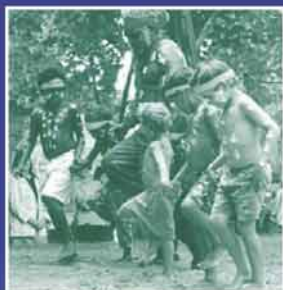


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The Journal of the Australian Institute of Environmental Health



*...linking the science and practice
of Environmental Health*





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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.

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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

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The recent Beaconsfield Mine incident has brought the issue of hazards in the mining industry to Australia's attention. While the occurrence of earth tremors and rock-falls might have a huge impact in the media, the environmental health impact of dust exposure attracts less attention – yet is no less dangerous to miners' health and wellbeing. There is a suite of three papers in this issue that are therefore topical and useful.

Banerjee, Wang and Pisaniello examine the mining environment and have compiled a review of iron-ore dust and its health impacts. Evaluation of the chemical composition of iron-ore dust might impact on methods of governing this environmental health hazard, as the review revealed that silica might play a major role in the risk of pulmonary diseases and disorders including lung cancer, rather than the iron-oxide itself. Regular testing of the mine environment might help prevent occupational iron-ore dust exposures, and medical examinations of miners might assist in early detection of symptoms. Further research could be conducted on nanoparticles of iron oxide, which might be used in lipstick, skincare and vitamins and might penetrate the epidermis.

Silica also remains the major cause of occupational interstitial lung disease, however, methods of diagnosis are not sensitive to early stages of the disease. Wang and Peng review the use of nitric oxide as a non-invasive measure of silica induced health effects. They focus on the possibility of using exhaled nitric oxide investigations as a screening measure of silica-induced harmful effects to identify the early stages of lung disease.

Then Wang and Pisaniello look at the possible role of nitric oxide synthases in the development of asbestos-induced mesothelioma. Mesothelioma rates in Australia are the highest reported national rates in the world. The review reveals that the utilisation of different NOS inhibitors might provide valuable information to reveal the underlying mechanism of malignant mesothelioma development. Wang and

Pisaniello propose to establish animal models for asbestos-induced mesothelioma to examine further the role of nitric oxide synthases in the response to asbestos exposure and mesothelioma development.

The following papers also found in this issue of *Environmental Health* move from dust exposure to more varied environmental health issues, such as Indigenous health and legislation, sun protection policies relating to outdoor activities, and pathogens found in rainwater tanks.

Gray and Bailie examine Northern Territory public health legislation to assess its current and potential impact on Indigenous health. Barriers to improving Indigenous health are identified and the authors propose that public health legislation in the Northern Territory has more symbolic than practical value.

Outdoor sporting activities produce many health benefits, yet also increase the risk of skin cancer from prolonged sun exposure. Earl and Tenkate researched the implementation of sun protection measures and policies within sporting and recreation organisations in Queensland to provide an insight into sun protection activities. They found that encouragement might be needed for local, smaller clubs to promote sun protection.

Tan et al. explore the levels of pathogens in private rainwater supplies in rural Western Australia. The study aims to assess to what extent *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. are present in rainwater tanks. Thermotolerant coliforms and *E.coli* were detected.

Tenkate also provides us with two book reviews, one on *Social Learning in Environmental Management: Towards a Sustainable Future*. The other on *The End of Poverty: Economic Possibilities for Our Time*. His reviews and recommendations are most interesting and informative.

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Iron-Ore Dust and its Health Impacts

Kishore Kumar Banerjee, He Wang and Dino Pisaniello

Department of Public Health, University of Adelaide

Different lung diseases including lung cancer are the major occupational diseases among the workers of iron ore dust. However, health hazards that arise due to exposures to the ore dust vary considerably due to its variable composition in different mines. Iron oxide and silica predominantly contribute to its toxicological properties. By intratracheal instillation in experimental animals, iron oxide exhibits low toxicity and much less inflammation and damage compared to crystalline silica. Carcinogenicity of iron oxide is found to be negative. Silica is the probable contributor of the elevated incidence of lung cancer among the iron ore dust-exposed workers. Epidemiological findings in different parts of the world due to this occupational hazard have also been discussed in this article. It has been concluded that iron ore dust-exposed workers need to be examined periodically in order to determine early symptoms. Routine environmental monitoring and chemical composition evaluation of iron-ore dust are also recommended.

Key words: Iron Ore Dust; Lung Cancer; Silica; Dust Toxicity; Lung Inflammation; Pneumoconiosis

The environmental impact of mining is an extremely complex topic because it involves so many geographical, technical, scientific, and socioeconomic issues. However, dust generation can be found in nearly all workplaces and has remained one of the most intractable of workplace problems since metal ores were first mined. Operations carried out in industrial workplaces today, mining, crushing, sieving, grinding, smelting, welding and so on, all contribute to particle generation. While many of these dusts are relatively harmless causing transient irritation, some give rise to lung fibrosis, others to carcinoma, bronchitis, asthma or other lung disorders (Ripley, Redmann & Crowder 1996).

Global production of iron ore is almost 1000Mt per year in about 50 countries. The former Soviet Union, Brazil, Australia, China, India, and the United States are among major producers. Australia continues to be the world's leading exporter of ore. Having met its obligation to the Western Australian government to process iron ore, BHP Billiton planned to increase production

capacity at its Yandi Mines. The iron ore mines are both open cast and underground. Despite a downward trend in production over the past decade, iron ore is still a most valuable non-petroleum mineral (Godin 1994). Iron ore is the source of primary iron for the world's iron and steel industries. When heated in the presence of a reductant, the mineral substance, metallic iron (Fe) is formed.

Due to the variable composition of iron ore in different mines in different locations and countries and contradictory and conflicting findings from epidemiological studies, it is believed important to review the findings from iron ore dust and its toxic properties in different studies. In order to determine specific components responsible for its toxicological properties, many studies have been carried out with its major constituents, such as iron oxide or silica. This paper focuses mainly on the health hazards that arise due to occupational exposures to iron ore dust and its possible mode of action in experimental and epidemiological studies.

Composition

Iron ore consists mainly of iron oxides; the primary forms are magnetite (Fe_3O_4) and hematite (Fe_2O_3). Miller (1976) has described the impurities commonly found in iron ores. The major constituent impurities are the common 'rock' substances: quartz, alumina, lime and magnesium. The minor constituents often found in iron ore include phosphorous, sulphur, titanium, vanadium, zinc, copper and more rarely chromium, nickel, arsenic, lead, tin, and cadmium. Amounts of these substances vary considerably from one ore to another. Its physical properties are that it is a reddish-brown solid, is not combustible and is insoluble in water.

Workplace Exposure

Limit

The following exposure limits (New Jersey Department of Health and Senior Services 1998) are for iron oxide (dust and fume) measured as Iron:

- OSHA: The legal airborne permissible exposure limit (PEL) is 10 mg/m^3 averaged over an 8-hour work shift.
- NIOSH: The recommended airborne exposure limit is 5 mg/m^3 averaged over a 10-hour work shift.
- ACGIH: The recommended airborne exposure limit is 5 mg/m^3 over an 8-hour work shift

Occupational exposures

Many occupations involve exposure to iron oxide and iron including iron ore miners, drillers, welders, caulker/burners, metal dressers, oxyacetylene cutters, pigment workers, turners, grinders, fettlers, steel and iron rolling mill workers and silver polishers. Welders, particularly, can be exposed to high levels of iron oxide dust and fumes. Typical occupation exposures are around 3000-50,000 (μm^3) (Flahive 2001; Lay et al. 2001).

Health effects

Exposure to iron ore dust can cause metal fume fever. This is a flu-like illness with symptoms of metallic taste, fever and chills, chest tightness and cough. Prolonged or repeated contact can discolour the eyes causing permanent iron staining. Repeated exposure might cause changes seen on a chest x-ray. Silica being a common constituent of iron ore dust, prolonged exposure might cause silicosis and other related lung diseases. Iron is an essential nutrient for both plants and animals. In some circumstances, marine systems, for example, iron might be in sufficiently short supply to limit plant production. At the other extreme, very high concentrations produced in rivers and lakes downstream from iron ore operations have been known to kill salmon roe and to have harmful effects on plankton and benthic organisms (Sirois & MacDonald 1983).

Animal Studies

The respirable fraction of iron ore dust from the North West of Western Australia was studied for biological properties by inhalation and intrapleural implantation trials using rats and mice. Pulmonary histology indicated significant levels of interstitial pneumonia, occasionally associated with bronchopneumonia, bronchiectasis, emphysema and lung collapse, over that found in age-matched control animals. While there was a significant increase of the incidence of tumours in general in WAG inbred rats for up to two years following dust exposure, this did not persist into old age. No mesotheliomas were induced by any treatments associated with iron ore dust, although the rats were shown to be susceptible to crocidolite-induced mesothelioma (Keast, Sheppard & Papadimitriou 1987).

Iron oxide appears to exhibit low toxicity in vivo compared to crystalline silica. Intratracheal instillation of 10mg/hamster crystalline silica resulted in recruitment of

PMN and elevation of RBC, LDH and albumin in lavage fluid, which was significant 1-day post exposure (Beck, Brain & Bohannon 1982), whereas the authors found that iron oxide caused much less inflammation and damage than silica, and that the pulmonary reactions were quickly resolved. A NIOSH study by Hubbs et al. (2001) with rats 30 days after instillation of 10mg/rat iron oxide where broncho-alveolar lavage markers of inflammation and damage were not different from control levels, and were significantly lower than values after silica sand instillation. Iron oxide was not found to induce sustained inflammation and lung damage or pulmonary fibrosis to rats (Hubbs et al. 2001). This might indicate that other components, probably silica, might be responsible for possible lung damage by iron ore dust.

The influence of the respirable fraction of iron ore dust on immunity has been investigated (Keast, Sheppard & Nguyen 1989). The iron ore dust enhanced the specific immunity responses both to sheep erythrocytes and bacterial lipopolysaccharides. The IgG class of antibody was enhanced significantly and its pattern of enhancement suggested that the iron ore dust was functioning as an adjuvant. The splenocytes, from animals that had received iron ore dust for various lengths of time, exhibited enhanced polyclonal mitogenic activity for phytohaemagglutinin *in vitro*. These properties were seen when the dust had been inhaled or injected into the pleural cavity and persisted for over one year following injection or inhalation of the dust. However, attempts to demonstrate the induction of interleukin-1 by macrophages from mice implanted intraperitoneally with the iron-ore dust were not successful and iron oxide, in contrast to silica, failed to induce significant secretion of inflammatory cytokines (TNF- α and IL-1) (Antonini et al. 1996).

Animal investigations of carcinogenicity of iron oxide have clearly been negative. Intratracheal instillation (380mg iron oxide

by multiple injections) resulted in no lung tumours in a 2.5-year study with rats (Steinhoff, Mohr & Hahnemann 1991). Similar results were reported by Saffiotti et al. (1968) after 15 weekly instillations of 3mg iron oxide each. Inhalation of iron oxide (40 mg/m³ for 2 years) also gave negative results in hamsters (Nettesheim, Creasia & Mitchell 1975). Therefore, the International Agency for Research on Cancer (IARC 1987) views evidence for the lack of carcinogenicity of iron oxide in animals as convincing.

Epidemiological Investigation

The relatively low fibrogenicity of iron oxide reported for animal models is generally consistent with epidemiological studies of exposed workers. For example, Teculescu and Albu (1973) studied 14 workers exposed for an average of 10 years to pure iron oxide dust and found no pulmonary function changes that would be consistent with pulmonary fibrosis. Lay et al. (1999) reported that the presence of inflammation following the intrapulmonary installation of iron oxide in human subjects, resolved rapidly after the exposure ended. Iron oxide particulates are also administered intravenously in humans as radiologic contrast agents.

Pulmonary siderosis is one kind of pneumoconiosis caused by the long-term inhalation of iron dust. It occurs in a number of occupations including steel rolling and grinding, welding, polishing, casting, boiler scaling, iron ore mining and emery working. Siderosis is considered a benign condition that resolves over a number of years once exposure stops. Siderosis is generally considered an overload, where the exposure to the dust is in excess of the lungs' ability to clear the particles (Flahive 2001). Yu et al. (1993) reported a case of pulmonary siderosis. A 49-year-old male who had worked in an iron foundry for 30 years was admitted because diffuse micronodular lesions were seen in both lung fields on a routine chest

radiographic study. A restrictive pulmonary function test disclosed a mildly ventilatory defect. A transbronchial lung biopsy revealed a significant amount of iron dust deposited within a fibrous nodule and diagnosis of pulmonary siderosis was confirmed.

Six males with systemic sclerosis were observed in the work forces of two iron ore mines (Martin et al. 1999). The usual spectrum of clinical features encountered in systemic sclerosis patients was present. Histological examination of pulmonary tissue was performed and showed features both of silicosis and scleroderma but to different degrees and stages of development. Exposure to high levels of silica-containing dusts had occurred in all cases.

Pavlenko et al. (1992) found the morbidity prognosis of silicosis and dust bronchitis, and cochlear neuritis among workers in the iron ore mines of the Krivoi Rog region. In Canada, joint medical and technical investigations of an iron ore mining and processing operation, over a two-year period has confirmed the presence of pneumoconiosis associated with respirable dust exposure above threshold limit values (Moore 1991). Vysochin et al. (1991) reported data on the 1961-1985 occupational morbidity rates in the workers engaged in iron-ore mines and discusses morbidity rates, and the structure and dynamics for different professions, age groups and duration of work. An analysis was given of the concomitant somatic diseases.

The mortality risk of iron ore (hematite) miners between 1970 and 1982 was investigated in workers from two mines in China. Among 490 underground miners who died, 205 (42%) died of silicosis and silicotuberculosis and 98 (20%) of cancer, including 29 cases (5.9%) of lung cancer (Chen et al. 1990). Early epidemiology studies reported an elevated incidence of lung cancer in workers exposed to iron oxide. Most of these studies are plagued with confounding exposures (Stokinger 1984).

For example, Boyd et al. (1970) reported a 70% higher than normal lung cancer mortality in underground iron-ore miners. However, surface iron mineworkers did not exhibit higher lung cancer mortality and underground miners were exposed to high radon levels. In addition, a case control study of factory workers exposed to high amounts of iron oxide dust found no excess cancer in the respiratory system or other sites (Axelson & Sjoberg 1979). Therefore, the human data for iron oxide-induced cancer are viewed as negative (Stokinger 1984). In contrast, a proportional mortality study of 1075 iron ore miners in Lorraine, France, who died between 1960 and 1976 showed a significant excess of lung cancer mortality (proportionate mortality ratio = 2.25). Moreover, proportionate lung cancer mortality increased with the duration of work underground and was higher among pneumoconiotic (siderotic) miners than among nonpneumoconiotic miners.

These results were confirmed by a case-control study nested in the mortality study. Although the proportion of smokers among contemporary iron ore miners was larger than in the French male population, occupational factors might play a role; radiation exposure can be ruled out because there was no detectable radioactivity in the Lorraine mines. Dust exposure could be considered as an etiologic factor owing to the relationship between siderosis and lung cancer (Mur et al. 1987).

Two independent observers for evidence of pneumoconiosis have examined plain chest radiographs of Pilbara iron-ore miners from Western Australia (Musk et al. 1988). Both readers observed the prevalence of radiographic abnormalities. A case-control study on employees at an iron ore surface mining plant was undertaken to determine which indices of occupational dust exposures related most closely to radiologic categories for pneumoconiosis. The association between dust composition and ILO radiologic category for simple pneumoconiosis was consistent, with

respirable quartz being the best differentiating index between the case and control groups (Moore et al. 1987).

Taconite is a low-grade iron ore consisting of iron, quartz, and numerous silicates. Clark et al. (1980) found taconite workers at risk of silicosis. Reichel et al. (1975) found that FeO (OH) is the most powerful fibrogenic inhibitory agent of quartz particles. This study was based on an X-ray survey of a group of iron ore miners.

Conclusion

It is important to keep updated information of health hazards associated with iron ore dust due to its variable composition. There are enough possibilities to have synergistic, antagonistic or additive effects of iron ore dust components in human exposures. However, silica, the predominant noxious agent present in most iron-ore dust might play a major role in risk of pulmonary

diseases and other lung function disorders, including lung cancer rather than iron oxide itself. While the size of particles derived from dusty trades such as mining usually examined range from 100-1000 nm, an interesting issue for toxicological research would be the interactions of nanoparticles (< 100nm) of iron. Nanoparticles of iron oxide have been used in lipstick, pharmaceuticals applied to the skin and in the delivery of vitamins and nanoparticles have been found to penetrate the epidermis.

Workers need to be examined periodically in order to determine early symptoms, particularly with lung functions. Routine environmental monitoring might be useful to protect or prevent the workers from occupational iron-ore dust exposure. The evaluation of the chemical composition of iron-ore dust might have more scientific impact in governing health hazards.

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Nitric Oxide: A Non-Invasive Measure of Silica Induced Health Effects and its Potential Role in Silica Induced Effects

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Although standards of industrial hygiene have improved markedly over the past 30 years, silica remains the major cause of occupational interstitial lung disease. The diagnosis of silica-induced diseases is mainly based on clinical findings, lung function and X-Ray examinations. None of these are sensitive to early stage disease or subtle progression. There is a need to find an alternative to remedy the above shortcomings. Great efforts have been made in investigations of exhaled nitric oxide (eNO) as a marker of various diseases and the possible causal pathways for NO to contribute to the diseases, whereas little research is available to elucidate the possibility of eNO as a non-invasive alternative to reflect silica-induced harmful effects. This review focuses on whether eNO can be used as a screening measure of silica-induced harmful effects and whether the role of NO in silica-induced lung reactions is at an initial stage. It is concluded that further research is warranted to testify the feasibility to use eNO and NO derivatives in sputum as a non-invasive screening tool for silica related disorders. In addition, better understanding of NO in silica-induced harmful effects might benefit both prevention and treatment of silica related occupational lung diseases.

Key words: Silica; Biomarkers; Lung Inflammation; Exhaled Air; Nitric Oxide; Silicosis

Although standards of industrial hygiene have improved markedly over the past 30 years, silica exposure remains a significant cause of occupational lung disease in Australia (National Occupational Health and Safety Commission 2003). Globally, it remains the major cause of occupational interstitial lung disease (World Health Organization 2000). Silica is frequently encountered in the workplace in many industries. The National Occupational Health and Safety Commission estimated in 1993 that the number of workers in Australia occupationally exposed to silica-bearing dust is about 136,000. Mining and manufacturing as well as construction industries, despite improvements in hygiene, remain significant sources of exposure and there are probably many smaller workplaces and self-employed individuals who are 'outside the loop' of workplace safety surveillance.

There is always a lag period between silica exposure and diagnosis of silica-induced diseases such as chronic obstructive pulmonary disease (COPD) and silicosis. The diagnoses of silica-induced diseases are mainly based on clinical findings, lung function and X-Ray examinations. None of these are ideally sensitive to early stage diseases or to subtle progression. Equally, although the diseases are poorly treatable at present, none of these modalities are likely to offer a means to measure the success of novel treatments. There is a need to find an alternative to remedy these shortcomings.

Nitric Oxide as a Potential Marker

It is noted that researchers are seeking new biomarkers that can be measured simply, non-invasively and repeatedly in exhaled breath, such as nitric oxide (NO), carbon monoxide and ammonia, to evaluate lung inflammatory diseases such as asthma, non-silica related COPD, cystic fibrosis and

interstitial lung diseases (Kharitonov et al. 2002). Extensive research has been conducted and progress has been made in examining the changed concentration of exhaled NO (eNO) and the potential role of NO in pulmonary inflammatory diseases. However, the exact mechanism for NO in silica-induced harmful effects is not yet completely understood, especially in regard to the role of this agent in the development of silica-induced inflammation and carcinogenesis. This paper focuses on the possibility of eNO as a measure of silica induced pathophysiological effects and the potential role of NO in these reactions, as a better understanding of NO in silica induced harmful effects might benefit both prevention and treatment of silica-related occupational lung diseases.

Silica-Induced NO Production in Animal Models

NO, a free radical produced by nitric oxide synthase (NOS), is an important signalling molecule that functions in many tissues to regulate a variety of physiological processes. Among three categories of isoform of NOS, namely neuronal NOS (nNOS), endothelial NOS (eNOS) and inducible NOS (iNOS), iNOS is considered the major contributor of NO in inflammatory reactions (Ricciardolo et al. 2004). In animal studies, silica induces overproduction of NO, represented by nitrite/nitrate, in the lung by bronchoalveolar lavage cell (BALC) populations, especially alveolar macrophages (AM) and neutrophils, which show early changes due to silica exposure before changes are detectable in lung tissue (Huffman et al. 2003). One day after intratracheal instillation of silica in rats, BALC (iNOS) mRNA steady-state levels were up-regulated 3-fold higher in silica groups than in the saline group. In contrast, steady-state levels of BALC iNOS mRNA from lavaged lung tissue of silica-treated rats were not significantly different from controls. The results clearly proved that silica instillation significantly increased

iNOS mRNA steady-state levels in BALC (Blackford et al. 1994). When these BALC were cultured, adherent cells enriched for AM and nonadherent cells enriched for neutrophils both exhibited significantly increased iNOS mRNA steady-state levels. Silica did not cause an increase in iNOS mRNA steady-state levels in lavaged lung tissue, suggesting that silica does not induce iNOS gene expression in nonlavagable parenchymal cells of the lung. In the same study, it was also shown that iNOS-dependent NO production, represented by nitrite/nitrate was increased in AM from silica-treated rats, but the authors did not measure NO production from neutrophils although the increase in the intracellular iNOS mRNA was demonstrated (Blackford et al. 1994).

In Vitro NO Production

It has also been shown that in vitro exposure of AM to silica does not stimulate NO production or enhance iNOS message. However, treatment of naive AM with conditioned medium from BALC harvested from silica-exposed rats does increase iNOS message and NO production by these AM. The potency of this conditioned medium is dependent on interaction between AM and neutrophils (Castranova et al. 1998). Direct in vitro exposure of rat AM to silica not only had an insignificant effect on NO production but also no additional effect on the increase of NO production induced by lipopolysaccharide and interferon- γ (Huffman et al. 1998). This might be an indication that silica-induced NO production is closely related to cell to cell interactions and its level change might be via a different pathway from NO production by other inflammation-inducing agents.

Potential Role of NO in Silicosis Development

Comparison of BALC from a single healthy volunteer, a silica-exposed coal miner with a normal chest radiograph, and a silica-

exposed miner with an abnormal chest radiograph showed a correlation between pathology and both the levels of iNOS messenger in BALC and the magnitude of NO-dependent chemiluminescence from AM, indicating enhanced NO expression in human pulmonary phagocytes and the potential effects of NO in silica-induced inflammation/fibrosis (Castranova et al. 1998). Studies investigating the effect of inhaled occupational dusts, including crystalline silica, on pulmonary reactions indicated that endogenous production of NO appeared in a time course that was similar to that of lung damage and inflammation. These findings strongly indicate that NO might play an important role in the initiation and progression of silicosis and pneumoconiosis (Castranova 2004; Castranova et al. 2002). Consistent evidence suggests the potential pro-inflammatory effects of NO in lung damage after silica exposure (Zeidler et al. 2004). However, NO is considered to have both pro-inflammatory and anti-inflammatory effects (Kharitonov et al. 2000). NO inhalation can be beneficial to a number of inflammatory pulmonary diseases and NO might play an anti-inflammatory role in the lung inflammation process (Kharitonov et al. 2000). Further study is needed to clarify the exact role of NO in silica-induced lung inflammation.

Potential mechanisms

NO is a potent activator of guanylate cyclase, the enzyme catalysing cyclic guanosine monophosphate (cGMP) synthesis. This suggests that a number of factors which can induce cGMP formation through a common mechanism involving release of NO. Formation of cGMP by guanylate cyclase is a common signal transduction pathway utilised by a diverse family of biological messengers. cGMP regulates a variety of enzymes and proteins and inappropriate formation of this nucleotide is likely to contribute to a number of pathological disorders (Zapol et al. 1997).

NO might also mediate a cytotoxic effect following its reaction with superoxide anion to yield peroxynitrite, which is a potent oxidant that can react with DNA, proteins and lipids leading to DNA damage and other toxic effects. Importantly, the host cells synthesising and releasing NO possess some inherent immunity against its toxic effects and thereby are not destroyed themselves (Zapol et al. 1997). Since inflammatory cells are the producers of NO in silica-induced inflammation, this might be an important pathway for inflammatory cells to cause injury of lung parenchyma cells. Immunostaining with a polyclonal antibody against nitrotyrosine indicated the formation of nitrotyrosine, a biomarker of peroxynitrite, in the granulomatous lesions of silica exposed rat lungs (Setoguchi et al. 1996). Peroxynitrite can be mutagenic (Tretyakova et al. 2000) and this might lead to the development of carcinoma (Yoshie et al. 1997). It is as yet unknown whether there is an increase in NO and peroxynitrite production in silica exposed human lung, although it has been demonstrated that iNOS-derived NO contributes to the pathogenesis of silica-induced lung disease in mouse models (Zeidler et al. 2004). It is noted that NO mediates a number of other physiological actions that are independent of cGMP and peroxynitrite. But, in silica induced pathological changes, NO-cGMP or peroxynitrite pathway may be much more important (Ricciardolo et al. 2004).

Change of exhaled NO

There is no direct evidence that the NO level changes in exhaled air of either silica-exposed workers or silicosis patients. However, NO levels change in a number of diseases and after exposure to a number of airborne pollutants. It has been shown that NO levels in exhaled air increases in children and adults with asthma (Kharitonov et al. 1996; Kroesbergen et al. 1999) and decreases in children with cystic fibrosis (Kroesbergen et al. 1999). It has also been shown that NO levels increase in

exhaled air in chronic obstructive pulmonary disease (COPD) patients during exacerbations (Agusti et al. 1999) and does not increase in stable COPD patients (Rutgers et al. 1999). In liver and kidney diseases, NO levels in exhaled air can also increase (Matsumoto et al. 1995; Matsumoto et al. 1999; Sogni et al. 1995). Moreover, smoking can also affect the level of NO in exhaled air.

Increased endogenous NO in exhaled air can also be attributed to outdoor air pollution (Steenenberg et al. 1999). Increased NO output has been reported in exhaled air of workers experiencing pot room dust exposures (Lund et al. 2000). Pulp mill workers with a history of peak ozone exposure were also reported to have an increased NO output in exhaled air (Olin et al. 1999). Another study conducted in children indicated that ambient ozone caused an acute inflammatory response from levels slightly lower than current air quality guidelines by using an exhaled NO test (Bernard et al. 2005).

Exhaled NO and its derivatives

NO output in exhaled air can originate from different parts of the respiratory system (Schedin et al. 1995). In a study of asthma, it has been shown that the elevated levels of exhaled NO in asthmatic patients are derived predominantly from the lower respiratory tract (Kharitonov et al. 1996). In a separate study, it has been suggested that the increase in exhaled NO in patients with allergic rhinitis is likely to be due to increased local production (Kharitonov et al. 1997). No matter what the origin of the exhaled NO is, to assess the production of NO comprehensively, the levels of nitrite and nitrate as well as peroxynitrite should also be measured because NO is not so stable that they can become nitrite/nitrate in an aqueous environment, or combine with superoxide to form peroxynitrite. Indeed, superoxide overproduction in silica-induced inflammation has already been well documented. Peroxynitrite formation in

silica-induced inflammation is quite likely. Therefore, one can assume that measurement of eNO, nitrite/nitrate and peroxynitrite reflects the whole picture of NO production and avoid the possibility of decreased eNO due to increased turnover from NO to derivatives nitrite/nitrate or to peroxynitrite leading to a false conclusion. A study has already suggested that in the lower airways significant amounts of NO are converted to nitrate and studies on NO in pulmonary diseases should therefore include determination of nitrate in lower airway fluids (Grasemann et al. 1997).

Induced sputum and NO derivatives

To collect airway fluid, either bronchoalveolar lavage (BAL) or sputum induction can be utilised. In asthmatic and COPD patients, it was found that the production of endogenous NO increased in both groups and the measurement of NO derivatives in induced sputum (IS) might be valuable in assessing airway inflammation in these patients (Kanazawa et al. 1998). Another study confirmed the usefulness of NO derivatives in monitoring the degree of inflammation in asthmatic subjects as the increased level of NO metabolites was paralleled with the severity of asthma (Jang et al. 1999). The same conclusion was also drawn in children with asthma (Kumar et al. 2005). In these studies, IS was preferred to evaluating the amount of NO derivatives due to its non-invasiveness against bronchoalveolar lavage fluid (BALF).

Conduct of IS can offer some other advantages over BALF because of not only non-invasiveness and measures of NO derivatives but also because of equal or even better representation of airway lining fluid. It has been shown that the analysis of induced sputum is a useful, non-invasive alternative to bronchoalveolar lavage for assessing the effects of anti-inflammatory drugs in asthma (Nocker et al. 2000). It has also been shown that IS has the advantage over BAL of higher density of cell recovery and stronger signal for fluid-phase markers

(Pizzichini et al. 1998). Through comparison, it is suggested that IS can be used to monitor the presence and severity of airway inflammation in asthma (Grootendorst et al. 1997). In the assessment of occupational exposure to hazardous dust, IS may also be a useful non-invasive technique (Fireman et al. 1999).

Screening of silica-induced disorders should include measures of markers in IS for a number of reasons. First, NO in exhaled air is not a specific indicator of lung conditions because its levels change in a number of diseases (Kharitonov et al. 1996). Second, it is also suggested that NO determination in exhaled air can be affected by some compounds derived from both endogenous and exogenous sources such as water vapour, carbon dioxide, acetone, heptane, acetonitrile, oxygen, nitrous oxide and enflurane (Binding et al. 2000). Therefore, it is important to be aware of the influences of some common confounding factors, for example smoking, in order to obtain unbiased and reliable results. Third, although both lavage fluid and sputum measurements are more direct reflections of airway inflammation than eNO, IS is non-invasive and simple. Moreover, it has been documented that silica exposure can cause changes of some cellular and molecular

markers in IS from silica exposed workers (Fireman et al. 1999). The sputum changes correlate with those in BALF and can be more sensitive in terms of molecular markers. The combination of NO in exhaled air and cellular or molecular markers (cytokines such as Tumour Necrosis Factor- α and Interleukin-1 β , small molecules in addition to NO derivatives) in IS may serve as a simple and sensitive alternative to assess the health of silica affected workers than BAL.

Conclusion

Animal studies have demonstrated that NO production changes after silica exposure, whereas there is no direct demonstration that eNO changes in silica-exposed workers. Importantly, because of the simplicity and non-invasiveness, the combined methods of eNO, and nitrite/nitrate and peroxynitrite in IS can be conducted repeatedly for follow up and hence exclude transit changes caused by non-occupational factors. The measures of eNO, nitrite/nitrate and peroxynitrite might help to elucidate the underlying mechanism of silica-induced effects and be beneficial in developing new therapeutic strategies via NO- related mechanisms to inhibit silica-induced harmful effects.

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The Role of Nitric Oxide or its Metabolites in the Development of Asbestos Induced Mesothelioma

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Asbestos exposure can lead to the development of malignant mesothelioma (MM) that is a fatal disease, which develops insidiously mainly after higher than background asbestos exposure. Nitric oxide (NO) production or NO synthase (NOS) expression was demonstrated in response to asbestos exposure and in the process of mesothelioma development in animal models and human subjects. NO is an important mediator and can be produced by three kinds of isoform of NOS enzymes. The role of NOS expression in mesothelioma development is still not clear. Since inducible nitric oxide synthase (iNOS) and endothelial nitric oxide synthase (eNOS) are both strongly expressed in human malignant mesothelioma, there might be a functional link between iNOS and eNOS. A number of inhibitors of NOS have already been widely used in animal studies. The inhibitors can inhibit the activity of NOS either selectively or indiscriminately. This review suggests that utilisation of different NOS inhibitors may provide valuable information to reveal the underlying mechanism of MM development.

Key words: Asbestos; Mesothelioma; Nitric Oxide; Nitric Oxide Synthases

Asbestos exposure can lead to a number of serious conditions, including pulmonary fibrosis, lung cancer and malignant mesothelioma (MM). MM is a fatal disease, which develops insidiously, mainly after higher than background asbestos exposure. Australia has maintained a total national MM case register since 1980 and there has been a marked increase in the incidence of this disease (Carbone et al. 1999; Leigh & Driscoll 2003). Indeed, the incidence rates in Australia are the highest reported national rates in the world so far and will continue to increase as predicted. Although it is well known that MM development can be attributed to asbestos exposure, the underlying mechanism for asbestos to induce malignant change is still not revealed. In addition to studies using human tumour samples (Kumar et al. 2005) and various cell lines (Fox et al. 2005; Fung et al. 1997), animal models such as rats (Hesterberg et al. 1998), mice (Altomare et al. 2005) and hamsters (McConnell et al. 1999) have also been used to observe the patho-physiological

changes in the development and evolution of asbestos-induced MM. Results of the animal model studies, although they cannot be directly extrapolated to humans, do provide valuable insights to understand the mechanism and hence facilitate future diagnosis and treatment.

In a number of experimental studies (Dorger et al. 2002; Puhakka 2005), nitric oxide (NO) production or NO synthase expression was demonstrated in response to asbestos exposure and in the process of mesothelioma development. In in vitro studies, increased iNOS mRNA after exposure to asbestos by alveolar macrophages was shown (Quinlan et al. 1998; Thomas et al. 1994a; Thomas et al. 1994b). Asbestos fibres upregulated the formation of nitrite, nitrotyrosine and iNOS protein expression in mesothelial cells (Choe et al. 1998). Asbestos inhalation can induce iNOS activation and peroxynitrite (a product of NO combination with superoxide) formation in vivo (Tanaka et al. 1998). Although a recent review indicated

that most researchers considered NO as a damaging endogenous agent (Zeidler & Castranova 2004), it is still controversial as to whether it is protective or damaging in asbestos-induced reactions.

NO Synthase

NO has been demonstrated to be an important mediator involved in a wide range of physiological and pathophysiological processes (Puhakka 2005; Ricciardolo et al. 2004). It is produced by three kinds of isoform of NOS enzymes, namely inducible NOS (iNOS), neuronal NOS (nNOS) and endothelial NOS (eNOS) (Ricciardolo et al. 2004). nNOS and eNOS are important in maintaining normal physiological functions and are expressed in normal activities. iNOS is not activated in normal status, but it does produce a large amount of NO in inflammatory reactions. Malignant mesothelioma cells are capable of synthesising iNOS but no significant association was found between iNOS expression and apoptosis or vascular density in malignant mesotheliomas (Soini et al. 2000). The role of iNOS expression in mesothelioma development is still not clear. iNOS (mainly soluble, cytokine -inducible; Ca^{2+} -independent) and nNOS are soluble and found predominantly in the cytosol, while eNOS is membrane associated. eNOS is mainly particulate and regulated by Ca^{2+} /CaM. Some researchers investigated the immunohistochemical expression of iNOS in healthy pleura and in malignant mesothelioma and a strong expression of iNOS in mesothelioma was shown (Puhakka 2005; Soini et al. 2000). eNOS is also strongly expressed in human malignant mesothelioma (Soini et al. 2001). Superoxide can also be produced by eNOS (Stroes et al. 1998) indicating a possible role of this enzyme in mesothelioma development. Interestingly, a study demonstrated that high concentrations of NO produced by iNOS can inhibit the activity of eNOS (Scott et al. 2002). Restoration of eNOS activity following

selective inhibition of iNOS can be achieved in a rat model of sepsis. This suggests that there might be a functional link between iNOS and eNOS.

Effects of NO

The toxicity of NO, the products of various NOS, and its metabolites, especially peroxynitrite, a potent toxic free radical, has been intensively investigated for better understanding of relevant mechanisms. NO and peroxynitrite might lead to DNA damage (Fellei-Bosco 1998). Although it has been reported that the cytotoxicity of NO and its metabolites outweighs the genotoxic effects (Stopper et al. 1999), most studies showed that NO and peroxynitrite are potent oxidants leading to direct DNA damage including DNA base deamination, DNA base and sugar oxidative modifications, oxidation of deoxyguanosine, peroxynitrite-DNA adducts formation, single strand breaks in DNA (Burney et al. 1999; Chao et al. 1996; Fellei-Bosco 1998; Upadhyay & Kamp 2003); and indirect damage, including the inhibition of various DNA repair processes (Graziewicz et al. 1996). For example, NO and peroxynitrite inhibit the DNA-adduct excision in nucleotide excision repair (Chien et al. 2004). NO can also interact with other molecules such as amines, thiols, and lipids (Fellei-Bosco 1998). NO enhances vascular endothelial growth factor (VEGF, governing angiogenesis) production by augmenting its expression in tumour cells (Dulak & Jozkowicz 2003). Some studies indicated that nitrogen species including NO can not only lead to DNA damage and mutations but also activate oncogene products and/or inactivate tumour suppressor proteins contributing to their carcinogenesis (Fellei-Bosco 1998; Ohshima 2003; Ohshima et al. 2003). Genetic instability and apoptosis can also occur with the involvement of NO (Janssen et al. 1998; Upadhyay & Kamp 2003). In an in vitro study, the presence of NO in cultured cells might either lead to the generation of another reactive, mutagenic species, such as peroxynitrite, or inhibit a DNA repair enzyme(s), leading to an increase in

mutations (Park & Aust 1998). However, a virus called SV40, a considered co-carcinogen of asbestos in the development of mesothelioma, by inhibiting the synthesis of NO, could favour the survival of transformed, potentially neoplastic cells (Aldieri et al. 2004). It appears difficult to elucidate the role of NO without consideration of NOS activities of different isozymes.

Attempts were also made to elucidate the role of NO in pathological processes in regard to its concentration. A study demonstrated that exhaled NO could increase in primary lung cancer patients and the increase might be attributed to inflammation in response to the developed cancer (Liu et al. 1998), although no direct role of NO in the development of cancer had been proposed (Chhatwal et al. 1996; Wink & Mitchell 1998). Reports published so far indicate a multiple and not fully understood role of NO in tumour initiation and progression (Stepnik 2002). Evidence is accumulating that sustained high levels of NO might have protective effects against carcinogenesis (Yerushalmi et al. 2006). In a relatively recent experimental study, it was demonstrated that induction of iNOS and NO production does not mediate but actually prevents tumour promotion and NO has both chemopreventive and tumoricidal effects (Dhar et al. 2003). Another study using Min mice also established the strong inhibitory effect of NO-releasing aspirin in intestinal carcinogenesis and suggested that this agent merits further evaluation as a chemopreventive agent against colon cancer (Williams et al. 2004). NO induces apoptosis in human breast cancer cell lines (Pervin et al. 2003), which may help inhibit cancer development. Actually, a hypothesis has been developed stating that a high concentration of NO prevents tumour development whereas low concentration promotes it (Jenkins et al. 1995; Xie & Huang 2003).

iNOS in animal models

Due to the lack of the iNOS pathway in hamster mononuclear phagocytes, an experiment was conducted to investigate

whether the lung iNOS expression on exposure to asbestos is different between rats and hamsters, and whether the differences in iNOS expression are related to the extent of pulmonary inflammation. Enhanced iNOS induction together with an acute inflammatory reaction in rats compared with no iNOS expression and a milder inflammation in hamsters indicates iNOS might play a key role in asbestos induced pulmonary injury (Dorger et al. 2002). It is still controversial whether NO is tumorigenic or antitumorigenic. Some researchers considered that the hamster was the best model of the animals studied for mimicking human lung antioxidant enzyme activities. Rat lung antioxidant enzyme activities were markedly different from any of the other species including humans examined (Bryan & Jenkinson 1987). The pulmonary reactions of rats and hamsters upon exposure to asbestos fibres are well known to be disparate. In comparison, instillation of asbestos fibres in hamsters resulted in a significantly milder inflammatory reaction of the lung with no induction of iNOS in pulmonary cells (Dorger et al. 2002). Mesotheliomas in hamsters are similar to those in humans in biologic behaviour (McConnell et al. 1999). After intratracheal instillation of crocidolite in male Syrian golden hamsters, besides lung carcinomas, a number of mesotheliomas of the epithelioid type occurred (Mohr et al. 1984). I.p. injection of glass fibres to rats and hamsters can induce mesothelioma (Pott et al. 1980) and this might be an indication that iNOS is less important in mesothelioma development. NO can increase the mutation induced by asbestos fibres in cultured hamster fibroblasts (Park & Aust 1998). Animals were exposed nose-only for 6 hours per day, 5 days per week, for 18 months (hamsters) or 24 months (rats) in inhalation studies for mesothelioma (Hesterberg et al. 1995). Asbestos exposure can induce iNOS activity and an increase in nitrotyrosine in rats' lungs and pleural fluid (Tanaka et al. 1998).

Inhibition of NOS

The administration of the iNOS inhibitor aminoguanidine (AMG) prior to insult reduces the severity of damage and improves mortality in thioacetamide-induced acute hepatic failure in rats (Rahman & Hodgson 2003). Treatment with (w-Nitro-L-Arginine Methyl Ester) L-NAME, a non-selective NOS inhibitor, elicited a chronic inflammatory response characterised by a persistent and extraordinarily severe accumulation of large numbers of inflammatory cells in the subcutaneous tissues, in muscle and in tendon. These findings indicate that in the case of injured tendon and synovium, NO could act to protect the healing tissue from an uncontrolled inflammatory response (Darmani et al. 2004). It is well known that high concentrations of NO produced by iNOS are associated with various inflammations. Inhibition of iNOS serves as a possibly novel experimental approach to treat inflammation. In a recent study (Kankuri et al. 2001), increased expression of iNOS protein and mRNA was found in acute chemical-induced colitis along with neutrophil infiltration, inflammatory oedema, and tissue damage in experimental rats. Subcutaneous injections of a novel highly selective iNOS inhibitor, N-[3-(aminomethyl)benzyl]acetamidine (1400W), (5 or 10 mg/kg t.i.d.) produced a 56 and 95% reduction in inflammatory oedema formation, a 68 and 63% reduction in neutrophil infiltration (measured as myeloperoxidase activity), and a 19 and 26% decrease in the size of mucosal lesions as compared with vehicle treatment. Administration of L-NAME (35 mg/kg) failed to produce any significant beneficial effects as compared with vehicle treatment in this experimental model of acute colitis. These results support the idea that selective iNOS inhibitors have promise in the treatment of colitis.

The Hypothesis

In asbestos-induced reactions, both asbestos fibres and asbestos-induced cytokines might act on pulmonary cells to produce NO. The iNOS can produce large amounts of NO and

superoxide in the initial inflammation. The high output might be a protective reaction. In the later stage, the NO production is low and this will be a harmful sign and can induce mesothelioma. iNOS and eNOS can work differently and play different roles in the initiation of mesothelioma. When iNOS becomes activated in the initial stages, they produce a large amount of NO, where eNOS might work later and produce a small amount of NO, which contributes to the development of mesothelioma. Inhibition of the enzymes might modify the reaction and the development of mesothelioma.

Further Studies

Based on the above review, the authors are proposing to establish rat and hamster models for asbestos-induced mesothelioma and examine the role of endothelial nitric oxide synthase and inducible nitric oxide synthase in the response to asbestos exposure and in the process of mesothelioma development.

Specifically, future studies should include the following four steps:

1. establishing animal models using rats and hamsters, and comparing the reactions to asbestos exposure in nitric oxide synthase activity of eNOS and iNOS.
2. using L-NAME, an inhibitor of all NO syntheses and L-N6-(1-iminoethyl)lysine (L-NIL), an iNOS inhibitor to intervene in the development of asbestos-induced reactions, including lung inflammation, lung cancer and mesothelioma development.
3. examining the changes of cell profile, cytokines and NO derivatives in pleural fluid of the animal models.
4. examining if inhibition of NO syntheses can modify the evolution of pathological changes or not, including inflammation, fibrosis, lung cancer and mesothelioma.

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Can Public Health Legislation Improve Health in Remote Aboriginal Communities in the Northern Territory?

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The potential for public health legislation to improve health outcomes for Indigenous Australians has been the focus of recent interest in Australia. This paper examines the Northern Territory's package of public health legislation in an attempt to assess its current and potential impact on health and to identify barriers to its ability to achieve better health for Indigenous people living in remote communities. It is argued that the current legislation in the Northern Territory has little or no practical application in health improvement. This is primarily due to the fact that its directive and regulatory approach is unsuited to dictating the social and environmental conditions that would lead to health improvements in Indigenous communities, or to generating the social change that would be required to initiate these improved living conditions. Proposed technical amendments to the legislation are thus unlikely to alter this lack of effect. Public health legislation does, however, have symbolic value and ensures that the health of Indigenous Australians remains on the political agenda. It may be that a more appropriate role for public health law in this context would be as an educative and non-directive structure, which aims to promote and protect health by supporting the attempts of Indigenous Australians to achieve reconciliation and self-determination.

Key words: Public Health; Legislation; Indigenous Australians; Remote Communities; Northern Territory; Environmental Health

It is widely accepted that the Australian Indigenous population experiences poorer health outcomes than both non-Indigenous Australians and the Indigenous populations of historically comparable nations such as New Zealand, Canada and the United States (Ross & Taylor 2002). The reasons for this are complex and encompass issues as diverse as living environment, education, poverty, dispossession from land and disempowerment (Royal Australasian College of Physicians [RACP] 1997). The recognition that health outcomes might be socially determined accords with the ecological approach to public health (Earls & Carlson 2001). This advocates the development of a multidisciplinary and integrated exploration of the biological mechanisms and social processes that influence health (Susser & Susser 1996). It also suggests that health improvement

programs need to look beyond the healthcare system for initiation and support (Jackson & Ward 1999).

One social mechanism that has a potential impact on health outcomes is the law (Hanney et al. 2003). Examples of this impact abound in public health law which provides structures within which outbreaks of infectious diseases might be contained (Senanayake & Ferson 2004), hazardous products might be regulated and, through the enforcement of safety standards, injuries might be reduced (Gostin 2000). In light of this success, the prospect of using public health legislation as a tool for improving Indigenous health outcomes is of interest, particularly in light of the perceived symbolic force of the law in bringing about the social change which would be required for Indigenous Australians to achieve self-determination (Garrow & Murray 1999).

Pursuant to section 51 of the Commonwealth Constitution, the power to make laws with respect to health is held by the States and Territories¹. As such, each Australian State and Territory has a package of laws that may be classified as public health legislation. In general, this package includes a Public Health Act and legislation relating to infectious diseases, water, hazardous waste, sewerage and sanitation, building safety, housing conditions and occupancy, food safety, injury prevention, the provision of essential services, such as electricity, and the creation of local government structures within whose power the enforcement of many of these Acts remain. Even a cursory glance at this subject matter reveals that it addresses many of the living conditions leading to poor health in Aboriginal communities, for example, limited or erratic water supply, poor water quality, inadequate sewerage and sanitation infrastructure, unhygienic food preparation and storage, and poorly maintained and overcrowded housing.

Given this legislative coverage of relevant subject matter it is perhaps surprising that the capacity of the law to affect Aboriginal health has not been more comprehensively evaluated. Only in Queensland has an organised analysis of the impact of public health legislation, in this case the *Health Act 1937* (Public Health Services 2001a), *Water Act 2000* (Public Health Services 2001b) and *Local Government Act 1993* (Public Health Services 2001c), in remote Indigenous communities been carried out. Of concern, the resultant Reports highlighted the cultural inappropriateness of many of the legislative provisions, an almost complete lack of enforcement in remote communities despite clear evidence of health hazards, and significant structural and operational weaknesses (Public Health Services 2001a,b,c). The conclusion was that, to date, all three legislative enactments have failed to affect positively health outcomes. However, while recommendations for legislative and

administrative change were made, the central assumption that public health legislation is an appropriate tool for improving Aboriginal health remained unquestioned.

In 2002, the National Public Health Partnership prepared a summary of all State and Territory public health laws of relevance to remote and Aboriginal and Torres Strait Islander communities (National Public Health Partnership 2002) and the Department of Health and Ageing commissioned a similar report focusing on the accountability of government agencies for the provision of environmental health services pursuant to public health legislation (Urbis Keys Young 2002). While both reports are valuable for presenting the public health legislation in an accessible format and raising questions about potential legal and practical impediments to the functioning of this legislation, the creation of strategies to resolve these barriers remained beyond their scope. Similarly, an assessment of whether the identified impediments were specific to the legislation as enacted, or whether they would be inherent in any attempt to improve Aboriginal health through the use of public health legislation, was not undertaken.

The purpose of this paper is twofold. The first is to examine critically the current and potential impact of the Northern Territory's public health legislation on the health of Aboriginal people who live in remote communities. The second is to consider whether or not public health legislation can ever be an effective tool for achieving measurable health improvements in Aboriginal communities.

To this end, the second part of the paper considers the applicability of the Northern Territory's public health legislation to remote communities on both Aboriginal land and Crown land. The next part of the paper then considers issues surrounding the enforcement of some specific provisions of the public health legislation to assess the capacity of this legislation to improve health

in the communities to which it applies. In the last part, the paper argues that when the complex historical, social and economic determinants of Aboriginal health are taken into account, it becomes apparent that legislation is both unable to dictate the conditions that would lead to health improvements in Aboriginal communities, and is too blunt an instrument to generate the social change required to initiate these. The paper concludes by raising the possibility that a more constructive role for public health law might be as a non-directive and educative structure, which focuses more on the promotion and protection of health than the enforcement of specific legal provisions.

Application of the Northern Territory's Public Health Legislation to Remote Aboriginal Communities

There are three primary forms of land ownership in the Northern Territory: private land, Crown land, and land granted under the *Aboriginal Land Rights (Northern Territory) Act 1976* (Cth) (hereafter referred to as 'Aboriginal land'). While there is no doubt that public health legislation applies to private land, much of the land in remote Aboriginal communities is either Aboriginal land or Crown land and the applicability of the legislation to these forms of land ownership is unclear. As such uncertainty constitutes a significant barrier to the potential use of legislation as a tool for improving health in Aboriginal communities, this issue warrants analysis.

Aboriginal land

The *Aboriginal Land Rights (Northern Territory) Act 1976* (Cth) (ALRA) is stated in its preamble to be "an Act providing for the granting of Traditional Aboriginal Land in the Northern Territory for the benefit of Aboriginals, and for other purposes". As such, it creates the mechanism by which Aboriginal people, through land trusts created for this purpose, could obtain

inalienable freehold title to unalienated Crown land of which they were traditional owners, and all Aboriginal reserves in the Northern Territory (Aboriginal and Torres Strait Islander Commission [ATSIC] 1998). By 2002, approximately 43% of land in the Northern Territory was Aboriginal land (National Public Health Partnership 2002).

The application of Northern Territory laws to Aboriginal land is dealt with in sections 71, 73 and 74 of ALRA. The general principle, elucidated in section 74, is:

This Act does not affect the application to Aboriginal land of a law of the Northern Territory to the extent that that law is capable of operating concurrently with this Act.

The prima facie test of applicability of a Northern Territory law, then, is consistency with the rights and obligations conferred by ALRA (House of Representatives Standing Committee on Aboriginal and Torres Strait Islander Affairs 1999). Of particular importance in this context is the right of Aboriginal people to use or occupy Aboriginal land in accordance with their traditions, broadly defined by the Act as including traditions, customs, beliefs and observances². The content of these traditions is considered to be flexible and is capable of changing over time. This right is set out in section 71(1) of ALRA:

...an Aboriginal or group of Aboriginals is entitled to enter upon Aboriginal land and use or occupy that land to the extent that that entry, occupation or use is in accordance with Aboriginal tradition governing the rights of that Aboriginal or group of Aboriginals with respect to that land, whether or not those rights are qualified as to place, time, circumstances, purpose, permission, or any other factor.

Section 71 thus narrows the legislative power of the Northern Territory in that it is unable to apply laws to Aboriginal land which conflict with or restrict these rights of traditional use, as defined over time. This limitation of legislative power is further reinforced by section 73, which confers

upon the Northern Territory government the power to enact laws only to the extent that these laws are able to operate concurrently with ALRA and any regulations made under it. It would seem, therefore, that the key determination to be made when considering whether a law of the Northern Territory applies to Aboriginal land, is whether it interferes with the rights of Aboriginal people to use and occupy that land in accordance with Aboriginal tradition (House of Representatives Standing Committee on Aboriginal and Torres Strait Islander Affairs 1999).

The primary consequence of this approach is that the determination of the applicability of Northern Territory laws to Aboriginal land must be carried out on an ad hoc basis. This is likely to be a cumbersome process as, while it is unlikely that entire Acts will be inconsistent with ALRA, multiple provisions within these Acts might be. For example, Regulation 12 of the *Public Health (General Sanitation, Mosquito Prevention, Rat Exclusion and Prevention) Regulations 1960* provides that an occupier must abate overcrowded living conditions when a Medical Officer of Health believes that they constitute a hazard to health. However, it might be that to reduce the number of occupants in this way would be contrary to Aboriginal notions of extended family. Similarly, some of the local government structures created by the *Local Government Act 1993* to oversee the provision of health services are likely to be incompatible with traditional Aboriginal forms of governance. Indeed, it can even be argued that the entire prescriptive nature of the Northern Territory's public health legislation, which relies on specific offences, is inconsistent with Aboriginal people's customs and beliefs regarding appropriate techniques for problem solving.

In addition, Aboriginal traditions are not static and vary both between communities and over time. Consequently, the same legal provision may be held to apply in one community but not in another. The result is

a public health legislative framework which is uncertain, potentially unpredictable and unduly resource intensive because every attempt to enforce the legislation is vulnerable to challenge on the grounds of applicability. The existence of such strong disincentives to utilise public health legislation is clearly a significant barrier to its ability to influence health outcomes for people living on Aboriginal land.

In 1998, a review of ALRA was carried out by John Reeves QC (the Reeves Report). This Report assumed that the purpose of public health legislation was to benefit the entire community, including Aboriginal people. By allowing this legislation to be inapplicable when it is inconsistent with Aboriginal traditional use of land, Reeves argued, Aboriginal people were failing to avail themselves of the opportunity to obtain health gains. His solution was to amend ALRA in two ways. First, he recommended that section 74 be repealed. This would effectively reverse the onus of proof so that Northern Territory laws would apply to Aboriginal land unless they could be shown, pursuant to section 109 of the Commonwealth Constitution³, to be directly inconsistent with ALRA (Reeves 1998). Second, he suggested that section 71 be amended to state that Northern Territory laws covering specific subject areas will apply to Aboriginal land, even if these laws are inconsistent with ALRA. The subject areas specified were public health and safety, the supply of essential services, environmental protection and conservation, and the maintenance of law and order. In an attempt to reduce the inevitable concern of Aboriginal people, Reeves added the qualification that "all reasonable steps shall be taken to minimise any negative effects on the use or occupation of the land" as a result of these changes (Reeves 1998, p. 412).

Occurring in the context of a Report whose recommendations would have significantly reduced meaningful Aboriginal control over Aboriginal land (House of Representatives Standing Committee on

Aboriginal and Torres Strait Islander Affairs 1999; Oxfam Community Aid Abroad 1999), these proposed amendments to ALRA were, not surprisingly, controversial. Although there was some commitment expressed by the Northern Territory Government to creating legislative certainty, many Aboriginal groups argued that, by focusing on the role of legislation, such an approach was misguided. In its submission to the House of Representatives Standing Committee that was reviewing the Reeves Report, the Central Land Council (CLC) stated that inconsistencies between Northern Territory legislation and traditional uses of land should be resolved by "consultation, negotiation and agreement" (Central Land Council 1999). Indeed, the CLC argued that to legislate so as to systematically subjugate Aboriginal traditional land uses to the legislative power of the Northern Territory government in this way would be to diminish the right of Aboriginal people to enjoy their culture to such an extent that it would breach Article 27 of the *International Covenant on Civil and Political Rights* (Central Land Council 1999).

Objection was also taken to the inference that, in asserting their traditional rights, Aboriginal people were depriving themselves of the benefit of public health legislation and were thus in some way to blame for their own poor health status. The provision of services for health, housing and education are the legitimate function of all governments, and the Northern Territory government should not be excused from its responsibilities by such a legal technicality. In the words of Oxfam Community Aid Abroad (1999) in its submission:

...The reality is that Aboriginal Territorians are entitled to Government services on an equal basis to any other citizen of the Northern Territory and should not have to sacrifice their hard won land rights in order to receive these services.

While this is an important point in its own right, it is also useful for its inference that public health legislation is not the only, or

even the most important, source of the government's responsibility for providing adequate health services, housing and education.

Ultimately, these recommendations were not adopted by the Commonwealth government and sections 71, 73 and 74 of ALRA remain unchanged (House of Representatives Standing Committee on Aboriginal and Torres Strait Islander Affairs 1999). It thus remains impossible to assess prospectively whether or not a particular legislative provision will be held to apply to Aboriginal land. From a legal perspective, this uncertainty of application and, in turn, reduced likelihood of implementation and enforcement, is a significant limitation to the ability of public health legislation to influence health outcomes in Aboriginal communities. However, the debate surrounding the recommendations of the Reeves Report was also valuable for questioning whether legislation can ever be an appropriate tool for improving Aboriginal health. This issue is discussed in more detail below.

Crown land

In its review of public health law in Australia, the National Public Health Partnership (2002) commented that one of the major limitations of the Northern Territory's public health legislation is that the *Public Health Act 1952* is not stated to bind the Crown. This raises significant doubts as to whether this Act, and therefore its regulations, can apply to Crown land and instrumentalities of the Crown in the Northern Territory. Other legislation which falls within the Northern Territory's package of public health laws, such as the *Water Act 1992*, *Water Supply and Sewerage Services Act 2000*, and the *Waste Management and Pollution Control Act 1998* are stated to bind the Crown both in right of the Northern Territory and the Commonwealth and thus clearly apply to Crown land and instrumentalities.

The relevance of whether or not a statute is stated to bind the Crown stems from the long established principle of statutory

construction that there exists a presumption of Crown immunity from statute. This presumption means that statutes do not apply to (or bind) the Crown unless they expressly or impliedly state their intention to do so (ARLC 2000). Commonwealth, State and Territory governments are all entitled to the protection afforded by this presumption (Taylor 2000) and thus, generally, statutes should indicate their intention to bind both the Crown of the enacting legislature (in this case the Northern Territory) and the Commonwealth (ALRC 2000).

Over the past 15 years there have been some significant changes to the test to be applied in order to assess whether a statute impliedly intends to bind the Crown. In 1990, the Australian High Court in *Bropho v Western Australia* (1990) 171 CLR 1 rejected the traditional narrow approach that only the terms of the statute could be considered in finding such an intention⁴. The majority of the Court in *Bropho* held that, in ascertaining whether or not there is an implied legislative intent to bind the Crown, all relevant factors should be examined. These factors include the subject matter of the legislation, its purpose, the identity of the Crown entity in question and the context and circumstances of the legislation's enactment.

It might thus be that an intention to bind the Crown should be implied into the *Public Health Act 1952*. Although the Act does not state a purpose, it is presumably to improve the health of the population as a whole. This is supported by section 10 which provides for the making of regulations in relation to, inter alia, the prevention of disease, the maintenance of health, sanitation, the care and treatment of sick persons, the promotion of public health, and measures for the control of diseases. Such a population-based approach to health would be significantly restricted if the Act did not apply to Crown land, or if agencies of the Crown were not subject to the same health standards as the rest of the population.

Further, there would seem to be no reason in logic or fairness why people on Crown land should not receive the same health protections as the rest of the population. Indeed, such an approach would reduce the effectiveness of the legislation significantly and raise important issues of equity. Interestingly, the conclusion that it should be implied that the *Public Health Act 1952* was intended to bind the Crown, is further supported by the fact that the draft new Northern Territory *Public Health Act* is expressly stated to bind the Crown.

It is thus arguable that the Northern Territory's *Public Health Act*, and associated regulations, would be held to bind the Crown, at least in the Northern Territory⁵. This means that the Act probably does apply to Crown land and that Northern Territory Crown agencies should be bound by it. However, in the absence of a specific judicial determination to this effect, the application of the Act remains uncertain and potentially subject to legal challenge. Once again, this acts as a disincentive to its implementation and enforcement and thus reduces its capacity to achieve its aim of improving the health of the population.

Implementation and Enforcement of the Northern Territory's Public Health Legislation in Remote Aboriginal Communities

To date, the only comprehensive evaluation of the effectiveness of public health legislation in improving the health of Aboriginal people has been conducted by Queensland Health's Public Health Law and Indigenous Health Project (Public Health Services, 2001a,b,c). It found that, despite evidence of significant environmental health problems, the relevant provisions of the *Health Act 1937* were rarely enforced (Public Health Services 2001a). This failure was attributed largely to the fact that the Aboriginal and Torres Strait Island councils, who comprised the local government structures for the communities being

evaluated, were unable to fund an adequate environmental health workforce to implement and enforce the legislation (Public Health Services 2001a). This implies a belief that with increased funding, the legislation would be more effective.

In fact, the practical barriers to the capacity of legislation to improve health are more complicated than this suggests. The Northern Territory's public health legislation, like that in Queensland, is prescriptive and comprises multiple specific offences. Thus statutory powers are granted to Health Departments, Health Officers and Municipal Councils to compel owners and occupiers of land to abate health risks on their properties, to condemn premises which are hazardous to either health or safety, and to regulate the hygiene standards of community stores (Urbis Keys Young 2002). A typical example of such a provision is section 7C(1) of the *Public Health Act 1952* (NT) which states:

Where, in the opinion of the Chief Health Officer, an owner or occupier of land has committed an offence against this Act or the Regulations which, in the opinion of the Chief Health Officer, causes or may cause a risk to public health, the Chief Health Officer may...by notice in writing, require the owner or occupier of the land to cause the risk to be removed within such time as he specifies in the notice.

Sections 7C(2) and (3) go on to confer power on the Chief Health Officer to organise for the work to be done if the owner or occupier refuses, and to recover the cost from that owner or occupier.

While this approach may be appropriate for the regulation of privately owned premises located in an urban area, it is unlikely to be feasible in impoverished remote Aboriginal communities. A written notice of abatement of risk is of limited practical relevance to an Aboriginal person living in a remote community who neither reads nor writes English, who is unable to afford the repairs even if a contractor could be found, and for whom a Western regulatory framework is quite foreign. Closing a community store that

does not strictly adhere to food hygiene standards is not a realistic option if it is the only store in the community. By ignoring the underlying issues of poor food security, lack of knowledge concerning traditional food sources and the loss of role models for food preparation within communities, it is also a grossly inadequate response. Similarly, condemning an inadequate house is of no value if the occupying family does not have access to alternative accommodation. The lack of legislative implementation and enforcement observed in Queensland, which no doubt would be equally striking in the Northern Territory, thus does not, as has been suggested, stem primarily from an inadequate workforce. Rather, it is due largely to the inappropriateness of the attempt to impose the current regulatory scheme onto remote Aboriginal communities (National Public Health Partnership 2002; Urbis Keys Young 2002). Further, as explored above, the uncertain applicability of public health legislation to Aboriginal communities further reduces the incentive for recourse to such measures.

The practical consequences of living in remote areas should also not be underestimated. Despite the fact that there are over 680 discrete Aboriginal communities in the Northern Territory (Australian Bureau of Statistics 2001), the *Power and Water Corporation Act 2002* has been interpreted as only requiring the Corporation to supply electricity to urban areas. Although this has been tempered by a Commonwealth-Territory Agreement, even this only requires the Corporation to supply 80 of the largest Aboriginal communities Territory-wide (National Public Health Partnership 2002). Similarly, the *Building Act 1993*, based on the *Commonwealth Building Code*, which establishes standards for the building and construction industry, applies only to gazetted areas. Virtually all remote Aboriginal communities fall outside its scope.

In order to compensate for this failure of public health legislation to protect people living in Aboriginal communities, the

Northern Territory Government developed a set of environmental health standards for remote communities (Northern Territory Government Environmental Health Task Group 2001). While these are non-binding, they are based on notions of the importance of information, advice, advocacy and practical support (Urbis Keys Young 2002), and are arguably more appropriate than the theoretically enforceable but practically limited provisions of the major public health statutes.

The legal and practical barriers to the effectiveness of legislation in remote Aboriginal communities render it unlikely that the Northern Territory's current package of public health legislation is capable of improving health outcomes in these communities. It is thus vital that their existence, combined with the success of public health legislation in other contexts, does not lead to complacency and a belief that progress is being made when it is not.

The Appropriateness of Public Health Legislation as a Tool for Improving Health in Remote Aboriginal Communities

The aim of the first evaluation of public health legislation that focused on Aboriginal communities was to inform the Legislation Reform Working Group of the National Public Health Partnership (Bidmeade & Reynolds 1997). It is thus not surprising that when the legislation's effectiveness was questioned in the evaluation, the response was to recommend legislative reform, although the need for more fundamental responses such as empowerment through land, employment and resources were also highlighted (Bidmeade & Reynolds 1997). Nevertheless, the language of legislation has also predominated in subsequent recommendations with talk of "improved compliance" with statutory standards, the importance of "certainty in the application of legislation", the "modernisation of public health laws", and the need to support local

government structures in their attempts to "maintain public health standards" (National Public Health Partnership 2002). Indeed, even recommendations for the development of an adequate environmental health workforce have focused on its potential contribution to the operation of public health law. Rather than promoting environmental health officers as people who can liaise with communities and provide information, advocacy and support, they have been seen as the key to "law enforcement strategies" (Public Health Services 2001a,b,c). As mentioned earlier, such an approach unduly simplifies the issues surrounding the poor health outcomes of Aboriginal people in remote communities.

This is not to argue that there have been no attempts to adapt public health legislation to Aboriginal traditions, customs and beliefs. For example, the Queensland Public Health Law and Indigenous Health Project recognised the inappropriateness of the terms 'owner' and 'occupier' which are widespread throughout Australia's public health law (Public Health Services 2001a). However, their suggested response was merely to change the language of the statutes so as to take account of the various different types of ownership and occupation of land in Aboriginal communities (Public Health Services 2001a). The appropriateness of laws creating statutory offences, which are committed by individual owners and occupiers in communities, regardless of how they are labelled, appears to have been accepted without question.

Indeed, a review of the recommendations for legislative amendment made by the Queensland Public Health Law and Indigenous Health Project (2001) and the National Public Health Partnership (2002) reveals that there exists support for broadening the application of public health legislation to Aboriginal land and for developing complex legal and governance frameworks to underpin the implementation and enforcement of these statutes. Further,

although proposals for preambles and statements of objectives, which both emphasise the population based aims of the legislation and place it within a social context, have been popular^a, the proposed legislative content remains largely regulatory and is based on specific statutory offences, usually committed by an individual. The legislation would thus continue to function by requiring individual Aboriginal people to undertake specific actions in their capacity as 'owners' and 'occupiers' and a failure to comply with these requirements would continue to attract penalty, irrespective of the reasons for this failure. Barriers to action such as poverty, lack of access to resources and services, workforce limitations and cultural inappropriateness would remain no defence to these legislative offences.

Interestingly, statutes that impose on government the responsibilities for essential service provision take quite a different approach (Centre for Comparative Constitutional Studies University of Melbourne 1999). For example, both the *Power and Water Corporation Act 2002* and the *Building Act 1993* remain free to exclude remote Aboriginal communities from their scope on the basis of difficulties of access, resources and workforce. Indeed, rather than create enforceable service provision requirements which attract penalties for non-compliance, governments have taken a broad approach and have addressed their legitimate responsibilities through the creation of non-legislative standards, guidelines and policies. Examples include the *Environmental Health Standards for Remote Communities in the Northern Territory* (2001), the *National Framework for the Design, Construction and Maintenance of Indigenous Housing*, and the Northern Territory policy document *Building Healthier Communities: A Framework for Health and Community Services 2004-2009*.

These documents are promoted as being consultative, flexible, culturally appropriate and comprehensive. Some, such as the *Environmental Health Standards for Remote*

Communities in the Northern Territory (2001), explicitly acknowledge the Northern Territory Government's responsibility to ensure that environmental health problems are minimised. Indeed, this document concedes that the ability to access essential services and a healthy living environment are basic human rights (Northern Territory Government Environmental Health Task Group 2001). The symbolic powers of these standards are considerable and they provide valuable guidance for those developing interventions. It thus seems incongruous that while the Government has recognised that strict legal requirements for service provision are inappropriate and unable to be enforced, it has not similarly recognised the limitations of imposing regulatory public health legislation on Aboriginal people in remote communities.

This approach seems to place the legal responsibility for health on Aboriginal people rather than governments (Garrow & Murray 1999). Public health law is currently a collection of legislative offences which suggest that if Aboriginal people kept their houses cleaner, lived in less overcrowded conditions and disposed of their waste in a 'safe' manner, they would be healthier. Such a view disregards the complex historical and social determinants of Aboriginal health and, in addition, downplays the government's legitimate service provision function. Further, it forces Aboriginal people to participate in a legal and governance framework within which decisions may be made very differently from their methods of problem solving. Even more disturbingly, in its focus on creating 'offences', it seeks to achieve health gains by punishing the people it claims to protect. In the light of this, it is little wonder that the debate surrounding the Reeves Report revealed such a widespread suspicion and distrust of legal mechanisms by Aboriginal people (Central Land Council 1999; Oxfam Community Aid Abroad 1999).

A simplistic and rigid legislative response to public health in Aboriginal communities

is thus inappropriate. Health reform cannot stand alone. Rather, it must be integrated with broad government strategies which recognise the overlap between health and education, the supply of environmental health infrastructure, socio-economic development and the empowerment of Aboriginal people through issues relating to land rights and reconciliation (Anderson 2002). The improvement of Aboriginal health requires social change, which in turn requires the creation of a process for consultation, discussion, negotiation and understanding. A legislative framework built of regulatory offences, such as the current package of public health legislation in the Northern Territory, is inherently incapable of producing or supporting such a process. Indeed, its techniques are, by definition, opposed to it.

Conclusion

The failure of public health legislation to improve health in Aboriginal communities is not due simply to uncertainty as to its application or a lack of resources to ensure its implementation and enforcement. Rather, the failure results from a directive and regulatory approach to public health legislation which appears unsuited to generating the social change which is required to address the issues surrounding Aboriginal health. Legislative amendments that clarify legal requirements, improve

terminology and enhance mechanisms for law enforcement are unlikely to alter this situation and bring about measurable health improvements. Under such circumstances it is difficult to escape the conclusion that not only is public health legislation currently failing to improve Aboriginal health, it is by its nature unable to either dictate the conditions which would lead to health improvements in Aboriginal communities or generate the social change required to initiate these.

The law can, however, have a significant symbolic force (Garrow & Murray 1999; Territory Health Services 1997) and debates concerning legislative amendments can keep Aboriginal health firmly on the political agenda. Further, documents such as the *Environmental Health Standards for Remote Communities in the Northern Territory* (2001) can be useful tools for initiating constructive dialogue between Indigenous and non-Indigenous people. This raises the question of whether a more appropriate role for public health law would be as an educative and non-directive structure which aims to promote and protect health by supporting the attempts of Aboriginal people to achieve reconciliation and self-determination (Garrow & Murray 1999), perhaps through the utilisation of human rights discourse.

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Endnotes

1. Section 51 of the Commonwealth Constitution sets out the legislative powers of the Commonwealth. Subject matter not specified in this section remain the legislative domain of the States and Territories.
2. *Aboriginal Land Rights (Northern Territory) Act 1976* (Cth) Section 3.
3. Section 109 of the Constitution states that when there is an inconsistency between a Commonwealth and State or Territory law, the latter is invalid to the extent of the inconsistency.

4. The traditional narrow approach was set out in *Bombay v Municipal Corporation of Bombay* [1947] AC 58.
5. A detailed examination of the controversy surrounding the application of the presumption of Crown immunity in the federal context is beyond the scope of this paper (see Taylor 2000).
6. This is evidenced by the increasing tendency of legislation to have a general statement of purpose in these terms. See, for example, the *Waste Management and Pollution Control Act 1998* (NT) Section 5 and the Preamble to the *Housing Assistance Act 1996* (Cth).

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Legislation

Commonwealth

Constitution of the Commonwealth of Australia
Aboriginal Land Rights (Northern Territory) Act 1976
Housing Assistance Act 1996

Northern Territory

Building Act 1993
Local Government Act 1993
Power and Water Corporation Act 2002
Public Health Act 1952
Public Health (General Sanitation, Mosquito Prevention, Rat Exclusion and Prevention) Regulations 1960
Water Act 1992
Water Supply and Sewerage Services Act 2000
Waste Management and Pollution Control Act 1998

Queensland

Health Act 1937
Local Government Act 1993
Water Act 2000

Cases

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Sun Protection Policies and Practices of Sporting and Recreation Organisations and Clubs in Queensland

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There are many public health benefits of participation in outdoor sporting activities. However, extended exposure to the sun is a significant risk factor for skin cancer. As there is limited information available on the factors that facilitate the implementation of sun protection measures and policies within sporting and recreation organisations, a study was conducted to investigate this in Queensland. This study was exploratory in nature and utilised a cross sectional design involving a telephone survey methodology. The study participants were chosen from state sporting and recreational associations to provide an insight into sun protection activities undertaken by their affiliated clubs. The study results indicate that these state associations were active in promoting sun protection to their clubs, that the clubs with sun protection policies tended to have a more comprehensive approach to sun protection, but that additional sun protection measures are needed at a club level. Consequently, it is important to continue encouraging local clubs to adopt sun protection policies and this appears more likely with the assistance of the state sporting and recreational associations.

Key words: *Sun Protection Measures; Sun Protection Policies; Sporting and Recreational Associations; Sporting and Recreational Clubs*

Organised sporting and recreational activities provide a significant setting in which to undertake health promotion and public health prevention programs. In fact, the latest data from the Australian Bureau of Statistics (ABS) indicate that an estimated 4.3 million people aged over 15 years or 27% of the Australian population are involved in organised sport and physical activity (ABS 2005), and 1.6 million or 62% of children aged between 5 and 14 years participate in organised sport (ABS 2004). Participation in sporting activities is integral to the 'Australian way of life' and provides many health-related benefits including physical activity and community cohesion (Fletcher et al. 1996; Nelson & Gordon-Larsen 2006; Warburton, Whitney Nicol & Bredin 2006). In addition, the Australian climate encourages many of the activities to be undertaken outdoors, with ABS data confirming that for children, all of the most

popular sports are played outdoors. In decreasing order of participation these are for boys - outdoor soccer, swimming, Australian Rules football, tennis, outdoor cricket and basketball; and for girls - netball, swimming, tennis and basketball (ABS 2004).

Despite the positive benefits of participation in outdoor sporting activities, extended exposure to the sun is a significant risk factor for skin cancer in the community (Kricker et al. 1994; Rosso et al. 1996), with recreational sun exposure a particular risk factor for melanoma and basal cell carcinoma (BCC) (Armstrong & Kricker 2001). In addition, exposure to UVR during childhood and adolescence, particularly a history of sunburn, plays a role in the future development of both melanoma and BCC (Gallagher et al. 1995; Kricker et al. 1995; Westerdahl et al. 1994; Whiteman et al. 2001). For Queensland, skin cancer

prevention is a significant public health priority (Queensland Health 2001). This is due to Queensland having the highest rates both of melanoma and non-melanoma skin cancer (i.e. BCC and squamous cell carcinoma) in the world (AIHW 2004; Queensland Cancer Registry 2000), and because non-melanoma skin cancers are the most expensive cancer in Australia to treat (AIHW 2005).

As thousands of people join sporting organisations each year, sporting associations and clubs are in a position to play an important role in promoting sun safe behaviour thus protecting both participants and spectators from skin cancer (Queensland Cancer Fund 2005). To help community based sporting and recreation organisations manage this risk, there is a range of sun protection strategies that can be implemented. Formalising sun protection strategies within the policy framework of an organisation is considered necessary to ensure the sustainability of these strategies.

A recent report from the Task Force on Community Preventive Services in the US confirmed that education and policy approaches that focused on increasing sun-protective behaviours were effective when implemented in these recreational settings (Centers for Disease Control and Prevention 2003; Saraiya et al. 2004). To assist sporting organisations with this task, there are a number of model policies available for use, with the Queensland Cancer Fund (QCF) *SunSmart Policy for Sporting Organisations* (QCF 2004) being comprehensive and widely adopted.

Despite the availability of model policies, there is limited information on the level of and barriers to implementation of sun protection policies and practices by sporting and recreation organisations. A recent survey of sporting clubs in Victoria, found that only 34% of clubs had a sun protection policy, although policies were more prevalent among clubs that competed in summer. This study also found that having a designated person responsible for policy at

the club was significantly associated with the club having a sun protection policy, with barriers to developing and implementing policies generally related to a lack of resources, advice and training, and a lack of control over facilities and other club priorities (Dobbinson & Hayman 2002). In the absence of similar information available for Queensland, the study reported in this article was conducted into the adoption and implementation of sun protection policies and measures within sporting and recreation organisations. The study was divided into two main sections: the first section focused on sun protection policy adoption and implementation at the association and club levels; and the second was framed around the contents of the current QCF SunSmart model policy, with the specific purpose of determining the level of club compliance in terms of meeting the QCF policy requirements.

Methods

A cross sectional design was utilised for this study (Morton, Hebel & McCarter 1990; Protney & Watkins 1993) with data collected via a telephone survey using convenience sampling (Portney & Watkins 1993; Streiner, Norman & Munroe-Blum 1989). As the study was exploratory in nature no formal hypothesis testing was undertaken. The results have been tabulated to show actual counts and percentages.

Participant Selection

The target population for this study was key personnel within Queensland state sporting bodies or associations. The study participants were recruited from the Sport and Recreation Queensland database (Sport and Recreation Queensland 2004) and were selected on the basis of the nature of activities undertaken (a) outdoors and (b) during daylight hours, for example, cricket and golf as compared to indoor figure skating or indoor rock climbing.

At the time of the study, the database contained details of 103 sporting and

recreation organisations that ran activities themselves or represented clubs that provided a range of sporting and recreational activities. Based on the above criteria, 58 organisations were considered to be potential participants and of these, a total of 35 (60.5%) participated in the study. Those that elected not to participate did so either because they did not consider the study to be relevant to them (8.5%; $n=5$), they were not contactable for the interview (27.5%; $n=16$), or did not wish to participate (3.5%; $n=2$).

Data Collection

A survey instrument was developed to explore: (a) background information on the respondents and their organisations, (b) sun protection policies, (c) actions undertaken at both the association and club level, and (d) ideas for improving sun protection for this setting. The instrument was pilot tested and modified prior to being used in the study. An expert group composed of health promotion practitioners, researchers and epidemiologists contributed to the instrument development. The questionnaire was designed for completion in 20 to 40 minutes and contained a combination of closed and open-ended questions.

Telephone interviews were undertaken between the 1st and 31st of March, 2004. To maximise participation each association was contacted prior to the interview in order to: (a) identify the most appropriate representative/s, and (b) determine a convenient time to conduct the interview. Interviews were conducted by a single interviewer who was an experienced health promotion practitioner.

The questionnaire comprised three sections. The first section related to background information on the representative and the organisation. This section was used to collect data on the representative's position within the organisation and duration of their tenure, size of the organisation, number of members, age groups, owners of the facilities, seasons

when the sport is played and timing of training and fixtures. The second section focused on the association's approach to sun protection practices and included questions on the prioritisation, policies and influences on the sun protection policy and practice of affiliated clubs. The third section sought information on the affiliated clubs and included questions on the priority placed on sun protection and strategies utilised at the club level.

Results

Study Participants

It was considered important that the representatives chosen to participate in the study had good knowledge of their organisations. The study participants were therefore selected on the basis of their key positions (chief executive officers to state development officers) and duration of tenure, with the majority having been in their position for three or more years (60.0%; $n=21$).

Ownership of facilities has an obvious impact on the level of modification or addition that can be made for sun protection. This survey found that 80% ($n=28$) of the grounds and facilities used by organisations in the study were owned by local government. The remaining facilities were under the ownership of the state, non-government organisations or the sporting clubs themselves.

Timing of Activities

In terms of seasonality of club activities, the survey results indicated that:

- all of the clubs operated in autumn and spring;
- more than two thirds of the clubs operated in summer (69.0%; $n=24$); and
- over half of the clubs operated all year round (54.0%; $n=19$).

In terms of the timing of fixtures and training sessions, the study participants reported that:

- almost all of their clubs held fixtures during the daylight hours (94.0%; $n=33$), with 89% ($n=31$) of these fixtures held during the peak ultraviolet radiation (UVR) times (10am to 3.00pm); and
- almost all of their clubs conducted training sessions during daylight hours (97.0%; $n=34$), with half of these sessions being held during the peak UVR times (50.0%; $n=17$).

Notably, all of the clubs operating during the summer months reported holding their fixtures during daylight hours (100.0%; $n=24$), with the majority of these being in the peak UVR times (87.5%; $n=21$). In regard to training sessions, nearly all of these clubs conducted training during daylight hours (96.0%; $n=23$), with half of these sessions held during the peak UVR times (50.0%; $n=12$).

Associations' involvement with affiliated clubs

Eighty-six percent ($n=30$) of the associations in the study were directly involved in the operation of their affiliated clubs. This involvement included contributing to policy development, training, officiating, accreditation programs, insurance and risk management. Eighty percent ($n=28$) of the associations actively encouraged their clubs to implement sun protection measures. This included the provision of sun protection resources, mail outs and presentations that included sun protection messages, and finally through the direct promotion of sun protection policies.

Prioritisation of sun protection

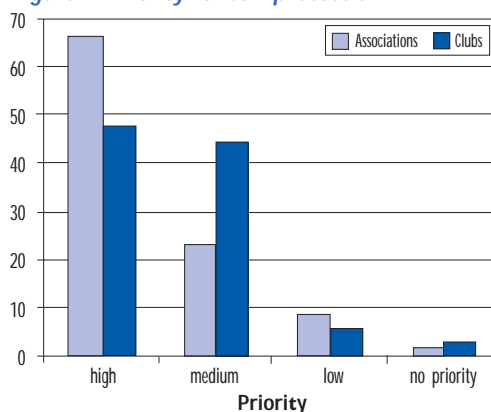
The study participants were asked to rank the priority placed on sun protection within their organisations (no priority, low priority, medium priority, high priority). Sixty-six

percent ($n=23$) of the study participants considered that their organisation placed a high priority on sun protection. The most common reason given for this priority was in concern for the safety of their members and the general public (60.0%; $n=21$).

Only 11.5% ($n=4$) considered sun protection to be a low or no priority for their organisations (refer to Figure 1). The reasons given for this response were: pressures of other priorities; an expectation for people to self-manage; a limited amount of activities were outdoors resulting in a reduction in priority; and finally, the implementation of a temperature related policy (ie. cancellation of fixtures in extreme heat conditions).

The study participants were asked to rank the priority given to sun protection in their affiliated clubs (no priority, low priority, medium priority, high priority). According to the study participants, 46.0% ($n=16$) of the affiliated clubs considered sun protection a high priority, 43.0% ($n=15$) as a medium priority and 9.0% ($n=3$) as a low or no priority with one participant not providing a response (3.0%; $n=1$) (Figure 1). As mentioned previously, the most common reason given for the higher priorities was the safety of the club members (60.0%; $n=21$).

Figure 1: Priority for sun protection



Sun protection policies

More than half of the participating associations reported having developed and adopted a sun protection policy (57.0%; $n=19$). These policies were subsequently

supplied to the research team for verification. However, only three quarters of these associations indicated that the policies should undergo a regular review process (74.0%; $n=14$).

Thirty-nine percent of study participants ($n=13$) believed that the majority ('all' or 'most') of their affiliated clubs had sun protection policies. Eighteen percent ($n=6$) of the study participants indicated that only some of their affiliated clubs had policies while a further 18.0% ($n=6$) were certain that none of their clubs had policies. The remainder of the study participants (25% $n=9$) did not know if their clubs had a sun protection policy.

From the 19 (54.0%) study participants that reported either 'all', 'most' or 'some' of their affiliated clubs had adopted sun protection policies:

- 63.0% ($n=12$) reported that the policy adopted by their affiliated clubs was the QCF model policy;
- 68.0% ($n=13$) reported that these policies were being used by the clubs; and
- 37.0% ($n=7$) reported that their clubs had a policy review process.

When asked to identify who initiated the policy adoption process within their clubs, 29.0% ($n=10$) of respondents nominated the associations, while 17.0% ($n=6$) nominated the clubs themselves and a further 8.6% ($n=3$) identified joint action between the association and the clubs).

There was an apparent link between the associations and their affiliated clubs having sun protection policies, with 21.0% ($n=10$) of the study participants reporting that the association and the majority ('all' or 'most') of the affiliated clubs had sun protection policies. This compared to one study participant reporting that most of their affiliated clubs had sun protection policies while their association did not.

Sun protection measures at club level

Questions pertaining to sun protection measures at club level were based on the 11 recommended sun protection measures from QCF SunSmart model policy for sporting clubs (QCF 2004). The study participants were asked to provide an indication of how many affiliated clubs were using each of these measures (no clubs, some clubs, most clubs, all clubs). As the QCF model policy was considered to represent good practice for this study, this information provided an insight into the level of compliance within the clubs.

Of the strategies identified in the QCF policy, the most common sun protection measures reported by the study participants were: (a) expecting officials and members to wear sunscreen, (b) parents/guardians supplying their children with sun screen, and (c) providing shade for spectators. The least used strategies were (a) having sunscreen for sale, (b) recommending only SPF 30+ sunscreens, and (c) providing sunscreens as part of the club membership (Table 1).

There appeared to be an association between the existence of a sun protection policy and the number of sun protection measures used at the club level. Based on the observations provided by the study participants, the clubs with sun protection policies (57.0%; $n=19$) utilised between five and eight sun protection measures from the QCF model policy, while the clubs that did not have sun protection policies (43.0%; $n=15$) were reported to be using only between one and five of the sun protection measures identified in the QCF model policy.

Improving sun protection

The overwhelming majority of study participants (91.5%; $n=32$) considered it would be beneficial to improve sun protection at the club level. Fifty-four percent (54.0%; $n=19$) of the study participants believed that increased funding would be helpful in enabling clubs to purchase shade structures (54.5%; $n=12$),

Table 1: QCF sun protection measures used by affiliated clubs

Sun protection measures identified within the QCF sun protection model policy	Clubs utilising recommended sun protection measures				
	Respondents who reported all clubs	Respondents who reported most clubs	Respondents who reported some clubs	respondents who reported no clubs / don't know	Total number of respondents in the study
Expect officials & members to use sunscreen	34.3% (n=12)	34.3% (n=12)	11.4% (n=4)	20.0% (n=7)	100.0% (n=35)
Parents/guardians providing children with sunscreen	34.3% (n=12)	25.7% (n=9)	11.4% (n=4)	28.6% (n=10)	100.0% (n=35)
Provision of shade	20.0% (n=7)	42.9% (n=15)	17.1% (n=6)	20.0% (n=7)	100.0% (n=35)
Parents/guardians providing children with hats (not caps)	17.1% (n=6)	22.9% (n=8)	20.0% (n=7)	39.9% (n=14)	100.0% (n=35)
Lighting provided	14.3% (n=5)	28.6% (n=10)	31.4% (n=11)	25.8% (n=9)	100.0% (n=35)
Promotion of sun protection	14.3% (n=5)	17.1% (n=6)	17.1% (n=6)	51.4% (n=18)	100.0% (n=35)
SunSmart uniforms	8.8% (n=3)	17.6% (n=6)	23.5% (n=8)	49.9% (n=17)	*100.0% (n=34)
Hats for sale (not caps)	5.7% (n=2)	11.8% (n=4)	32.4% (n=11)	49.9% (n=17)	*100.0% (n=34)
Sunscreen with membership	2.9% (n=1)	8.8% (n=3)	32.4% (n=11)	55.8% (n=19)	*100.0% (n=34)
Recommended the use of SPF30 only	0.0	11.8% (n=4)	11.8% (n=4)	76.5% (n=26)	*100.0% (n=34)
Sunscreen for sale	0.0	5.9% (n=2)	23.5% (n=8)	70.6% (n=24)	*100.0% (n=34)

Note: *Missing datum = 1

sunscreen (27.0%; n=6), sun protective clothing (14.0%; n=3) and education materials (4.5%; n=1).

Eighty percent (n=28) of the study participants wanted more information on sun safety with the majority of this group (83.0%; n=25) particularly interested in educational material. Other information sought included details on the model policy, an explanation of the benefits of SPF30+, and the links between duty of care and legislation. Additionally, 9.0% (n=3) of study participants requested further human resource support. Suggestions to improve sun protection policy development and implementation in sporting and recreation organisations and clubs in Queensland included more leadership from state officials, consistency across clubs and better role modelling.

Discussion

There is much evidence that UVR exposure can be extremely high during organised sporting activities (Moehrle 2001; Moehrle et al. 2000; Rigel et al. 1999), and for school

children, sporting activities are responsible for most of their UVR dose (Moise et al. 1999). Unfortunately, many participants in recreational/sporting activities experience sunburn (Wolley & Raasch 2005), with sun protection measures/behaviours of participants undertaking these activities often being rated as poor or not used (Moise et al. 1999; Sun 2003). In addition, epidemiological studies have confirmed associations between skin cancer and recreational sun exposure during childhood and adolescence (Corona et al. 2001) and also with participation in water sports (Hertzfeld et al. 1993; Rosso et al. 1996). Such evidence provides a compelling argument for the widespread use of sun protection measures during organised sporting activities, particularly for children. In addition, sporting/recreational clubs are particularly effective settings through which to implement and model sun protection measures, as interventions in these settings have been shown to be effective in improving adult sun-protective behaviour (Saraiya et al. 2004). It is, therefore,

encouraging that participants in this study identified sun protection as an important issue, with the majority acknowledging the importance of reducing sun exposure for their members and spectators. The most commonly stated reasons for the high level of priority were public safety and duty of care. However, as many of the clubs in this study was active during the daytime over the spring, summer and autumn months, this places their members/participants at risk of high UV exposure.

More than half of the study participants (57.0%; $n=19$) reported that their associations had a sun protection policy. Of this group 68.5% ($n=13$) reported that the majority of their association's affiliated clubs had adopted sun protection policies, with many of these using the QCF model policy. Critically though, there did appear to be a decrease in the reported review of these policies by these clubs. When compared to the results of Dobbinson and Hayman's (2002) survey of sun protection policy development and practices by sporting clubs and organisations in Victoria, the results from this study seem to indicate a higher level of development and use of sun protection policies in Queensland. Such a result may be anticipated due to the warmer climate and the common knowledge that 'Queensland is the skin cancer capital of the World'. Given this understanding, however, the level reported is disturbingly low.

The results of this study concur with Dobbinson and Hayman's (2002) findings that state sporting and recreation associations exert a major influence on sun protection activity at the club level. Eighty percent (80.0%; $n=28$) of the associations in the Queensland study were actively encouraging their clubs to improve sun protection and 29.0% ($n=10$) had been influential in getting their clubs to adopt policies. Therefore, it would seem to be beneficial to engage these associations in health promotion activities focusing on sun protection. In fact, Dobbinson and Hayman (2002, p. 1) identified that 'communication

channels from State Sporting Associations to clubs are crucial to the strategies used to promote policy development at a club level'. Unfortunately, almost half of the participating associations (43.0%; $n=15$) did not have sun protection policies at the time of the Queensland study. This is therefore a barrier to improved policy development at club level.

The results of this study indicate a low level of compliance by Queensland clubs with the range of sun protection measures recommended within the current QCF SunSmart policy. There would appear to be a strong reliance on personal responsibility for sun protection and very limited organisational supports such as the sale of hats (not caps), sunscreen or sun protective clothing. The provision of shade was reported as one of the most common strategies utilised at the club level with this complementing the use of sunscreen. By comparison, the Victorian survey found that the most common sun protection strategy was the encouragement of junior members to be 'SunSmart', which includes covering as much skin as possible with appropriate sun protection rated clothing and hats and using shade and sunscreen. Other key strategies identified in the Victorian survey were scheduling of training outside of peak UV periods, providing sunscreen, portable shade and, in the case of life saving and cricket clubs, provision of legionnaires or broad brimmed hats (Dobbinson & Hayman 2002). On a positive note, it was noted that Queensland clubs with sun protection policies employed a wider range of sun protection measures than those without policies.

Overall, the findings from this study show that improvement is needed both at the association and club levels through the implementation of a comprehensive approach to sun protection supported by a policy framework. The majority of the study participants (91.0%; $n=32$) were supportive of such an arrangement. Barriers to developing a policy framework such as this

have previously been identified as including: the need for more training advice and resources, limited control by the clubs over the facilities they use, and other club priorities taking precedent over sun protection (Dobbinson & Hayman, 2002). The participants in the Queensland study also identified a need for more resources to improve sun protection at club level. Eighty percent of the study participants ($n=28$) indicated that more educational information was needed and 54.0% ($n=19$) identified a need for increased funding to clubs for this purpose.

Limitations

There are two main limitations to this study. First, the use of convenience sampling methodology prevents the findings from being extrapolated to organisations outside of the study (Fowler 1993). Consequently, the findings were only discussed and should be interpreted as applying to the organisations in the study (Daly, Kellehear & Gliksman 1997). Second, there are sections within the study that rely on the study participants being able to accurately comment on sun protection measures at the club level. The study was, therefore, exploratory in nature and the results on club level sun protection practices need to be validated through further research with individual clubs.

Conclusion

In this study, the sun protection practices of Queensland sporting and recreational associations and their affiliated clubs were investigated through a survey of representatives from state sporting and recreational associations. More than two thirds of the participating sporting and

recreational organisations were active during the hottest months of the year, with many fixtures and training sessions being held during peak UVR times. Generally, the priority for sun protection was considered high both at the association and club levels in order to minimise the risks to their members and spectators.

It would appear from the results of this study that the majority of sporting associations actively promotes sun protection to their clubs and that the clubs with sun protection policies have a more comprehensive approach to sun protection. These are important findings in terms of progressing strategies and policies at club level. Given the current situation of non-melanoma skin cancer having an estimated incidence rate that is considerably higher than the next most common cancers in Australia (prostate cancer in men, and breast cancer in women) (AIHW 2004) and melanoma incidence is continuing to increase (AIHW 2005; Coory et al. 2006), it is essential that local sporting clubs are encouraged to adopt sun protection policies, with state sporting and recreational associations having a key role.

Overall, this study found the level of compliance with the range of recommendations in the current QCF SunSmart Policy to be disappointingly low. Many of the clubs affiliated with the participating associations would struggle to meet current good practice in sun protection and there is considerable room for improvement. The majority of the study participants indicated a desire to see more sun protection measures undertaken at club level, indicating that this would require increased funding and access to education material.

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Identifying the Presence of *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. in Private Rainwater Supplies

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*Tank rainwater is a common source of water, particularly in rural areas of Australia. In non-metropolitan areas of Western Australia (WA), 31% of households have rainwater as a source of water. However, the health risk associated with the use of this untreated supply is still an area of uncertainty. One area of uncertainty is to what extent pathogenic microorganisms are present. The aim of the study is to assess to what extent *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. in particular those that cause illness in humans, are present in rainwater tanks in rural Western Australia. The method used was to take rainwater samples and tank sediments from 10 private rainwater supplies in Mount Barker, WA. The presence of *Cryptosporidium* and *Giardia* spp. was assessed in the collected samples using USEPA Method 1623. In the event of positive samples, PCR was conducted and isolates genotyped. Samples for *Campylobacter* and *Salmonella* spp. were analysed using Australian Standards and typed using serology. None of the samples tested positive for *Cryptosporidium*, *Giardia*, *Campylobacter* or *Salmonella* spp. However, thermotolerant coliforms were detected in three tanks and *E.coli* in two tanks. The study shows that rainwater tanks sampled were free from *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. The absence of these pathogens might indicate that either animals present in Mount Barker were not reservoirs for these pathogens or that the current rainwater systems and maintenance were adequate in preventing contamination. The absence might also be a result of lower temperatures and higher water volume in winter months.*

Key words: Rainwater Tanks; Pathogens; Western Australia

In the rural regions of Australia, tank rainwater is a common source of water. In 2004, 62% of households in rural Western Australia (WA) owned rainwater tanks and 53% had tank rainwater as their main drinking source (Australian Bureau of Statistics 2004b). In WA, the most common uses of tank rainwater by households that own a rainwater tank are drinking water (72%), garden watering (46%), and food preparation (20%) (Australian Bureau of Statistics 2004a). To date, there has been limited research on the presence of pathogenic organisms in tank rainwater in WA.

Several studies have examined the presence of *Cryptosporidium* spp. and *Giardia* spp. in rainwater supplies (Abo-Shehadeh et al. 2004; Birks et al. 2004; Crabtree et al. 1996; Rigby 2002; Simmons et al. 2001), but not all have identified the species of *Cryptosporidium* and *Giardia* that were present. The presence of *Campylobacter* spp. and *Salmonella* spp. in rainwater supplies has also been investigated (Brodribb 1995; Eberhart-Phillips et al. 1997; Koplan et al. 1978; Merritt et al. 1999; Savill et al. 2001; Simmons et al. 2001; Taylor et al. 2000). Most of these studies were localised studies that looked at outbreaks related to the

consumption of tank rainwater. No studies have been performed to assess the presence of pathogenic species of *Campylobacter* spp. and *Salmonella* spp. in tank rainwater in WA to date.

The verification of the presence of *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. is not enough to determine the health impact on humans. Not all species of these organisms are pathogenic and it does not necessarily follow that species carried by birds and small animals are always harmful to health (Xiao et al. 2004). Therefore, the human health impact could be overestimated, if based on the presence of presumptive pathogens only.

Aims

The aim of this study was to determine the prevalence of pathogenic species of *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* in 10 private rainwater supplies in rural Western Australia. The secondary aim was to investigate the association between the presence of pathogenic species of *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* and the structure and maintenance of the rainwater tanks and their catchment areas.

Methods

Sample selection

Ten rainwater tanks located in the Shire of Plantagenet, where private rainwater supplies are provided to members of the public in roadhouses, or bed and breakfast establishments, were approached to participate in this pilot study. The Shire of Plantagenet is situated 359km from Perth and 50km from Albany in Western Australia. These properties were selected on the basis of providing a cross-section of private water supplies for public use and because contamination of the tank rainwater of such properties would pose a greater threat to public health. There were in total 15 properties that met this criterion. All tanks were aboveground tanks.

Water and sediment sampling

Water and sediment samples were collected from 10 rainwater tanks via a submersible pump. Samples were taken on two occasions: 30 August 2005 and 20 September 2005. Ten litre water samples and 600ml sediment samples were collected for *Cryptosporidium* and *Giardia* analysis. Water samples (500ml) and sediment samples (300ml) were collected for *Campylobacter* and *Salmonella* analysis. The submersible pump and other collection apparatus were disinfected prior to each collection. The samples were kept on ice at all times. At the second sampling water samples (500ml) were collected for *Campylobacter*, *Salmonella*, *E.coli* and thermotolerant coliforms analyses.

Data collection

Data were also collected by questionnaire and on-site observation. Photographic records were made and temperature, pH and turbidity measurements were also taken. Table 1 summarises the data collected by the questionnaire and site observation. Temperature and rainfall data prior to the sampling dates were obtained from the Bureau of Meteorology website (<http://www.bom.gov.au/climate/dwo/IDCJ/DW6088.latest.shtml>).

Table 1: Summary of data collected on rainwater tanks and catchments

Area of Interest	Information Collected
General	Property address, number of rainwater tanks, number of people working or staying at property.
Tank	Age, capacity, material, first flush mechanism, sealed roof, screened inlet, mosquito breeding, supplement of other water supplies to tank, frequency and method of sludge removal.
Roof and Gutter	Material, presence of overhanging objects and frequency of roof and gutter maintenance.
Rainwater Usage and Treatment	Main drinking water supply, filtration of rainwater, type of filter, frequency of filter maintenance, method and frequency of treatment before consumption and use of rainwater.
Other	Duration of rainwater as a drinking source and illnesses and symptoms of property inhabitants.

Laboratory analysis

Cryptosporidium and *Giardia* were concentrated using USEPA Method 1623. The water was filtered using cartridge filtration, and then immunomagnetic separation was used to remove the *Cryptosporidium* oocysts and *Giardia* cysts. DNA was extracted using a Qiagen stool kit and partial 18S rRNA regions of each parasite amplified by PCR using previously described methods (Hopkins et al. 1997; Ryan et al. 2003).

Campylobacter, *Salmonella*, *E.coli* and thermotolerant coliforms were analysed using the relevant Australian Standards within 24 hours upon collection. The samples were filtered through a membrane that was then placed into pre-enrichment broth. The culture was then inoculated into enrichment broth. Selective media were used to grow the target microorganisms. Tests were conducted to confirm the species of *Campylobacter* and *Salmonella*. If the test could not confirm the species, serotyping was done for species identification.

Results

There was virtually no rainfall in the days prior to sampling but rainfall on the first date of sampling was 6.8mm. Also, the average rainfall for the months was high: 78mm and 76.6mm for August and September respectively. The average temperatures for the Shire of Plantagenet in the two months were fairly low and similar with a minimum of 6°C and a maximum of 15°C.

Cryptosporidium, *Giardia*, *Campylobacter* and *Salmonella* spp. were not detected in the water and sediment samples on the first and second sampling dates. On the second sampling date, thermotolerant coliforms were detected in three of the tanks, while *E.coli* was identified in two of the tanks. A summary of the tank conditions and maintenance is shown in Table 2. All tanks were aboveground tanks and rainwater from these tanks was the main source of drinking water. All of the rainwater tanks had good roof and gutter conditions and there were no

visible signs of faecal contamination. There were also no reported illnesses among the property inhabitants in two weeks previous to water sampling.

Table 2: Characteristics of sample tanks and treatment of that tank water

Characteristics	Number of tanks
Age of tank (years)	
<2	1
2-3	1
4-9	3
>10	2
Not stated	3
Tank Material	
Galvanised Iron	5
Concrete	3
Fibreglass	1
Zincalume	1
Tank capacity	
8kL	1
25kL	1
56kL	2
92kL	5
130kL	1
Screened inlet	
Yes	7
No	3
First-flush mechanism	
Yes	3
No	7
Boil water prior use	
Sometimes	3
Never	3
Not Stated	4
Disinfect water prior use	
No	4
1-2 times/yr	2
UV	1
Not Stated	3
Filter water prior use	
Yes	6
No	3
Not Stated	1
Tank Last Desludged	
Never	4
>6 Months	1
6months - 1 year	1
2-5 years	1
Not Stated	3

Characteristics of tanks tested positive for *E.coli* and/or thermotolerant coliforms

Tank 1

- estimated count for both *E.coli* and thermotolerant coliform in 100ml was 4
- leaves were present in tank
- tank had the lowest capacity among the ten tanks sampled (8kL)

- inlet hole to tank was uncovered
- there was no first flush mechanism
- disinfection carried out 1-2 times a year
- tank had never been desludged.

Tank 2

- estimated counts for both *E.coli* and thermotolerant coliforms in 100ml was 29
- a frog was detected in the tank at the time of sampling
- tank had no screened inlet
- there was no first flush mechanism
- tank water was never disinfected before use
- tank was desludged 2-5 years ago.

Tank 3

- estimated count for thermotolerant coliforms was 3
- tank was desludged less than 6 months ago
- tank water was never disinfected before use.

Discussion

The results of this survey suggest there was a negligible risk of exposure to human pathogens through drinking tank rainwater in the 10 rainwater tanks sampled in the Mount Barker region of Western Australia. The results might be reflective only of the exposure in winter, and there is a possibility that the risk is higher in late spring or summer when the temperatures are more conducive to pathogen growth and survival. There are a number of other possible reasons for the negative results.

Rainwater system maintenance and characteristics

There was no obvious faecal contamination of the 10 rainwater catchment areas sampled. There were minimal overhanging objects over the roof catchment area and all gutters had been cleaned in the past six months. In addition, all the rainwater tanks had sealed roofs and all were aboveground. This prevented easy access of animals to the systems. These factors might have contributed in minimising the contamination of the rainwater system.

Absence of reservoir hosts

A factor that might have contributed to the results was that there were few animals harbouring *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. that could access the rainwater system. The rainwater systems that were sampled were primarily outside the town area where human-related pollution is less. Carriage rates of pathogens have been found to be higher when animals are in contact with human settlements (Broman et al. 2004). Fenlon (1981, 1983), for example, found that animals that feed off refuse tips have a higher probability of carrying pathogenic microorganisms. The study identified 13% of gulls to carry *Salmonella* spp. whereas gulls near a sewage outfall had significantly higher carriage rates of 17-21%. Animals in the areas of sampling would have minimal contact with human activities and hence were less likely to carry pathogenic microorganisms. If this is a valid explanation then the risk of exposure will be low throughout the year.

High rainfall and the dilution of pathogens

High rainfall during the winter season might have caused a high turnover of rainwater in tanks. This would have diluted the concentration of possible pathogens that were already present in the rainwater tank. All of the tanks sampled on the first sampling date were full and the overflow

valves were released in all of the tanks allowing freshwater to enter the already full tanks. This indicated that there was sufficient rainfall during the winter season to fill up the tanks. The low concentration of possible pathogens in the rainwater tank might have minimised the chance of positive detection because only a small volume of water sample was taken for analysis: 10L for *Cryptosporidium* and *Giardia* spp. analysis; and 100ml each for *Salmonella*, *Campylobacter*, and *E.coli* and thermotolerant coliform analysis.

Temperature and pH

The low acidity and low temperature of the tank water at the sampling time were more favourable to the growth of *Salmonella* spp. compared with *Campylobacter* spp because *Salmonella* spp. are still able to grow in low temperatures. The growth of *Campylobacter* spp. would be favoured during summer months when the temperature is higher. When *Cryptosporidium* and *Giardia* spp. are washed into tanks, they will not continue to grow in the tank environment, because they need to be in a host reservoir to grow (O'Donoghue 1995). However, they are more resilient to temperature and pH differences. Chlorination might affect the viability of *Campylobacter* and *Salmonella* spp. Therefore, rainwater tanks that are chlorinated might be safe from *Campylobacter* and *Salmonella* spp. but *Cryptosporidium* and *Giardia* spp. might still pose a risk to health if no further water treatment is undertaken.

Tank capacity

Tank capacity has been shown to be a significant determinant of the tank rainwater quality (Plazinska 2003). All high capacity tanks tested were negative for thermotolerant coliforms. Theoretically,

high capacity tanks are able to minimise the concentration of contamination and minimise the resuspension of microorganisms in the sediment of the tank (Plazinska 2003). Therefore, the pathogens could be present in the water at a safe concentration, provided they do not multiply. Five of the six high capacity tanks in this study tested negative for thermotolerant coliforms, and these data are consistent with past studies.

The use of indicator organisms

On one hand, several past studies have shown that there was no correlation between thermotolerant coliforms and the presence of pathogens (Abo-Shehada et al. 2004; Crabtree et al. 1996; Rigby 2002; Savill et al. 2001; Simmons et al. 2001; Taylor et al. 2000). On the other hand, the absence of these indicator organisms might not necessarily indicate the absence of the pathogenic microorganisms. Other factors such as seasonal variation and rainwater treatment might destroy the indicator organism but not the pathogenic microorganisms. The findings of the study were consistent with past studies that indicated the absence of pathogens despite the presence of *E.coli* and thermotolerant coliforms.

Conclusion

The pilot study found no pathogenic species of *Cryptosporidium*, *Giardia*, *Campylobacter* and *Salmonella* spp. in the 10 tanks sampled in the Shire of Plantagenet in the late winter/early spring period. While the tanks sampled in this study were a good representation of the tanks in Mount Barker, further sampling during spring through to autumn might be warranted to confirm that rainwater is a potential source of pathogens.

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The End of Poverty: Economic Possibilities for our Time

Jeffrey D. Sachs

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On the surface, many may ask why a book about ending poverty might be of interest to environmental health practitioners. Even though it is not the intention of *The End of Poverty* to answer this question, it does so in an eloquent and forceful manner that is hard to ignore. For example, the text states that:

more than one million African children, and perhaps as many as three million children, succumb to malaria each year. This horrific catastrophe occurs despite the fact that the disease is partly preventable - through the use of bed nets and other environmental controls that do not reach the impoverished villages of Malawi and most of the rest of the continent - and completely treatable. There is no conceivable excuse for this disease to be taking millions of lives each year (p. 7).

As can be seen from the quote above, the author is passionate about this issue and argues that from both an economic and humanity perspective:

our safety and prosperity depend at least as much on collective decisions to fight disease, promote good science and widespread education, provide critical infrastructure, and act in unison to help the poorest of the poor. When the preconditions of basic infrastructure (roads, power and ports) and human capital (health and education) are in place, markets are powerful engines for development (p. 3).

The End of Poverty provides a thesis on how we can end extreme poverty within our own generation and do so without impacting the quality of life we have strived so hard to achieve. The author describes what poverty is. For example, extreme poverty equals an income of \$1 per day per person and this means that households cannot meet basic needs for survival. They are chronically hungry, unable to access health care, lack safe drinking water and sanitation, cannot afford education for their children and lack rudimentary shelter. The lessons learned

from his vast involvement in economic development activities around the world, presents sound arguments for why extreme poverty can be eliminated, and he provides an action plan for achieving this.

Sachs boldly identifies the economic possibilities of our time. These are to meet the Millennium Development Goals by 2015; to end extreme poverty by 2025; to ensure that well before 2025 all of the world's poor countries can make reliable progress up the ladder of economic development; and to accomplish all of this with modest financial help from the rich countries, with these countries providing more than they do now but within the bounds of what they have long promised.

A basic tenet of this thesis is that some countries are trapped in a perpetual downward poverty spiral that they cannot get out of, principally because they cannot even get onto the bottom step of the economic development ladder. It is, therefore, the duty of the rich countries to help these poor countries to get onto the bottom step of the ladder, with this having significant benefits for both. Sachs goes on to state that ending extreme poverty is achievable and affordable within the next 20 years for the following reasons: i. despite more than 1.1 billion people living in extreme poverty and an additional 1.5 billion people who are classified as being poor (this equates to a total of 40% of the world's population), the numbers of extreme poor have declined to a smaller proportion of the world's population than at any other time; ii. the goal is to end extreme poverty and not all poverty, and is definitely not to try and close the gap between the rich and the poor; iii. success in ending the poverty trap is much easier than it appears - by targeting funding at practical investments in

infrastructure, water and sanitation and disease control; iv. the rich world is so vastly rich that, for example, in the year 2000, the 400 hundred richest people in the US had a combined income that was more than the combined income of four of the poorer countries in Africa (Botswana, Nigeria, Senegal, and Uganda) who have a total population of 161 million; and v. our technology is more powerful than ever and new breakthroughs are occurring everyday.

Even though the author places much of the responsibility for ending poverty on the shoulders of the rich countries (for example, the 35 richest countries have already agreed to, but have not provided, 0.7 % of GNP in official development assistance, with the US currently providing only 0.15%), he acknowledges that:

poor countries must take ending poverty seriously, and will have to devote a greater share of their national resources to cutting poverty rather than to war, corruption, and political infighting (p. 266).

The starting point is, therefore, a 'Global Compact' between rich and poor countries, and the author suggests that a framework for achieving this is the *Millennium Development Goals-Based Poverty Reduction Strategy*.

Sachs concludes the text by providing the following nine steps for ending poverty within our lifetime: commit to ending poverty, adopt a plan of action, raise the

voice of the poor, redeem the role of the US in the world, rescue the IMF and the World Bank, strengthen the United Nations, harness global science, promote sustainable development, and make a personal commitment.

Overall, *The End of Poverty* is impressive, insightful, and optimistic, as well as being disturbing, confronting and challenging. It presents an intelligent and decisive 'plan for action' for an issue that the world has too long placed in the 'too hard basket'. The challenge of our generation is therefore "to help the poorest of the poor to escape the misery of extreme poverty so that they may begin their own ascent up the ladder of economic development" (p. 24). This is certainly a challenge in which the environmental health profession is well placed to have a significant role. I would, therefore, recommend this text to all those who would like to take up the challenge, who would like to have their comfort zone disturbed, or who would like to be reminded of the many comforts we should be both thankful for and eager to share.

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The Weather Makers: The History and Future Impact of Climate Change

Tim Flannery

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For some time now, global climate change has been taken seriously by the scientific community, but it has taken much longer for the general public, business and government, to realise the potential impacts of climate change and to accept that it is actually happening. In *The Weather Makers*, Flannery states that:

one of the biggest obstacles to making a start on climate change is that it has become a cliché before it has even been understood. What we need now is good information and careful thinking, because in the years to come this issue will dwarf all the others combined. It will become the only issue (p. 8).

The Weather Makers attempts to debunk the climate change cliché by discussing its history and potential impacts in a way that makes it easy for everyone (including scientists and politicians) to understand. It starts by describing the basic principles of how our Earth functions and what the basic survival needs are of the global ecosystem. Flannery then discusses what impacts we humans are having on the planet. From climate and ecological changes in Greenland and Antarctica to Africa and islands in the Pacific, to changes in the migration patterns of copepods and butterflies, and to coral bleaching in the Great Barrier Reef, and the extinction of many species such as the golden toad, he eloquently weaves scientific evidence into a readable format that describes the serious issues faced and provides a clear 'call to action'.

Flannery then turns his attention to discussing the ways in which future climate

change and its impacts are being predicted and the potential scenarios of these predictions. He goes on to describe the various international attempts, including the Montreal and Kyoto protocols, to try and address the issue, and for Kyoto, the reasons why Australia and the US are reluctant to sign up and why some business groups are still opposed while others have now become passionate converts for a need for action.

The text concludes with a series of chapters that discuss some of the potential solutions to climate change, for example, solar and wind energy, nuclear energy, hybrid vehicles and other engineering solutions. The final chapter is devoted to the personal actions that each of us can take to help address this most important issue. Flannery argues that:

it is my firm belief that all the efforts of government and industry will come to naught unless the good citizen and consumer takes the initiative... you can, in a few months rather than the fifty years allowed by some governments, easily attain the 70 percent reduction in emissions required to stabilise the Earth's climate (pp. 302, 303).

He goes on to say that:

"understanding how you use electricity is the most powerful tool in your armoury, for that allows you to make effective decisions about reducing your personal emissions of CO₂ (p. 303).

Overall, *The Weather Makers* is one of the most important books ever to be released on climate change because it demystifies the

science and politics of this extremely important global issue and does so in a very authoritative but easy-to-read way. It therefore achieves the broader goal of providing 'good information' on which 'careful thinking' can be had. As environmental health practitioners, climate change is the biggest issue that we face and this text gives us the necessary tools to be fully informed advocates in our own communities. I would, therefore,

recommend this text to all those who find the climate change issues complex and daunting, and for those who would like to communicate this issue with understanding, passion and relevance.

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