followed an extensive consultation period. Each State and Territory was surveyed to assess the status of food law reform. Additional consultation was carried out with organisations including Food Standards Australia & New Zealand, the Department of Health & Ageing and the Australian Local Government Association. The Food Safety Standard of Practice and the Australian Food Safety Assessment are initiatives of the South Australian and Victorian Divisional Food Safety Special Interest Groups. Funding was provided by the South Australian Department of Human Services.

Key words: *Food Safety; Assessment; Food Safety Standard of Practice*

A constant criticism from the food industry has been the variation between authorities in the application of legislation. The recent implementation of the Food Safety Standards (Chapter 3 of the *Food Standards Code*) across Australia highlighted the need for a nationally consistent approach to food safety assessment.

The Australian Institute of Environmental Health's Strategic Plan (2003-2004) states one of the organisation's goals to be: "Professional Development: To ensure professional excellence in the science and practice of environmental health".

In fulfilling its leadership and educational role, the Australian Institute of Environmental Health has published its first professional practice standard, which addresses assessment of food safety. The Food Safety Standard of Practice and Australian Food Safety Assessment (AFSA) aim to provide objective, non-prescriptive and nationally consistent professional guidance and tools for Environmental Health Practitioners (EHPs) who are currently dealing with the many changes to food safety surveillance.

Special Interest Groups Collaborate

After discovering similar and complementary initiatives, the South Australian and Victorian Divisional Food

Safety Special Interest Groups worked in partnership with the aim "to develop an approach for the assessment of food safety that is nationally uniform and consistent with legislative standards". Seeing merit in the concept, funding was provided by the South Australian Department of Human Services.

To ensure accuracy and relevance of the final products, project methodology included research of the current status of food law reform and consultation with key stakeholders. Key food safety representatives from each State and Territory were surveyed about issues such as timelines for the introduction of the Food Safety Standards, assessment frequency requirements, notification/registration/licensing and food safety program/auditing requirements. Due to the variation in administrative arrangements relating to these issues, it was necessary for the practice standard and assessment tool to be flexible in the manner it may be applied.

At least twenty enforcement agencies from South Australia, Tasmania and Victoria were involved in a trial of the draft practice standard and assessment tool over a period of one month during May 2003. Comment was provided via a questionnaire and the draft material further refined prior to consultation with Food Standards Australia New Zealand, Department of Health and Ageing and Australian Local Government Association. Feedback from consultation confirmed that the aims and objectives of the project had been met and that the final products will be useful to Environmental Health Practitioners when assessing food safety.

Purpose of the Food Safety Standard of Practice

The Standard of Practice aims to meet several purposes:

- To provide guidance to EHPs in the assessment of outcome based standards, responding to non compliance (in particular serious non compliance) and allocation of resources on a risk management basis.

- To promote consistency in the assessment of all food handling activities by EHPs against the Food Safety Standards.

- To provide a tool that enables comprehensive assessment of all food handling activities by EHPs.

- To inform the food industry of the minimum standards against which food handling activities are assessed.

It should be noted that the Food Safety Standard of Practice has no legal standing and is not a substitute for proper evidentiary processes and professional judgement taking into account the circumstances of each situation.

Food Safety Policy

An important part of the Standard of Practice is the food safety policy within the document, which is designed to guide EHPs in their decision making. In summary the policy states:

- Professional EHPs must conduct themselves in a consistent and accountable way.

- The assessment of food safety by EHPs must focus on whether food businesses are achieving or able to achieve the required food safety outcomes.

- Enforcement agencies have a legal responsibility to administer and implement the legislation and ensure that food businesses are meeting their obligations.

- Proprietors and operators of food businesses have a legal responsibility to ensure all food they sell is safe and

suitable for human consumption. Food businesses should be able to demonstrate that they are managing the food safety risks at any time.

- Food businesses should be able to choose a method for achieving compliance that is most appropriate for their business.

- A food safety program is a documented way that food businesses can demonstrate that they are complying with the Food Safety Standards

- Food businesses are responsible for ensuring any non-compliance is resolved.

An Outcome Based Approach

The National Food Safety Standards that have been adopted in Australia specify food safety outcomes that must be achieved, rather than setting prescriptive requirements. This means that a food business is able to achieve compliance through a number of ways.

The assessment of food premises should be based on the business demonstrating compliance with the required food safety outcomes.

As mentioned earlier, inconsistency is a constant criticism of Environmental Health Practitioners. Why is this the case? Why is there inconsistency when the same standards are being enforced? In developing the Standard of Practice this issue has been considered.

Previously inconsistency occurred when EHPs mandated how the business must achieve the required outcome. This could vary from one enforcement agency to the next depending on the differing interpretation and preferences of that agency. Many examples of this are seen in the way structural requirements are enforced between different authorities.

Structural requirements are in place so that food safety outcomes can be achieved such as keeping the premises clean, preventing access by pests and ultimately protecting food from contamination. This concept is reinforced in the Purpose of Standard 3.2.3 "Food Premises and Equipment".

Because the food safety outcomes are clearly set in the Food Safety Standards, the basis for improved consistency in assessment is provided.

Australian Food Safety Assessment

Australian Food Safety Assessment (AFSA) is a tool to encourage comprehensive assessment for compliance with safe food outcomes in a uniform and consistent manner using the Food Safety Standards as the framework for the assessment.

The key feature of AFSA is the food safety checklist. AFSA assesses all food handling processes that are undertaken in a food business including:

- Receiving
- Storage
- Processing
- Display/Serving
- Packaging
- Transportation
- Food recall/disposal

Activities that support food safety are also assessed such as:

- Health, hygiene and knowledge of food handlers
- Premises hygiene and maintenance
- Temperature measuring and recording.

AFSA has been developed in the form of a duplicate pad, which offers many benefits including the ability to provide immediate

written feedback to the business operator where food safety risks are not effectively controlled.

AFSA is suitable for use in any type of food business, including temporary food premises or businesses using a food safety program or other quality system. The Food Safety Standards apply to all types of premises where obligations and outcomes required are the same. The difference when dealing with, for example temporary premises, is that compliance may be achieved in a different way and not all risks are present in the business. A business with a food safety program or other quality system would keep records or may have access to its own scientific data and evidence in order the demonstrate compliance with food safety outcomes.

The Standard of Practice includes an Explanation Guide that is intended to be given to proprietors/operators to inform them of what is considered during an assessment and possible options for demonstrating compliance. It can also be used by EHPs to obtain clarification of the intent of the individual prompts on the assessment form. The need for EHPs to refer to the Explanation Guide will reduce with time as they become familiar with the issues and standards behind the prompts. The Explanation Guide is not intended as a comprehensive checklist.

To promote the basics of food safety to business operators and food handlers, advisory information is printed on the reverse side of the AFSA assessment form. An added benefit of using AFSA will be that comparable data about food business compliance can be collected from enforcement agencies. The results can be used to develop various national and local strategies to obtain better compliance and improved food safety.

Prioritising Non-Compliance

AFSA and the Standard of Practice identify two levels of non-compliance to assist EHPs to prioritise food safety risks. A serious non-compliance is defined as "a non-compliance that poses an immediate risk of unsafe food being sold or if allowed to continue will result in unsafe food being sold". The Standard of Practice provides examples of situations that might be serious non-compliance.

It is the responsibility of the food business to determine the corrective action that they need to take to rectify any non-compliance. EHPs have a wealth of knowledge and experience, and are in a position to give guidance to a business but should empower the business to make the determination of how they will achieve compliance.

The Standard of Practice and AFSA do not suggest the way in which non-compliance must be enforced. Different legal sanctions apply across Australia. However, AFSA does give the enforcement agency a consistent food safety risk basis for any enforcement action.

Risk-Based Determination of Assessment Frequency

The Food Safety Standard of Practice and AFSA advocate a risk-based approach to determine the frequency of assessment, where frequency is not prescribed by statute. Two factors determine assessment frequency - the nature of the business (intrinsic risk) and the level of compliance. The Priority Classification System for Food Businesses (Food Standards Australia New Zealand), provides a method of assigning a classification to a business, which takes into account the intrinsic risk. This classification is used to establish the "starting point" frequency of assessment. The Standard of Practice builds upon the Priority Classification System and offers a model for adjusting assessment frequency based on the level of compliance.

The benefits of the Assessment Frequency Adjustment Model, in conjunction with the Priority Classification System, are that it provides enforcement agencies with a means of:

1. Objectively determining business assessment frequency in an open and transparent manner that is consistent with national best practice; and

2. Allocating resources required for food safety assessment on the basis of risk.

Conclusion

The Food Safety Standard of Practice and AFSA provide EHPs with the opportunity to enhance their professional status by taking a consistent and progressive approach to food safety assessment.

The Food Safety Standard of Practice can be easily accessed by downloading a copy from the Australian Institute of Environmental Health website at www.aieh.org.au. Further information about AFSA and ordering details can also be found on the website.

Due to the new and evolving national food safety laws, the Food Safety Standard of Practice and AFSA will undergo regular review. Environmental Health Practitioners, either individually or collectively (for example, through your Food Safety Special Interest Group), are encouraged to provide feedback.

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Common Ground and Common Sense: Community-Based Environmental Health Planning

R. Nicholson, P. Stephenson, V.A. Brown and K. Mitchell (Eds)

Commonwealth of Australia, 2002, 152 pp. ISBN 0 642 820297, $19.90 (paperback)

Common Ground and Common Sense: Community-based Environmental Health Planning is a handbook developed at a workshop comprising multi-sector, multi-disciplinary participation in Canberra in 2001. The workshop was facilitated by Peter Cuming (Sustainable Futures Australia); the handbook follows the "Planning Web" framework proposed by Cuming. The Community-Based Action for Environmental Health (CEHAP) Action Web becomes the guiding structure for the handbook using six sections: people caring for place, communities in action, community as partners, multiple alliances, place-based planning, and future-directed action. This process is intended for environmental managers and health officers, public health officials, and community action groups who seek change in the environmental health of local communities. The handbook is designed to support action in: "linking the community commitment of voluntary programs to the legislative power of government; bridging the existing divide between health and environment; and bringing together the wide range of activities and resources from community, expert, and government practice".

The handbook is concise and well written, using stepwise, simple outline format with robust real-world examples of implementation of each CEHAP Action Web section. The major strength of this handbook is the tap to local communities and stress on integration of perspective, skills, and disciplines in approaching community-level action plans. Coming from the perspective of an American working in the public health arena, I found this a refreshing perspective, as the connection, interest, and empowerment of local communities is often overlooked in federal and state level planning. The handbook also provides multiple exercises to engage the individual reader as well as groups actively using the handbook to plan a community action. One example exercise helped this reviewer discover his ecological footprint - that of a consummate devil, shared by most Americans.

There are a few concerns with the handbook, which stem from the reviewer's American perspective. Industry is a major force of resistance in action plans to better environmental health. The handbook acknowledges the role of industry as a force to contend with but does not provide clearer statements that encourage engagement of community and industry up-front.

This said, the handbook is a highly valuable resource for community-level planning and is applicable to national level planning for environmental health. This reviewer, reading the handbook from an American point of view, saw much broader application of the CEHAP Action Web towards multi-sector, multi-disciplinary collaboration in the ever-evolving global village where public health issues that demand local participation have become major concerns to the international community.

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Health and Environmental Assessment of Site Contamination: Proceedings of the Fifth National Workshop on the Assessment of Site Contamination

A. Langley, M. Gilbey and B. Kennedy (Eds)

Environment Protection and Heritage Council and enHealth Council, 2003,
ISBN 0 642 32355 0 (paperback), ISBN 0 642 32371 2 (web version)

This Report on the Proceedings of the 5th National Workshop held in Adelaide in May 2002 represents a further valuable reference document in a series of similar reports since 1991, and is a useful addition to the library of those involved in the environmental and/or health risk assessment of contaminated sites.

Soil contamination became a major issue across Australia towards the end of the 1980s. Prior to this there certainly had been instances of environmental health concerns in relation to soil contamination, but it was the combination of urban encroachment into areas previously zoned industrial, followed by the establishment of Environment Protection Authorities that really drove the issue into the limelight in Australia. Some rather distressing and sometimes spectacular scenarios arose, such as the eviction of residents from a new housing estate at Ardeer in Victoria one evening - apparently without any prior warning - after significant residual lead contamination arising from fallout from an old battery recycling facility was detected.

At that time immediate actions were deemed necessary because of the perceived high risk from exposure to high levels of contaminants in soil. But in most cases there was no confirmed direct health evidence or epidemiological evidence that risks to health were being manifest. Even when it came to biomarkers of exposure (such as blood lead levels), the evidence for actual exposure to lead in soil from many sites was often marginal, although there was also good evidence from places like Port Pirie that significant exposures can occur where there is ongoing exposure to airborne contaminants through either inhalation or ingestion of contaminated dust particles. As a result many health authorities, in both state and local governments, found themselves in a difficult situation, being asked to provide public health opinion or support proposed risk management options, in the absence of a policy framework or agreed tools for carrying out risk assessments.

The first National Workshop in Adelaide in 1991 grew out of this background, facilitated by Dr Keith Bentley, Dr Andrew Langley and others with Commonwealth funding support. Somewhat remarkably, consensus was rapidly achieved between environment and health agencies on a protocol for the health risk assessment and management of contaminated sites (El Saadi & Langley 1991; Australian and New Zealand Environment and Conservation Council 1992). The general approach has continued to be effective to this day, with refinement of tools and broadening of scope of contaminants over the years being progressed through a series of national workshops involving both environment and health authorities in the examination of technical issues. Australia should now be the envy of many other countries in having a practical framework for risk assessment that is protective of public health but at the same time does not lead to massive expense, as witnessed at times in the United States with the so-called "Superfund". The

Australian approach has been made possible by the generous contributions and collaboration of many in partnership, and has managed to maintain a simple approach that avoids some pitfalls such as highly expensive consultancies.

The Fifth National Workshop in May 2002 saw both new and old players from environment and health agencies, as well as universities and research institutions, come together again to tackle the technical issues underlying assessment of old service station sites and sites contaminated by pesticides. Health Investigation Levels (HILs) are derived clearly and concisely for a number of soil contaminants (benzene, toluene, xylene and a range of other hydrocarbons; and pesticides including chlorpyrifos, bifenthrin, imidacloprid and endosulfan).

The work on benzene is particularly notable as a first attempt to make use of a "modified Benchmark Dose" risk assessment approach to carcinogens in soil for the purposes of HIL derivation. The NHMRC Working Group on Toxicity and Risk Assessment is currently further evaluating this methodology.

Other papers presented at the Workshop further advance our knowledge about bioavailability of metals and arsenic. They tend to confirm that while there is a range of availabilities depending on soil type and contaminant source, site-specific assessments are likely to reveal that some metal contaminant hazards do not translate into actual risk to humans, animals or plants as much as our standard conservative assumptions (e.g. that ingested arsenic in soil is 100% bioavailable) would suggest.

The awkward question of how to deal with asbestos (in a variety of forms) in soil has been dealt with in a thorough and practical manner. This is a welcome advance and this aspect alone would make this document worth obtaining for many practitioners.

Ecotoxicological risk assessment remains a difficult challenge, particularly given the huge range of species and soil habitats in Australia. However, there is a fascinating and useful discussion on approaches to assessing the toxicity of complex mixtures to terrestrial organisms.

It is interesting to reflect on the evolution of the knowledge base over the period since 1991 (e.g. the modelling of volatile hydrocarbons in crawl spaces under houses has been a significant technical advance). It is also satisfying to note the increasing understanding of the many complex issues underlying the risk assessment approach.

Future workshops, and there seems no reason to discontinue their successful format, will need to advance our approaches to the difficult area of ecotoxicological risk assessment, among other things. A review of epidemiological knowledge and contemporary methodologies for evaluation of health effects in relation to contaminated sites might be another fertile area for the next program.

The support of the National Environment Protection Council, Environment Protection and Heritage Council, Environment Australia and enHealth Council in bringing the work of the 5th National Workshop to fruition are to be applauded.

Much still remains to be done to conquer some of the remaining complex risk assessment challenges, with a number of areas requiring further investigation and applied research. However, it is unclear (to this author at least) whether we can be confident that present or future health bureaucracies will recognise the value of investing in maintenance of an environmental toxicology capacity across the different agencies and jurisdictions in Australia, where our existing partnerships have resulted in excellent synergies and

efficient, cost-effective ways of tackling the complex questions of health risk assessment and regulatory toxicology.

Note

This document is available on-line free at <http://www.ephc.gov.au/nepms/cs/worksho p _con_sites.html>

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