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# Environmental Health

*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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*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.

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

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### Research and Theory

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

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

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

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# Contents Environmental Health, Volume Six, Number Four, 2006

## Editorial



## Articles

### Research and Theory



### Practice, Policy and Law



### Reports and Reviews

# CONTENTS ENVIRONMENTAL HEALTH, VOLUME SIX, NUMBER THREE, 2006

## EDITORIAL



## ARTICLES

### RESEARCH AND THEORY



### PRACTICE, POLICY AND LAW

# Contents Environmental Health, Volume Six, Number Two, 2006

## Editorial



## Articles

### Research and Theory



### Practice, Policy and Law

# Contents Environmental Health, Volume Six, Number One, 2006

## Editorial



## Articles

### Research and Theory



### Practice, Policy and Law



## Reports and Reviews

*Environmental Health*, Volume 6, Number 4, brings us to the end of another successful year, drawing attention to a number of key issues, from the relationship between dogs and people in the Indigenous communities (Senions et al.), to temperature data awareness within a HACCP system (Robson & Varma).

The papers combine to produce a diverse issue, with the first paper by Boreland, Balding, Wlodarczyk and Lyle on lead dust within Broken Hill homes. Their paper investigates the relationship between house dust and children's lead levels. The utilisation of ecological analysis was able to confirm whether findings for individual children were consistent across the whole population.

An animal model of silicosis and silica induced inflammation is also explored by Wang, Peng and Lawson, as they undertake useful studies of potentially effective drugs, given that such experiments cannot be undertaken directly on humans. These studies indicate that silica is a strong inflammatory agent and can induce an overt and rapid inflammatory response. Wang, Peng and Lawson's second paper continues with an investigation related to silica carcinogenesis and the possibility to access the carcinogenesis by micronucleus formation in alveolar macrophages ex vivo. Wang, Peng and Lawson discovered a major advantage of this method of testing in that it can test not only the toxicity of silica itself but also its induced reactants.

Stoneham et al.'s paper on environmental health planning describes the process used to develop a forward service plan for health services within the City of Swan, Western Australia. The City of Swan is the largest metropolitan local government area within metropolitan Perth, and is one of the fastest growing urban corridors in Australia, thus an effective forward service plan is a necessity.

Of particular and direct interest to readers is the announcement by the AIEH of an Environmental Health Workforce Summit to be held in April 2007. There has been a longstanding issue around the EHO workforce and the lack of qualified practitioners across Australia. This Forum will bring together various stakeholders to look specifically at strategies to overcome this shortage, maintain EHO skills, and examine the place of environmental health technicians in the delivery of local environmental health services.

I hope you enjoy our current and final issue of *Environmental Health* for 2006. If you have any comments on any of the articles published within the Journal, or on a particular environmental health issue, please send a Letter to the Editor and allow your views to make a difference. I would like to take this opportunity to wish everyone a very happy and safe new year.

*Jim Smith*
*Editor*

Back to TOC

# Lead Dust in Broken Hill Homes: Relationship between House Dust and Children's Blood Lead Levels

**Frances Boreland[1], David M. Lyle[1], John Wlodarczyk[2] and William A. Balding[3]**

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*Objective: To determine the influence of indoor lead dust on children's blood lead levels in Broken Hill, Australia.*
*Method: Indoor lead flux (µg/m²/30 days) was measured in the homes of 74 pre-school aged children and compared with their routinely collected blood lead samples. Ecological analysis was used to confirm whether findings for individual children were consistent across the whole population.*
*Results: Compared with homes in the lowest indoor lead category, homes in the highest category had 11 times the lead flux, and children living in them had 50% higher geometric mean blood lead levels and were five times more likely to have significantly elevated blood lead levels (>15 µg/dL). Increasing indoor lead fallout from 100 to 1000 µg/m²/30 days was associated with 32% (95% CI 4-67%) higher blood lead levels. The correlation noted for individual children was supported by the ecological analysis.*
*Conclusions: Indoor lead flux is a useful predictor of elevated blood lead. Home remediation is most likely to reduce children's blood lead levels if indoor lead levels are reduced by an order of magnitude; small changes in indoor lead are unlikely to be effective.*
*Implications: Reducing indoor lead levels is likely to be an important component of a multi-faceted strategy for reducing blood lead levels among young children from lead mining or smelting communities such as Broken Hill. A useful indicator for identifying children who are most likely to benefit from home remediation is the indoor lead flux level.*

**Key words**: *Children; Blood Lead Level; Indoor Lead; Lead Dust; Lead Flux*

While the health effects of high blood lead levels in young children are well understood, how best to reduce them is less clear. Several factors determine children's blood lead levels, including physiology, lead levels in the environment and children's interaction with the environment. Along with soil and paint (Lanphear et al. 1998; Lewin, Sarasua & Jones 1999; Markowitz & Rosner 2000; Murgueytio, Evans & Roberts 1995), house dust is a potential source of lead for young children; accidental ingestion of contaminated dust is thought to be the primary route of exposure (Lanphear et al. 1996; Murgueytio, Evans & Roberts 1995; Trepka et al. 1997). Thus identifying and interrupting potential lead exposure pathways in the child's home (remediation or abatement) is an obvious approach to reducing blood lead levels, but research shows it to have varying effectiveness (Aschengrau et al. 1997; Farrell et al. 1998; Langlois et al. 1996; Lanphear et al. 2003; Leighton et al. 2003; Lorenzana et al. 2003; Weitzman et al. 1993). Understanding how better to target home remediation would benefit communities affected by lead; this paper discusses results from the Broken Hill Lead Management Program, which suggest lead levels in indoor dust as one such indicator.

The city of Broken Hill (NSW, Australia) grew up around one of the largest silver-lead-zinc ore-bodies in the world, with homes built within close walking distance of the mines. Mining has been continuous since 1884, with on-site smelting for the first 15 years (Figure 1). In the early 1990s, studies revealed a significant lead health issue among the city's pre-school children. An epidemiological study found indicators of high indoor lead levels in homes, such as poor seal against dust ingress, and vacuuming every couple of days, were associated with higher blood lead levels (Phillips 1998). Isotope studies revealed the sources of lead for children's blood to be similar to that for house dust, with the ore-body, paint and petrol all significant sources (Gulson et al. 1995). The Lead Management Program, a major government funded initiative, was subsequently established, and blood lead levels have since declined markedly (Lyle et al. 2006).

One of the major interventions, home remediation, commenced in 1994 and was evaluated with a randomised controlled trial. Despite reducing indoor lead levels by about 50% (Boreland & Lyle 2006) remediation had limited impact on blood lead levels (Corbett et al. 2000). The study reported in this paper helps to explain those findings.

## Methods

Between September and November 1995, 150 of 310 families with children who were due for an annual blood lead test were invited to join the study. Families were chosen sequentially from a list ordered by family number (a unique identifier assigned the first time a child receives a blood lead test), without reference to the child's previous blood lead level results or the house type and condition. This process was continued until 15 families had been chosen from each of the ten districts delineated for this study according to soil lead level and proximity to the mining lease (Boreland et al. 2002).

Seventy-two of the 150 families were eligible (could be contacted, were not planning to move or renovate during the period of the study and their homes were not scheduled for lead remediation during that period) and 61 of these agreed to participate in the study. A further 25 families with young children were included from 55 additional households purposefully recruited to the study to investigate a related issue: the degree to which lead flux (the amount of lead falling on a surface over a given time period, measured as µg lead/m$^2$/30 days) is influenced by house construction and condition. These additional homes had been purposively recruited to provide a mix of house construction types (i.e. built of stone, wood frame with iron cladding, or brick) and quality of seal against dust entry (classified as 'poor', 'adequate' or 'very well') that was as even as possible within each of the ten districts. Data were collected on indoor lead flux measurements and the age, sex, blood lead measurement, and date of test for all resident children.

### *Sampling*

Indoor lead levels were measured with petri dishes, as described in Boreland et al. (2002). Lead content was reported as loading (µg/m$^2$), which was then converted to flux (µg/m$^2$/30 days).

Blood samples were collected from children when they presented to the Environmental Lead Centre clinic for routine blood lead testing. Venous blood samples were collected from each child using the standard procedure (Standards Association of Australia 1988) after ensuring the skin over the venipuncture was free from dust and lead. Blood samples were stored at 4°C and transported by air overnight for testing the next day. Analysis was performed by electrothermal atomisation atomic absorption spectrometry. The Adelaide Women and Children's Hospital laboratory, which participated in internal and external quality assurance programs, was used throughout the study.

Children were recalled for blood lead testing at intervals of three to 12 months, depending on their blood lead level. The closest blood test within six months of the indoor lead measurement was used for the individual analysis. In secondary analyses we examined the relationship between indoor lead level and significantly elevated blood lead level (above 15 µg/dL) and correlated the average indoor lead flux measurements with age-sex standardised mean blood lead levels from children in each of the 10 districts. Blood lead levels were estimated from routine surveillance data for 1995. Where a child had more than one blood test, the first test for 1995 was used.

### *Statistical analysis*

Both blood lead and indoor lead data were strongly skewed to the right, and so were log transformed before analysis and reported as geometric means. Homes were divided into three groups (tertiles) according to indoor lead level. Descriptive statistics and the proportion of children within each blood lead range (0-9, 10-15, >15 µg/dL) were calculated for each tertile.

The relationship between blood lead level of individual children and the lead level in their homes was described initially with simple linear regression, and then multiple regression analysis was used to determine how indoor lead, sex and age affected blood lead level, with time lag between blood and indoor dust samples included as a potential confounding factor. As 22 homes had more than one child for whom blood lead results were available, a repeated subject design, with house as the repeated subject, was used to correct for the effect of such clustering on variance estimates. Residual analysis and checks for normality were undertaken to check that the assumptions of the multiple regression models were met. Examination of the residuals showed that assumptions of normality broke down because of two large residuals; these were excluded and the model re-run to check for robustness.

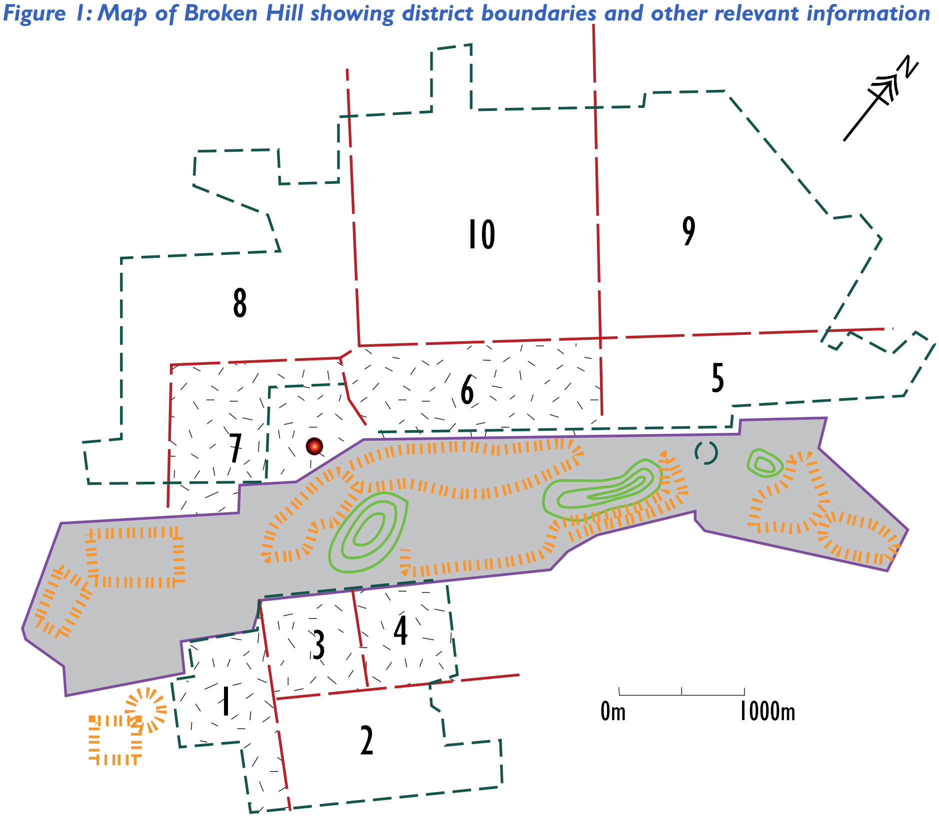
We used logistic regression to further explore the association between significantly elevated blood lead levels (> 15 µg/dL) and indoor lead level, after adjusting for potential confounders. To determine whether the relationship observed for individual children existed at district level, simple linear regression was used to describe the relationship between the mean blood lead level and mean indoor lead level in each district. Statistical analysis was performed using Microsoft Excel 2000 and SAS System for Windows v8e.

## Results

Of the 86 families with young children agreeing to the study, 36 were excluded from further analysis: 20 because of reported disturbance during the sampling period (renovation, excavation, and so on), five because the house had been remediated less than six months before the flux measurement, 10 because there was no valid blood test (the child was older than 60 months at the blood test closest to the dust measurement, or had not had a blood test within six months of the dust measurement), and a further home was excluded because valid results were available from only one room. This left 50 families with both the indoor lead dust measured in their home and children with a blood lead test within 6 months of the flux measurement. Thus, data from 50 homes and 74 children were available for assessing the relationship between indoor lead and children's blood lead levels. Mean blood lead level of the 74 children was 9.6 µg/dL (range 2-38 µg/dL) and mean indoor lead level was 421 µg/m$^2$/30 days (range 33-4,883 µg/m$^2$/30 days).

Compared with children living in homes in the lowest indoor lead tertile (geometric mean flux 138 µg/m$^2$/30 days, range 33 - 243 µg/m$^2$/30 days), children living in homes in

**Figure 1: Map of Broken Hill showing district boundaries and other relevant information**



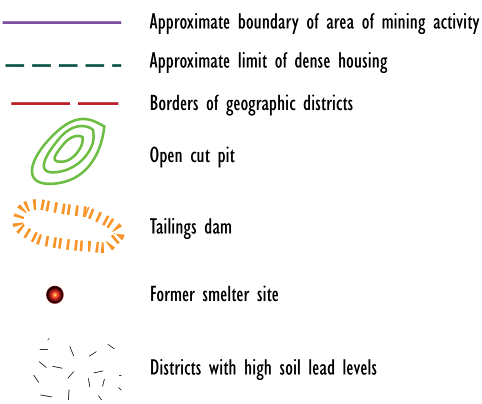



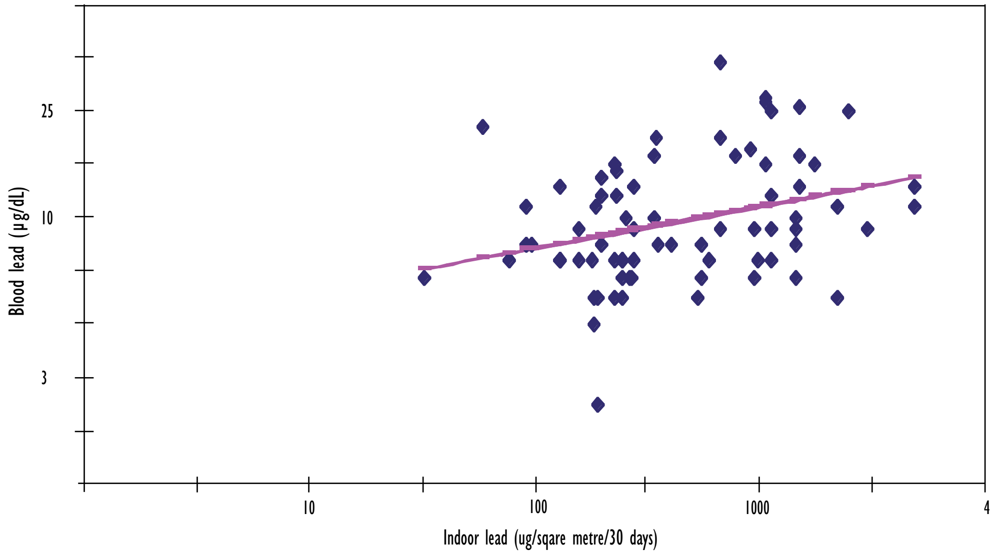

**Table 1: Descriptive statistics for blood lead levels by indoor lead level**

| | Indoor Lead level | | |
|---|---|---|---|
| | Lowest | Intermediate | Highest |
| Number of houses | 17 | 16 | 17 |
| Indoor lead: | | | |
| Flux range (μg/m²/30 days) | 33 - 243 | 248 - 667 | 780 - 4883 |
| Geomean flux | 138 | 368 | 1463 |
| 95% confidence interval | 103 - 184 | 302 - 449 | 1140 - 1878 |
| Blood lead levels: | | | |
| PbB range (μg/dL) | 2 - 22 | 5 - 38 | 5 - 28 |
| Geomean PbB | 8.0 | 9.3 | 12.2 |
| 95% confidence interval | 6.2 - 9.6 | 7.2 - 11.9 | 9.9 - 15.0 |
| Blood lead categories: | | | |
| % (n) <10 μg/dL | 68% (19) | 65% (13) | 38% (10) |
| % (n) 10-15 μg/dL | 25% (7) | 15% (3) | 24% (6) |
| % (n) >15 μg/dL | 7% (2) | 20% (4) | 38% (10) |
| Total | 100% (28) | 100% (20) | 100% (26) |

the highest tertile (geometric mean flux 1,463 μg/m²/30 days, range 1,140 - 1,878 μg/m²/30 days) had 50% higher blood lead levels and were five times more likely to have significantly elevated blood lead levels (>15 μg/dL) (Table 1).

Children's blood lead levels were higher in homes with high indoor lead levels ($R^2$ = 0.10, Adjusted $R^2$ = 0.09, P = .0057). Average blood lead levels were 43% (95% confidence interval 11-84%) higher when indoor lead levels increased from 100 to 1,000 μg/m²/30 days, which equated to an increase of 3.33 μg/dL in our sample (Figure 2).

**Figure 2: Relationship between indoor lead and blood lead for individual children living in non-remediated houses**




*Note: $\log_{10}$ Blood lead = .573121 + .156250 ($\log_{10}$ µg lead/m$^2$/30 days), $R^2$ = .10, P=.0057*

**Table 2: Effect of indoor lead, sex, and child age on blood lead levels among pre-school children in Broken Hill**

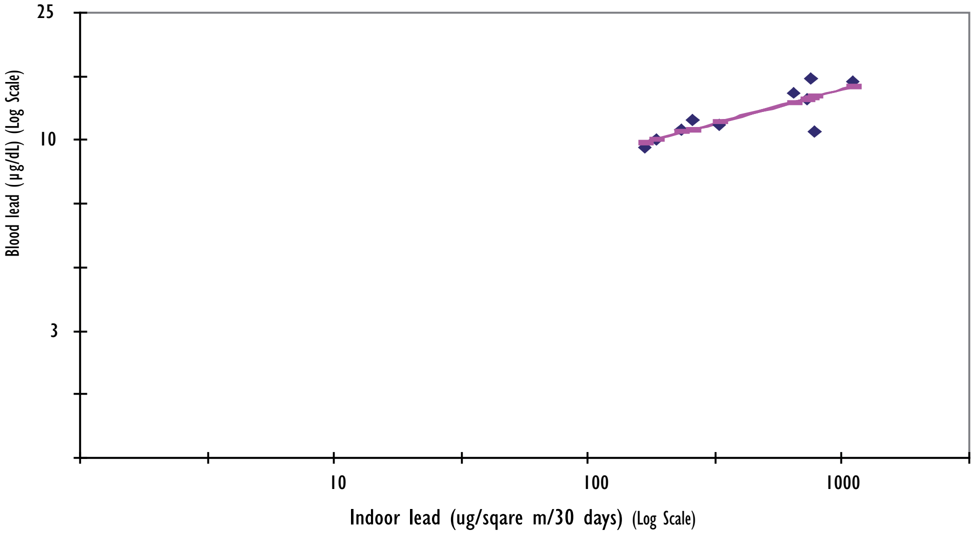
| Factor | Co-efficient | Ratio Measure | 95% Confidence Interval | P |
|---|---|---|---|---|
| LogFlux: | 0.1207 | 1.32 | 1.04 - 1.67 | 0.0199 |
| Sex: | | | | |
| Female | | 1 | | |
| Male | -0.0177 | 0.96 | 0.79 - 1.17 | 0.6916 |
| Age: | | | | |
| 6-11 months | | 1 | | |
| 12-23 months | 0.2630 | 1.83 | 1.31 - 2.56 | 0.0004 |
| 24-35 months | 0.3344 | 2.16 | 1.65 - 2.83 | 0.0000 |
| 36-47 months | 0.2917 | 1.96 | 1.40 - 2.74 | 0.0001 |
| 48-59 months | 0.2070 | 1.61 | 1.22 - 2.12 | 0.0007 |
| Time lag: | | | | |
| During | | 1 | | |
| Before (4-6mths) | .0569 | 1.14 | 0.89 - 1.46 | 0.2963 |
| Before (1-3mths) | -.0563 | 0.88 | 0.66 - 1.17 | 0.3814 |
| After (1-3mths) | -.0699 | 0.85 | 0.59 - 1.24 | 0.3967 |
| After (4-6mths) | -.0428 | 0.91 | 0.55 - 1.50 | 0.7015 |

**Table 3: Effect of indoor lead, sex, and child age on risk of developing significantly elevated blood lead levels (above 15 µg/dL) among pre-school children in Broken Hill**

| Factor | Co-efficient | Odds Ratio | 95% Confidence Interval | P |
|---|---|---|---|---|
| Age: | | | | |
| < 2 years | | 1 | | |
| 2-3 years | 1.7325 | 5.66 | 1.00 - 31.98 | 0.0500 |
| > 3 years | 0.1463 | 1.16 | 0.14 - 9.96 | 0.8940 |
| Sex: | | | | |
| Female | | 1 | | |
| Male | -0.5821 | 0.56 | 0.15 - 2.05 | 0.3801 |
| Indoor lead tertile: | | | | |
| First | | 1 | | |
| Second | 1.0703 | 2.92 | 0.54 - 18.95 | 0.2623 |
| Third | 2.2128 | 9.14 | 1.60 - 52.30 | 0.0129 |

Indoor lead remained a significant predictor of blood lead after child age and sex were accounted for, with blood lead 32% (95% CI 4-67%) higher when indoor lead levels increased from 100 to 1,000 µg/m$^2$/30 days ($R^2$ = 0.30, Adjusted $R^2$ = 0.19) (Table 2). Blood lead level was not affected by the child's sex or the length of time between the blood lead and indoor lead measurements, and no statistically significant interactions were found between indoor lead, child age, sex and length of time between the blood lead and indoor lead measurements. Exclusion of the two observations with large

***Figure 3: Ecological analysis: relationship between blood lead levels and mean indoor lead flux levels for 10 lead risk areas in Broken Hill, 1995***




*Note: $Log_{10}$ blood lead = .524204 + .211861($log_{10}$ µg lead/$m^2$ 30 days), $R^2$ = .68, P = .0035.*

residuals (which were for the children with the highest and sixth highest blood lead levels) confirmed that the conclusions were robust, with only minor changes in the estimated effect of each parameter.

After accounting for the effects of age and sex, children with significantly elevated blood lead levels were nine times more likely to live in homes which were in the highest tertile for indoor lead (P=0.0129); lower indoor lead levels in the second tertile may also be associated with increased risk but the effect was not statistically significant (OR = 2.92, P=0.2623) (Table 3). On average, children living in districts with higher indoor lead levels had higher blood lead levels ($R^2$ = 0.67, P = .0034). As indoor lead levels increased from 100 to 1,000 µg/$m^2$/30 days, blood lead level was estimated to be 63% higher (95% confidence interval 24-114%), which translates into 5.58 µg/dL (Figure 3).

## Discussion

The study is consistent with findings from other lead-exposed communities and localities, which have shown that indoor lead contamination is associated with a small but significant proportion of the variation in blood lead levels in young children. This study suggests that, within the range of exposure in Broken Hill homes, potential reductions in average blood lead levels of around 30% might be achieved if home remediation were able to reduce indoor lead levels from around 1,000 to around 100 µg/$m^2$/30 days.

While the study confirms that high indoor lead levels are one of a number of potential hazards (i.e. sources and pathways of lead exposure) to young Broken Hill children, the limitations of the cross-sectional design mean that the study cannot establish a causal link between indoor lead in dust and blood lead. Further work is required to demonstrate that when children move to a lower exposure level their blood lead levels decrease.

Not surprisingly we found considerable variation in blood lead level based on lead levels in children's homes. Seven percent of pre-school children living in homes that were in the lowest tertile for indoor lead levels had a blood lead level above 15 µg/dL (Table 1). While this is slightly above the level at which the National Health and Medical Research Council (NHMRC) recommends public health action (5%) (NHMRC 1987), it compares favourably with the overall proportion of children in Broken Hill who had blood lead levels above 15 µg/dL (14.1%) at the time of the study (Lyle et al. 2006). The average indoor lead level of this group of homes (138 µg/m$^2$/30 days) was similar to that recorded for 'very well' and 'adequately' sealed homes in low lead exposure areas of Broken Hill (112 and 252 µg/m$^2$/30 days respectively) (Boreland et al. 2002). Conversely, more than one in three children living in homes with the highest indoor lead levels had a blood lead level above 15 µg/dL. Similarly, after accounting for the effects of age and sex, children with blood lead levels above 15 µg/dL were nine times more likely to live in homes which were in the highest tertile for indoor lead (P=0.0129) (Table 3). The average lead level of these homes was 1,522 µg/m$^2$/30 days (Table 1), which is similar to the average lead level in 'adequately' and 'poorly' sealed homes in high lead exposure areas of Broken Hill (1,278 and 1,442 µg/m$^2$/30 days respectively) (Boreland et al. 2002).

We found that variation in indoor lead explained about 10% of the variation in children's blood lead level, and about 7% of the variation after age and sex was taken into account. Studies from other locations report indoor lead explains 15-17% of the variation in blood lead level before other factors are taken into account (Galvin et al. 1993; Lanphear et al. 1996; Lanphear et al. 1998; Murgueytio, Evans & Roberts 1995), and 4-6% of the variation after factors such as race, income and lead levels in soil, paint and water are taken into account. Although different measures of indoor lead [loading (µg/m$^2$) and concentration (µg/g)] were used, other studies reported an order of magnitude increase in indoor lead was associated with similar increases in blood lead (48% and 13% respectively) as found in this study (32%) (Table 2) (Lanphear et al. 1996; Lewin, Sarasua & Jones 1999).

The data from individual children suggest that an order of magnitude increase in indoor lead is associated with a 30% increase in blood lead levels, which translates into about 3.5 µg/dL (Figure 2 and Table 2). The ecological analysis suggests a similar increase of about 5.57 µg/dL (63%) (Figure 3). Results from the logistic regression were consistent, with blood lead levels above 15 µg/dL being strongly associated with very high indoor lead flux (i.e. in the third tertile) in particular.

If the relationship is causal, a large decrease in indoor lead levels might similarly be associated with a moderate decrease in children's blood lead levels, so that home remediation would have most benefit for children with elevated blood lead levels who lived in homes with high indoor lead levels, but less benefit for children living in homes with lower indoor lead levels. This might explain why a randomised controlled trial in Broken Hill found home remediation to have only a modest impact on blood lead levels (Corbett et al. 2000). Focusing on the impact of remediation on indoor lead levels, although remediation reduced these levels, closer inspection (Boreland & Lyle 2006) found that most of the benefit was received in homes with very high lead levels before remediation, and homes with moderate to low lead levels prior to remediation benefited very little. Additionally, the majority of homes did not have high lead levels on internal floors before remediation, with only 24% of homes exceeding US guidelines (40 µg/ft$^2$, ( 431 µg/m$^2$) (USEPA, 2001). Taken together, these studies reinforce the argument that the home is only one potential source of lead exposure for children, and highlight the importance of looking at other factors in addition to a child's blood lead level when considering home remediation for that child. Nonetheless, the results of the current study indicate indoor lead flux might be one useful indicator in deciding on an appropriate management plan.

Limitations of this study require some discussion. The sampling strategy is a potential source of bias. The initial recruitment was designed to maximise even recruitment across districts, and this was supplemented by a second phase of purposive sampling to recruit a mix of house types and conditions within districts. However, all homes were recruited without knowledge of the children's blood lead levels minimising the potential for selection bias that would result in a spurious association.

A major consideration for the ecological analysis is that indoor lead levels were estimated for 'very well', 'adequately' and 'poorly' sealed homes in broadly defined regions, rather than for individual districts. Thus the estimate of average lead flux for individual districts might not be accurate, as, owing to the non-random sampling design, the actual proportion of homes in each condition class is unknown. However, the general pattern of indoor lead being highest in districts close to and downwind of lead sources is similar to that shown for lead in soil (Boreland et al. 2002) and deposited dust (Chompikul 1998).

Measurements of lead in both blood and indoor dust were available for a relatively small number of children (74 children in 50 homes), which limits the precision with which the effect of indoor lead on blood lead levels can be estimated. Despite these limitations, the relationship between blood lead and indoor lead observed for individual children was similar to that observed at the district level.

## Conclusion

This study has confirmed indoor lead as a potential source of lead for young Broken Hill children, although clearly many other factors affect blood lead levels as well. Children living in homes with high indoor lead levels are much more likely to develop significantly elevated blood lead levels; and indoor lead levels might also serve as a useful indicator for identifying children with high blood lead levels who are most likely to benefit from home remediation. Access to information on the risks posed by indoor lead will help the community recognise, and effectively deal with, this aspect of the lead problem. The data also help provide realistic targets for reduction in blood lead level following interventions aimed at improving house condition, and provide a benchmark against which the effectiveness of interventions can be measured. The relative importance of indoor lead compared with other sources of lead for Broken Hill children remains to be determined.

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Back to TOC

# Animal Model of Silicosis and Silica-induced Inflammation

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*Animal models are useful in studies of the mechanism of silica-induced effects and the testing of potentially effective drugs because such experiments cannot be carried out directly on humans. Different animal species might react differently in response to silica exposure. Rats appear to be the most ideal animal species for studying silica-induced effects because of both strong fibrogenic and carcinogenic reactions. Moreover, the different responses of various species to silica exposure might assist in understanding the mechanisms of silica induced lung damage in humans. Many studies have been conducted to elucidate the mechanism of silica-induced acute inflammation. These studies indicate that silica is a strong inflammatory agent and can induce an overt and rapid inflammatory response. Silica-induced pulmonary inflammation persists, evolving into the destruction of normal lung structure and function. It is well known that silica particles persist in the lung, but persistence of inflammation might be attributed to not only the persistent particles but also to the infiltrated inflammatory cells. Continued recruitment of inflammatory cells and the defective clearance of the infiltrated cells might be more important in the persistence of inflammation. After silica exposure, the capacity of macrophages for phagocytosis decreases significantly and this worsens the persistent inflammation. Because persistence of pulmonary inflammation predisposes to pulmonary fibrosis and possibly lung cancer, the understanding of silica-induced acute inflammation promotion of its resolution might prevent fibrosis and carcinogenesis.*

**Key words**: *Silica; Acute Inflammation; Persistent Inflammation; Fibrosis; Macrophages; Cancer*

## Animal Model of Silicosis

Animal models are useful in studies on the mechanism of silica-induced effects and the testing of potentially effective drugs because such experiments cannot be carried out directly in humans. The development of pulmonary inflammation and fibrosis in response to silica has been shown in various animals such as rats (Driscoll et al. 1991; Reiser et al. 1982), guinea pigs (Lugano, Dauber & Daniele 1982), mice (Callis et al. 1985; Suzuki et al. 1996), rabbits (Dethloff, Gilmore & Hook 1986) and monkeys (Hannothiaux et al. 1991).

Different animal species might react differently in response to silica exposure. In a study with rats, mice and hamsters (Saffiotti et al. 1996), silica induced differential fibrogenic and carcinogenic responses in the three animal species. The authors ranked the responses as shown in Table 1.

***Table1: Species differences in the lung reaction to silicaa***

| Species | Fibrogenesis | Carcinogenesis |
|---|---|---|
| Rat | +++++ | +++++ |
| Mouse | ++ | - |
| Hamster | - | - |

*Note: Relative Intensities: +++++ = strong; ++ = moderate; - = absent*

*Source: Saffiotti et al. 1996*

Similar results have also been obtained in another study (Carter & Driscoll 2001). From these results, rats appear to be the most ideal animal species for studying silica-induced effects because of both strong fibrogenic and carcinogenic reactions. Moreover, the different responses of various species to silica exposure might assist in understanding the mechanisms of silica induced lung damage in humans. Unfortunately, it is not clear what protective factors hamsters have, or what pathogenic mechanisms are not switched on, which result in an absence of lung reactions to silica.

## Silica-induced Acute Inflammation

Silica-induced acute inflammation is well described in numerous experimental studies. Yuen and co-workers (1996) reported that intratracheal instillation of silica in rats induced neutrophilic inflammation as early as 5 hours after instillation. Maximal infiltration of neutrophils into the lungs occurred 5 to 6 hours after the exposure. In another study with rats (DiMatteo et al. 1996), neutrophil influx was detected as early as 4 hours after intratracheal instillation of silica and the inflammation was preceded by initial damage which appeared 2 hours after exposure. These studies indicate that silica is a strong inflammatory agent and can induce an overt and rapid inflammatory response.

Many studies have been conducted to elucidate the mechanism of silica-induced acute inflammation. Yuen and co-workers (1996) demonstrated that chemotactic activity for neutrophils in bronchoalveolar lavage fluid (BALF) could be detected 2 hours after intratracheal instillation of silica and this was before the influx of neutrophils into the lung. The authors also detected macrophage inflammatory protein-2 (MIP-2) as early as 0.5 hour after instillation, but could not exclude the possibility of a nonspecific response to the intratracheal instillation. The gene expression of MIP-2 correlated with the generation of chemotactic activity and neutrophil influx in the acute inflammatory response following instillation of silica, but could not explain the sustained neutrophilic response.

Driscoll and co-workers (1993) demonstrated that intratracheal instillation of silica increased macrophage inflammatory protein-1$\alpha$ (MIP-1$\alpha$) and MIP-2 messenger ribonucleic acid (mRNA) expression in whole lung, and that increased gene expression preceded the accumulation of inflammatory cells. They showed that rat fibroblasts and epithelial cells are also probable producers of MIP-1( and MIP-2, and that tumor necrosis factor (TNF) might play a regulatory role in the production of these molecules. In a separate study (Driscoll et al. 1995), acute intratracheal instillation exposure of F344 rats to $\alpha$-quartz or titanium dioxide ($TiO_2$) was shown to markedly increase levels of MIP-2 and cytokine induced neutrophil chemoattractant (CINC) mRNA in lung tissue, and the response was associated with a significant increase in neutrophils in BALF.

Although MIP-2 has repeatedly been demonstrated to be associated with the initial infiltration of neutrophils into silica-exposed lungs, it cannot maintain a steady state but diminishes and disappears rapidly (Yuen et al. 1996). This is in contrast to the silica-induced neutrophil response, which has been shown in numerous studies to maintain a sustained state. Some research suggests that nuclear factor-kappa B (NF-kappa B) activation in BAL cells might play a major role in the initiation and progression of silica-induced lung inflammation, cellular damage, and fibrosis based on the observation that the initial activation of NF-kappa B in BAL cells occurs more rapidly than pulmonary inflammation, cellular damage, and cytokine production by BAL cells (Castranova et al. 2002; Porter et al. 2002). However, the exact mechanism for pulmonary inflammation in response to silica exposure is not yet fully understood.

## Silica-induced Persistent Inflammation

In contrast to acute inflammatory reactions induced by agents, such as lipopolysaccharide (LPS) and bacterial infections (which can resolve quickly with the return of affected tissues or organs to normal structure and function), silica-induced pulmonary inflammation persists, evolving into the destruction of normal lung structure and function. In mice, silica-induced pulmonary inflammation persisted for the entire experimental period of 6 months and neutrophils were the principal protagonists of the inflammation (Bissonnette & Rola-Pleszczynski 1989). In studies with rats, it was demonstrated that silica can induce an increase in BALF neutrophils, which can persist up to 63 days (the end of experiment) after silica exposure (Driscoll et al. 1991). A similar study reported that chronic inflammation characterised by granuloma formation, alveolar lipoproteinosis and interstitial pneumonitis existed even one year after intratracheal instillation of silica to rats (Reiser et al. 1983). There is no report suggesting that silica-induced neutrophil infiltration into the dust-exposed lung can completely resolve. Neutrophil numbers in BALF of silica-exposed lungs maintain a higher level compared with that of the control.

## Why Does Silica-induced Inflammation Not Resolve?

It is well known that silica particles persist in the lung although the defensive system can remove the particles gradually. Studies have demonstrated that silica particles remain in the lungs of rats for the entire experimental period of 1 year with little clearance from the lungs (Reuzel et al. 1991). The persistence of the silica particles might be the reason for the persistence of silica-induced inflammation. However, a soluble agent, bleomycin, which can be readily removed from the lung after a single intratracheal instillation (Smith et al. 1996), can also induce persistent inflammation which eventually evolves into interstitial fibrosis. This might be an indication that the induced response itself can also play a role in the persistence of the induced inflammation. Pulmonary inflammation induced by radiation exposure can persist (Bjermer et al. 1993; Nilsson et al. 1992; Yi et al. 1996) and eventually evolve into pulmonary fibrosis (Yi et al. 1996), whereas pulmonary inflammation induced by exposure to $TiO_2$ particles can eventually resolve (Brown et al. 1991). This might further support the hypothesis that the response itself can determine the resolution or persistence of inflammation, since $TiO_2$ can persist in the lung whereas radiation can cause injury without any foreign agents persisting in the lung.

A recent study indicates that there is probably a threshold lung burden above which silica-induced lung damage can develop without further exposure, and demonstrated that pulmonary fibrosis progressed even after silica exposure was stopped (Porter et al. 2004). It seems reasonable to speculate that persistence of silica particles might be cyclic, commencing with engulfment by pulmonary alveolar macrophages (PAM) and subsequent death of these cells, releasing agents harmful to cells as well as the original engulfed particles. That can cause further inflammatory reactions. This speculation is seriously compromised by a study (Iyer et al. 1996), which showed that exposure of human alveolar macrophages to silica particles *in vitro* failed to induce significant necrosis of the macrophages. Instead, apoptosis of macrophages, which is actually an inflammation-limiting process, was observed. However, since silica is known to be cytotoxic particles to alveolar macrophages and other lung cells, normal apoptotic mechanisms might be impaired or ineffective in resolving silica-induced inflammation. In addition, it has also been found that neutrophils rather than alveolar macrophages are the primary inflammatory cells that undergo apoptosis indicating the apoptosis of neutrophils might play a more important role than that of macrophages in response to silica exposure (Zhang, Hartsky & Warheit 2002).

The persistence of inflammation might be the result of, not only the continued recruitment of inflammatory cells, but also the defective clearance of the infiltrated cells (Cox, Crossley & Xing 1995). The latter, if not removed in timely fashion, might undergo lysis and release harmful substances causing further damage and assist in the perpetuation of the recruitment of inflammatory cells.

In chronic pulmonary inflammation, granuloma formation can occur. Granuloma, which can be either immune in origin or induced by foreign substances, is a cluster of cells, mainly macrophages and epithelioid cells, and a small number of lymphocytes (Majno & Joris 1994). Because of its insoluble nature, the inhaled silica can persist in the lung and lead to granuloma formation. In an experiment with rats (Yoneyama et al. 1993), intratracheal instillation of silica (50 mg per rat) induced typical granulomata in lung tissue as early as 4 days after instillation. Granuloma formation not only changes the structure of lung tissue and precedes fibrosis, but might also be associated with silica-induced carcinogenesis (Williams & Saffiotti 1995). The mechanism for the formation of granuloma is not completely clear.

## Functional Changes in Macrophages

After silica exposure, the capacity of macrophages for phagocytosis decreases significantly (Warheit et al. 1991a). Silica exposure can also impair the chemotactic ability of alveolar macrophages (Donaldson et al. 1990). Inhalation exposure of silica dust in guinea pigs caused decreased production of N-acetyl-D-glucosaminidase, cathepsin D and acid phosphatase by alveolar macrophages and decreased phagocytosis capacity of free lung cells including alveolar macrophages (Fogelmark et al. 1983). Incubation of mouse macrophages with silica *in vitro* decreased the ability of macrophages to phagocytose both erythrocytes and bacteria and inhibited the cell's ability to kill the facultative intracellular bacterium *Listeria monocytogenes*. The inhibition of phagocytosis of macrophages is dose-dependent (Zimmerman, Canono & Campbell 1986). Inhalation of silica dust in rats caused a pronounced breakdown in pulmonary dust clearance by macrophages (Privalova, Katsnelson & Yelnichnykh 1987). In an inhalation study with rats, it was found that the clearance of silica was significantly less than $TiO_2$ (Driscoll et al. 1991). Overall, silica inhalation exposure produced functional deficits in exposed pulmonary macrophages, as evidenced by measured reductions in macrophage phagocytic capacity and chemotactic response (Donaldson et al. 1990; Warheit et al. 1991a).

Compromised function of macrophages, especially phagocytosis, might lead to decreased capability of dust clearance (Warheit et al. 1991b) as well as the clearance of damaged cells and cell debris. If the clearance process is delayed, greater opportunity exists for these cells to release harmful agents. The compromised function of macrophages might also be reflected indirectly by increased sensitivity to infections. In fact, silica-exposed mice developed worse lung injury after virus infection compared with the non-exposed group (Jakab & Hemenway 1992). It has also been demonstrated that silica-exposed workers might be more subject to various bacterial infections (Cordes et al. 1981) and have increased susceptibility to tuberculosis (Aungkasuvapala, Juengprasert & Obhasi 1995).

Acute inflammation induced by silica exposure is the earliest known reaction of the defensive system. It is also the initiation of lung injury and the lengthy process of silica-induced effects such as fibrosis and cancer. The persistence of pulmonary inflammation is an abnormal condition causing compromised function of the lung. It also predisposes to pulmonary fibrosis and possibly lung cancer. Therefore, better understanding of silica-induced acute inflammation might help elucidate the following lung damage and intervention of silica-induced persistent inflammation might prevent fibrosis and carcinogenesis.

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Back to TOC

# Silica Carcinogenesis and the Possibility to Assess the Carcinogenesis by Micronucleus Formation in Alveolar Macrophages *ex vivo*

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*Silica is well known to induce inflammatory and fibrotic reactions. During these processes, various substances such as reactive oxygen species and cytokines can be produced. Silica itself, and its induced reactants, may cause genetic damage of lung cells and hyperplasia of pneumocyte leading to the development of silica-induced lung cancer. Micronucleus incidence in alveolar macrophages has been used as an index of potentially genotoxic inhalable agents, which may or may not be inflammation inducing. Since alveolar macrophages physically exist in the surface of alveolar epithelial cells and they will have the same* in vivo *exposure as the epithelial cells. Therefore, alveolar macrophages may be useful in testing potentially genotoxic agents which induce pulmonary inflammation since these cells are easily obtainable in large numbers as well as being long-lived and proliferative. One of the advantages of this method is that it can test not only the toxicity of silica itself but also its induced reactants.*

**Key words**: *Silica; Carcinogenesis; Mutation; Hyperplasia; Micronuclei; Macrophage*

## Silica Carcinogenesis

Silica can induce inflammation and subsequent fibrosis. During the inflammatory and fibrotic process, various substances such as reactive oxygen species (ROS) and cytokines are produced. These cellular products have been demonstrated to have important biological activities. Silica itself, and its induced ROS, might cause genetic damage of lung cells and silica-induced cytokines cause hyperplasia of pneumocytes. These may contribute to the development of silica-induced lung cancer.

## Genotoxicity of Silica

Silica was demonstrated to induce increased micronuclei in cultured Chinese hamster lung fibroblasts (V79) when these cells were directly exposed to the dust, or to the dust pretreated with simulated pulmonary surfactant, although hamsters are not good models in *in vivo* studies (Liu et al. 1996; Nagalakshmi et al. 1995). Silica failed to induce sister-chromatid exchanges (SCE) in cultured hamster cells (Price-Jones, Gubbings & Chamberlain 1980). The Ames test showed that silica is not mutagenetic in *Salmonella typhimurium* TA98, TA100, TA1535, TA1537 and TA1538 tester strains (Gu & Ong 1996).

In silica-induced micronuclei of cultured Syrian hamster embryo cells, chromosomal breakage and spindle damage might both be involved (Gu & Ong 1996). In silica-induced micronuclei of cultured V79 cells, spindle damage was considered to be responsible for the micronucleus formation (Nagalakshmi et al. 1995). The negative results for SCE and the Ames test, however, indicate that the genotoxicity of silica remains unresolved.

In *in vivo* studies, intraperitoneal (ip) injection of silica failed to induce micronuclei in the bone marrow of mice (Vanchugova, Frash & Kogan 1985). This might be because of species effects, as silica administration did not induce tumours in mice. Alternatively, as silica is not soluble in body fluid, an insufficient dose might have reached the bone marrow. Intratracheally instilled silica induced hypoxanthine phosphoribosyltransferase (HPRT) gene mutation in type II alveolar epithelial cells of rats *ex vivo* (Driscoll et al. 1995; Zhang et al. 2002). However, there is still a lack of data on the *in vivo* genotoxicity of silica.

Silica can persist in the lung if inhaled and can induce potent and persistent inflammation. This makes it important to test the *in vivo* genotoxicity of silica because the cellular response to silica exposure might modify its genotoxicity. It would be desirable to obtain cells locally exposed not only to silica particles but also to the inflammatory agents from the lung. It is also more valid to examine micronucleus induction without the artefacts introduced by the culture method.

It has been considered that the chronic inflammatory response plays a crucial role in mutagenic effects of silica particles on the HPRT gene in lung target cells (Driscoll et al. 1995, 1997). It has also been shown that the exposure to silica can cause oxidative DNA damage both *in vitro* and *in vivo* (Schins et al. 2002; Seiler et al. 2001; Yamano et al. 1995). Cytokines and ROS might be factors in both inflammation and genotoxicity (Borm & Driscoll 1996).

## Silica-Induced Hyperplasia

Type II pneumocytes occupy about three percent of the alveolar surface, but account for nearly 60% of the total number of alveolar epithelial cells (Haies, Gil & Weibel 1981). The interaction of silica particles and the lung epithelium can cause type II cell hyperplasia (Warheit et al. 1991) which, together with hypertrophy, was demonstrated in the lungs of rats exposed to silica by intratracheal instillation, 7, 14, and 28 days after a single injection of silica (Miller et al. 1987).

An *in vitro* study (Melloni et al. 1996) demonstrated that silica-exposed human alveolar macrophages can release a range of molecules bearing similarity to PDGF, fibroblast-derived growth factor (FGF), and insulin-like growth factor-1 (IGF-1), all of which can stimulate cultured type II epithelial cell proliferation and might be involved in the hyperplasia of this cell type in silicosis. Indeed, hyperplasia of type II epithelial cells in experimental silicosis is well documented and might be a pretumour stage in the development of silica-induced lung cancer. The alteration of cell to cell adhesion molecules and the loss of the epithelial phenotype have also been suggested as an early event in silica related lung carcinogenesis (Blanco et al. 2004; Saffiotti 2005).

## Silica-induced Lung Cancer

Silica has been demonstrated to induce lung cancer in rats by inhalation (Johnson et al. 1987) and intratracheal instillation (Saffiotti et al. 1996). However, silica failed to induce tumours in both mice and hamsters (Carter & Driscoll 2001; Renne et al. 1985; Saffiotti et al. 1996; Wilson et al. 1986). Also it is interesting to note that silica cannot induce significant fibrosis in either of these two animal species (Saffiotti et al. 1996). This might be an indication that an intrinsic host factor is important in silica carcinogenesis and that fibrotic reaction may be related to silica carcinogenesis.

Although silica is a well-recognised carcinogen in rats, the underlying mechanism for this is not clear. Intratracheal instillation and inhalation of silica in rats regularly induces alveolar proteinosis and interstitial fibrosis, in combination with a dose-dependent increase of type II cell proliferation rate (Friemann et al. 1994). These authors believe that these events might be a prerequisite for tumour development. There is some evidence that TGF-β isoforms might play a role in the pathogenesis of silica-induced adenoma and carcinoma (Saffiotti 2005; Saffiotti et al. 1994; Williams & Knapton 1996). Silica-induced inflammation leads to an overproduction of nitric oxide (Blackford et al. 1994) and ROS (Castranova 2004; Shi et al. 1998) which might play a role in currently accepted models of multistage carcinogenesis (Tamir & Tannenbaum 1996). CyclinD1 and CDK4 are also found to be involved in the development of silica induced lung cancer (Yan et al. 2004, 2005).

Inflammation was shown to play a role in silica-induced carcinogenesis in a study conducted by Driscoll and co-workers (1997). The study found continuing neutrophilic inflammation in rats 15 months after intratracheal instillation of silica, with evidence of epithelial cell hyperplasia. Co-culture of a neutrophil-enriched bronchoalveolar lavage fluid (BALF) cell population from *in vivo* silica-exposed rats with a rat alveolar type II epithelial cell line showed increased HPRT mutation frequency and this effect could be blocked by the addition of catalase. This study demonstrated that exposure of rats to doses of particles producing significant neutrophilic inflammation is associated with increased mutation in rat type II epithelial cells. The ability of particle-elicited neutrophils to exert a mutagenic effect on epithelial cells *in vitro* supports the role for neutrophils in the *in vivo* mutagenic effects of particle exposure. The inhibition of BALF cell-induced mutations by catalase implies a role for cell-derived oxidants in this response.

Summarising the above studies, silica is an experimental carcinogen in rats. It is not clear, however, whether the carcinogenic effect of silica involves a genototoxic or nongenotoxic pathway. The finding of *in vivo* silica genotoxicity needs to be confirmed experimentally without *in vitro* treatment of the target cells.

## Micronuclei in Pulmonary Alveolar Macrophages

Pulmonary alveolar macrophages (PAM) are present on the wall of the alveolar space and have defensive functions. They are the first protective line encountered by inhaled material. Since it has been reported that the altered alveolar macrophages functions from lung cancer patients might lead to an inability to stimulate anti-tumour immunity (Pouniotis et al. 2006), the impaired functions of PAM might also contribute to silica induced carcinoma. The origin of the PAM is possibly the pulmonary interstitial macrophage. It has been demonstrated that circulating monocytes can migrate into the lung and differentiate into PAM, especially in pulmonary inflammatory reactions (Blusse van Oud Alblas, van der Linden-Schrever & Van Furth 1983). After challenge by particulate or other agents, PAM can divide (Evans et al. 1973), but they do not divide in normal circumstances (Van Furth 1970).

Mutations can manifest as DNA sequence changes without discernible chromosomal aberrations as well as by cytologically observable chromosomal changes. Micronucleus formation might be considered as a type of mutation with chromosomal changes. Micronuclei are small nuclei that arise from chromosomal fragments resulting from chromosomal breaks (double-stranded DNA breaks), or detached chromosomes (microtubule malfunctions in cell division) (Choy 1996). Therefore, agents which break chromosomes (clastogens), and/or induce nondisjunction and other events which produce structural or numerical changes in chromosomes, can produce micronuclei (Brusick 1982). In micronucleus formation, if the site of mutation has no involvement in the gene, which is critical to the survival of the cell, the cell with the micronucleus/micronuclei can still survive and divide. The mutated gene might be inherited by the daughter cell(s) or the DNA fragment might be expelled in the subsequent cell division. Micronucleus formation can be used to assess the genotoxicity of chemicals. Micronucleus incidence has been evaluated in various animal species including rats, mice, hamsters and monkeys (Choy 1996) and can be scored in any dividing cell population (Fenech & Morley 1985). Micronuclei allow for the detection of both clastogens and agents which induce aneuploidy (abnormal cell division resulting in loss or gain of intact chromosomes) (Choy 1996).

An early explanation of malignancy, still widely held, is the somatic mutation theory which states that a tumour can arise by clonal proliferation from a somatic cell that has been transformed by acquired modification of its DNA base sequence. Currently, the most commonly held view of carcinogenesis is that virtually all malignant tumours arise from single cells that retain proliferative capacity by a complex, multistage process, in which both genetic and epigenetic alterations are important (Couch 1996).

Although not widely used in genotoxicity studies, micronucleus incidence in PAM is considered to be a valid and sensitive method to detect genotoxicants by inhalation or intratracheal instillation routes. Sahu and Das (1995) used mosquito coil smoke and mosquito mat vapour to test micronucleus formation in PAM of rats. The authors found that the incidence of micronucleated (MN) PAM was significantly elevated compared with controls. The micronucleus test method was validated by a similar dose-response curve to that of chromosomal analysis. An earlier similar inhalation study (Balansky et al. 1993) also reported that whole body exposure to tobacco smoke significantly increased the incidence of MN in PAM in rats.

De Flora and coworkers (1993) reported that the increase in micronuclei induced by inhalation of cigarette smoke can be prevented by administration of N-acetylcysteine (NAC). This might be an indication that micronucleus formation in PAM is related to its carcinogenesis since NAC is an anticarcinogen exerting its action via multiple mechanisms (De Flora et al. 1991a; De Flora et al. 1991b). In this study, the micronucleus response was quantified as the proportion of MN PAM in the total PAM population, to allow for possible changes in cell composition in BALF. Radioactive agents can produce increased micronuclei in PAM without producing an inflammatory response (Johnson & Newton 1994; Talbot et al. 1986).

In an *in vivo* study (Izzotti et al. 1996), rats were intratracheally instilled with air particulate extracts and examined for micronucleus formation and cytological alterations. It was found that the neutrophils were increased by 37% after treatment and this was accompanied by a relative decrease of PAM. The authors concluded that the changes in cellular composition were biologically relevant yet not statistically significant, due to marked interindividual variations. Since the authors intratracheally instilled the rats for five consecutive days for both treatment and vehicle-only control groups, and since the rats were killed three days after the last instillation, the large interindividual variation may be due to the consecutive intratracheal instillations.

From the studies reviewed above, it can be seen that the micronucleus incidence in PAM has been used as an index of potentially genotoxic inhalable agents, which might or might not be inflammation inducing. Since PAM physically exist in the surface of alveolar epithelial cells, one can assume they will have the same *in vivo* exposure as these cells. This might make PAM useful in testing potentially genotoxic agents which induce pulmonary inflammation since these cells are easily obtainable in large numbers, as well as being long-lived and proliferative. However, good negative and positive controls are needed for comparison.

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Back to TOC

# Dogs and People in Aboriginal Communities: Exploring the Relationship within the Context of the Social Determinants of Health

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*Aboriginal people and dogs have a very long association. The archaeological evidence suggests that the dingo, which was intentionally brought to Australia, was present from about 3500 years ago. Dogs introduced by European settlers quickly replaced or interbred with dingoes at Aboriginal settlements. The outsiders' view of Aboriginal dogs appears to be polarised into two distinct groups. Dogs are either described as a health risk and a reservoir for a range of diseases, or are glossed over as being sacred and ceremonially important. Neither view has really examined the complexity of Aboriginal relationships with the dog, or the fact that attitudes towards dogs might be variable from region to region, and that attitudes to dog and dog ownership are not culturally static. This paper provides a review of the anthropological literature concerning people's relationships with dogs and the perceived function of dogs in communities, supplemented by insights from research in South East Arnhem Land. It will then relate these findings to dog health and dog control programs and stress the importance of developing these within a community development framework.*

**Key words:** *Dogs; Aboriginal Australians; South East Arnhem Land; Relationships with Dogs; Public Health*

In memory of Phil Donohoe, who was killed in a tragic accident in December 2006. As the executive officer of Animal Management in Rural Remote Indigenous Communities (AMRRIC), Phil encouraged us to write this paper.

As Hamilton wrote in her 1972 paper, '*Aboriginal man's best friend?*', the dogs in Aboriginal communities tend to make a dramatic first impression on outsiders and it is unlikely that there is an anthropologist who has not been chased or bitten at some point during their careers. Despite this, after a brief flurry of attention in the late sixties and early seventies (Hamilton 1972; Jones 1970; Kolig 1973; Meggitt 1965; White 1972), dogs have appeared to be not worthy of anthropological attention. Perhaps it was considered that all that needed to be said on the subject had been. This is a serious oversight, as the above authors tantalisingly point to a wide range of practices and cultural attitudes towards dogs, and that such attitudes have the propensity to change (see especially Jones 1972). Further, there is considerable interest in the health of dogs in communities, the effects that dogs might have on the health of humans, and the best way to implement dog control, or health improvement programs. Anthropological involvement in such debates has been minimal. In this paper, we argue that it is important to be aware of how dog and human interactions in Aboriginal communities might be changing, in order successfully to implement programs that are going to be successful and supported by the community. We also argue that it is important to regard dogs and their health and wellbeing within a framework that encompasses an understanding of the social determinants of health and community development.

## The Human/Dog Relationship

Dogs were the first animals to be domesticated. Conservative dates for this association, based on human and dog remains being buried together in a grave in Israel, are dated to be 13,500 years BC (Tarcon & Pardoe 2002). Archeologists, such as Tarcon and Pardoe, however, suggest that the relationship is far older, perhaps 100,000 years, and that the interaction brought important developmental changes and benefits for both species, for example, they argue that humans learnt about pack hunting and the advantages of living in closely bonded groups from wolves (Tarcon & Pardoe 2002).

The French anthropologist, Claude Levi-Strauss, discussed the importance of dogs in describing how we describe and classify our world. Dogs and other animals, such as birds, are 'good to think with' he argues, and they tell us something about human social life. According to Levi-Strauss (1966, p. 204), "birds love freedom, they build themselves homes in which they live a family life and nurture their young, and they communicate by acoustic means recalling articulate language". He argues that because of these analogies to the world of humans, the world of birds can be seen as a metaphorical representation of the world of humans, so that when humans name birds ordinary human names are used. Domesticated dogs are quite different. Because dogs, at least in French society, are raised primarily for the companionship they provide to humans, the names given to them must reflect the fact that they are different from humans, yet in some sense part of human society. To Levi-Strauss (1966, p. 205), this means that dogs will not be given ordinary names, but rather names that are akin to "stage names, forming a series parallel to the names people bear in ordinary life". These include names such as Fido, Sultan, Azor, which are similar to human names, although rarely held by humans. The importance of this discussion about naming is that the names we call various animals and plants are from an underlying structure that informs us about the organisation of our society. The way we name birds in comparison to dogs, gives us clues about the ways in which we behave and think about them. In the case of dogs, their nearness to human society must be checked by deliberately giving them almost human, but not quite human names.

Of all animals, dogs are the most closely bonded to, and dependent on, humans. This closeness and length of association has resulted in the dog acquiring a unique status among animals. In many human societies, dogs are regarded as 'not quite humans' or as Serpell describes, as an "interstitial creature, neither person nor beast, forever oscillating uncomfortably between the roles of high status animal and low status person" (1995, p. 254). This is what Douglas would regard as a marginal and ambiguous state (1966, p. 118). People who do not fit properly into society are regarded as potentially dangerous and polluting and capable of inflicting misfortune involuntarily. It is clear in many cultures that dogs are also considered in this way. They might be invaluable in hunting, but still feared and distrusted, or at least considered to behave in ways that are unsettling to the social order.

## Aboriginal/Dog Associations

The ambiguity of the dog's status is evident in Aboriginal conceptions of their status and the mythology associated with them. Maddock writes that dogs in Aboriginal societies are classed as humans, but they constantly break human social laws, by mating indiscriminately and not following kinship rules (1972, p. 97). 'They act like dogs' is said of Aborigines who disregard marriage restrictions (Maddock 1972, p. 97). A similar idea emerges in Berndt and Berndt's oral histories describing the trickster Bomoboma (NE Arnhem Land). This particular character is considered to be abnormal because he flouts social rules by having a series of illicit relationships. This breaking of rules and unpredictable behaviour is described as "running about like a dog" (Berndt & Berndt 1999, p. 407).

Kolig (1973, p. 123), describing the beliefs of the Wolmadjeri people of south Kimberley, argues that the main function of dogs is to warn humans about the approach of evil spirits. In the mythology of the region, the dog is represented as a dangerous animal, and this is the reason why people are loath to kill them.

Meggitt's study of the Walbiri in the period 1953-1954 offers some insights as to the relationship between dingoes and humans. The Walbiri captured dingoes as pups and tamed them, and on reaching maturity, the dingo usually returned to the bush. Although the Walbiri stated that the reason for acquiring pups was to train them to assist in hunting, Meggitt found that hunters who relied on skill and stealth were more effective without dogs (Meggitt 1965). Dingoes tended to forage for themselves, and it is for this reason Haydon argues that although dingoes might not have been great contributors to the diets of people, it was a low maintenance helper in the food quest and therefore worthwhile (Haydon 1975). Although Meggitt (1965) found that people were unwilling to kill any tamed dingoes, they had no such compulsion about wild dingoes, as they became involved in procuring dingo scalps for monetary reward.

European dogs were quickly adopted into Aboriginal life, even among the Tasmanian Aboriginal people who had not had dingoes previously (Jones 1970). Unlike dingoes, European dogs were less likely to hunt for themselves and became dependent on their owners. They did not return to the bush on reaching maturity, and bred within camps.

Jones describes the situation in Tasmania where Aboriginal people were only exposed to dogs from the period 1798-1804 because the dingo had not reached Tasmania. Despite this, dogs were readily incorporated into Aboriginal life and people changed their hunting techniques (Jones 1970).

> *Not only did the Tasmanian Aborigines not use dogs, they did not even know of their existence. In this total ignorance of the animal they were probably unique amongst the ethnographically known peoples of the world. Yet within a few years of seeing their first dogs, the Tasmanians had recognised the potentiality of the animal, formed close bonds with it, and had incorporated it fully within their culture* (Jones 1970, p. 259).

In Yalata, in South Australia, White observed that the people there made a distinction between hunting dogs and the rest. Hunting dogs were well trained and fed by their owners, while the others were not. Although neglect was evident for the non-hunting dogs, no one would kill a dog. White argues that hunting dogs made an important contribution to the food quest and that they were particularly effective in cornering and bringing down large kangaroos (White 1972). Indeed, White argues that the use of European dogs represents a significant innovation in hunting technique, which greatly improved the hunters' success:

> *The high proportion of successful kills made by the dogs, once kangaroos are sighted, leads me to believe that good hunting dogs have increased the supply of game food available in comparison with the old tribal times* (White 1972, p. 203).

In contrast, Hamilton, working with the Jankantjara people of the Everade Ranges, argues that the domestic dog's contribution to the food quest is small. She comments that dogs are more often in competition with people for food due to their foraging activities and theft of food (Hamilton, 1972). Affection for puppies was the main reason for keeping dogs:

> *Puppies provide a special emotional release for nurturing behaviour which normally would be expended on human children, but which is limited in its full expression in an environment which does not support a large human population* (Hamilton 1972, p. 294).

Another insight into the dogs' status is that dogs and children often appear to be rivals, they fights over food and they have a high level of interaction that is largely unchecked by adults. As children get older, they try to assert their dominance over dogs. It is often pointed out that children can be cruel to dogs, for example:

> *Puppies do not receive such tender care at the hands of the children, however, and probably the major cause of pup mortality is the constant 'play' that they suffer. No matter how devoted one might be to a pup, a child usually has precedence, and if a two year old cries to be allowed to carry a new-born pup about by its neck then no one will gain-say it* (Hamilton 1972, p. 289).

Dogs, however, often snatch food away from unsuspecting children:

> *Children learn early to eat standing up and holding their hands high; the small hand hanging at the side with a piece of food in it is a quick target for the dogs* (Hamilton 1972, p. 290).

Perhaps the most important insight into the status of dogs is shown when a dog is killed, either deliberately or accidentally. Berndt and Berndt (1999, p. 345) describe a mythological fragment that contains an important warning that retribution for the killing of dogs will be severe:

> *But offences against dogs, which are regarded almost as members of a family rather than as personal property, might have violent repercussions. In Western Arnhem Land, for instance, in one mythical case, several large camps are said to have been wiped out after a man's special pet dog was unknowingly killed and eaten.*

## Human-dog interactions in an Arnhem Land community

The following discussion of human dog interactions in an Arnhem Land community is based on anthropological work carried out in the community between 1999-2006. The South East Arnhem land community of Ngukurr is home to about 900 people and 280 dogs in 2006. This represents about three dogs per household. Many, but not all of these dogs are named and incorporated into the local kinship system. The kinship system in Ngukurr divides the world into two moieties. A moiety is one of two descent groups in a given population who usually intermarry. A descent group is a kin group whose members are recruited by one of the principles of descent; for example, matrilineal, patrilineal, or so on. In Ngukurr society the two moieties Dua and the Jiridya are further divided into semi-moieties and again divided into subsection or skin names. At birth, each child is given a skin name, which establishes that child's place within the descent group and sets rules for how the individual interacts with everyone else in the group.

There are strict rules governing marriage. A preferred marriage pattern is for a person to marry their mother's brother's daughter's child. A fundamental rule regarding marriage is that it must be exogamous. You must marry someone in the opposite moiety to yourself. The result is that you will be in a different moiety from your mother and you will be in the same moiety as your father.

A person's dog has an equivalent skin name to his or her own children. Dogs, however, cannot be expected to marry 'right way' and so puppies are either classified as if their mothers had chosen the correct partner (as would be done in a human wrong way marriage, which is a process described as 'straightening up'), or they acquire a new skin name through adoption by another human. The dogs' disregard for exogamous mating practices, are, as described by Maddock above, something that keeps them fundamentally not quite a human.

As well as a series of named dogs, there are also dogs, which are loosely attached to households, but not owned by anyone. These dogs are described as *Gubalga* (scavenging dogs) or *Walgnulu* (lost and lonely dogs). These dogs are never deliberately fed, but might obtain food through eating discarded remains or fighting more favoured dogs. Their survival is a matter of chance, with very little human intervention. For example, at one house only three of the nine dogs had names. The others consisted of a female dog and her puppies. The mother dog eventually left the household to scavenge at the shop. These dogs are not regarded as being useless. They combine with the other dogs to create a body of animals protecting a household from both human and spirit intruders. Sorcery is an ever-present threat in Ngukurr, as is the concern about strangers entering the community (Senior 2003). People comment that they feel much safer when they are surrounded by a large number of dogs. Protective dogs mean that visitors are forced to remain distant to the house and call out loudly to make their presence known: “When you visit houses you have to stand back and call long way, because of all the cheeky dogs”. The larger dog population as a whole, is also perceived as having special intuitive powers, for example, it is believed that dogs sense human deaths and have an important role in alerting community members: “When someone dies all the dogs start howling at once, top, middle and bottom camps all together, then you worry about who it was”.

There are two categories of important or valued dogs in the community. One is the traditionally valued category of hunting dogs, the other, we argue is an emerging category of pampered pet. Dogs that are described as hunting dogs are named, and often have an important ceremonial name. For example, one dog bears the name Mumbali that is a Dua subsection name. Hunting dogs are valuable and people often talk about buying such dogs from outside the community or from visiting non-Aboriginal people. It is not often assumed that a local scavenging or lost dog can be turned into a hunting dog. As an example of this, one of the authors (Senior) befriended a local lost dog, Spike, and eventually, after he was fed and treated for mange and other parasites, he became a strong and handsome dog. Local people offered to buy this dog and expressed their disbelief when she said it was actually a local dog in the first place.

Although people talk about the hunting prowess of dogs, the opportunities for them to demonstrate their skills are limited. The authors went on few hunting trips that included dogs. People talk about dogs being particularly useful for goanna hunting, but the number of goannas around the community has been dramatically reduced after the cane toad moved into the region. Despite the value given to particular dogs, it is often difficult for the outsider to distinguish them from the main dog population. They have considerable autonomy, are allowed to wander freely, are fed when they are close to the household and frequently look mangy and neglected.

The other groups of named dogs are pet dogs, which could not possibly have any role in hunting. In Ngukurr, there is a group of Chihuahuas, which are highly valued and treated by their owners as special pets. These dogs are generally well fed, are allowed inside the house and wear collars. One in particular wears a collar with a tag reading ‘spoilt’ and is in stark contrast to the dogs that surround it. At about the same time (in mid-2004) as this new category of dogs emerged, it became possible to buy tinned and dried dog food and dog grooming products at the local store. This change in dog ownership and grooming practices was heavily influenced by celebrities and media images, which were widely circulated in the popular media at the time. As individuals began acquiring special pet Chihuahuas they began lobbying the local storeowner to start selling various dog products. It is important to recognise that this change was driven by consumers and this highlights how important changes in health related behaviour might be influenced as much by fashion as by education (Lindenbaum 1989). Public health education campaigns must therefore be aware of the complex social and political context in which they are operating in order to maximise opportunities for changes in behaviour. While calls for improved dog health in communities are often associated with various interventions such as sterilisation or culling, the change in some of the Ngukurr residents' dog ownership practices suggests that changes at the level of the individual are intricately linked to the process of forming and expressing identity (Zukin & Maguire 2004).

Dogs are deeply embedded in the social life of the community; they are present at most important activities. People commented that dogs were restricted from accompanying the men to ceremony, but they were expected to follow the women, in the same way that children accompany women. Dogs also get involved in disputes between families. In a recent fight over a wrong skin marriage, the dog of one party bit an opponent. The person who was bitten swore that he would retaliate, while the owners of the dog swore that if anything happened to the dog, they would get their retribution, and thus the dispute continued.

## The Importance of Understanding Dog Health in the Context of Society

The important point for this paper is that in discussing the relationship between dogs and health, we have to talk about a wider concept of dog health that includes humans. Dogs are part of the physical environment, but they are also part of the human social environment, in the relationship they form with humans. As we have shown throughout this paper, dogs are involved and interconnected in a number of human social activities. So any argument about improving the health of dogs is also one about the health of the humans with whom they co-exist. This fits comfortably with current discussions about the social determinants of health (Carson et al. 2007) and has long been recognised by environmental health practitioners in Australia in Indigenous and non-Indigenous settings.

At the centre of many discussions concerning the social determinants of health, which might include a focus on education, housing, income and racism, is the finding that Australians at the lower end of the socioeconomic hierarchy suffer more ill health and that those health differences by socioeconomic position, are apparent at all ages. While poorer people are more likely to go to hospital and seek medical care, they are less likely to take advantage of preventive care and screening services. These inequalities are apparent from the earliest of ages among Australian children. Children from lower socioeconomic groups tend to have lower birth weight, higher rates of developmental problems and are more likely to experience poorer adult health, than children from higher socioeconomic groups (Najman 2001).

The question in much of the literature on the social determinants of health has been oriented to uncovering which social determinants are related to health outcomes. The publication that has been most influential in promoting this approach, Marmot and Wilkinson's (1999) *Social Determinants of Health*, sets the scene unambiguously in the foreword:

> *The health of populations is related to features of society and its social and economic organisation. This crucial fact provides the basis for effective policy making to improve population health. While there is, understandably, much concern with appropriate provision and financing of health services and with ensuring that the nature of the services provided should be based on the best evidence of effectiveness, health is a matter that goes beyond the provision of health services* (Acheson, in Marmot & Wilkinson 1999, p. xi).

It is the focus on the social environment, rather than dog health services, or individual dog psychology and behaviour, which defines this approach. We would argue that in order to understand and to alleviate the poor health of dogs in Indigenous communities, further study needs to investigate the social environment of dogs as they interact with human environments. Studies examining the health of individual dogs need to take into account that dog health is influenced by broader structures around them, such as current environmental and housing infrastructures, which will determine the capacity of individuals and groups to provide shelter, food, and health for dogs.

As an example, we can examine the situation of the dog Spike, described above. With veterinary attention and a good diet, he was transformed from a lost and unwanted dog to a dog that people valued. But there was a considerable cost associated with this transformation. When the first author had access to a car, she was able to take Spike to the vet in Katherine, some 300 kilometres away. Senior was also able to purchase dog food (which was not available in the community at the time) and treatments for his mange. This level of expenditure and especially the need to drive to Katherine for veterinary treatment would have been impossible for many community members who survive on government welfare payments. Currently, the community receives regular visits from a vet and the dogs appear to be in particularly good health.

One could also look at the issue of overcrowding in community houses. In some communities, arbitrary rules have been imposed, whereby households are limited to a maximum of three dogs, which reflects the restrictions in major urban centres. In urban centres, households are home to one, often a nuclear family, but this is not the case in remote Aboriginal communities where a house might be home to several families. Dogs are individually owned, and therefore imposing restrictions on dog numbers would mean that some individuals were missing out on the opportunity to own a dog. In this case, human overcrowding and dog overcrowding are strongly linked.

## Conclusion

Any account of dog health in Indigenous communities should also take into account Indigenous health and the social determinants influencing health as they are interrelated. Understanding the socio-cultural and economic context in which dogs are situated is critical if effective programs are to be developed and delivered. As illustrated in the Ngukurr case study, dogs and humans are intricately linked and importantly this link has changed over time incorporating new perspectives and dog keeping practices. As the social and economic climate of social groups changes through time so too will their relationship to the animals they choose to share their environment. The implications of this paper for environmental health practitioners are the benefits to be gained by practitioners enhancing the skills that enable them to explore the social and cultural dimensions in a particular place.

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Back to TOC

# Looking Forward: Environmental Health Planning at the Local Government Level in Western Australia

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*This article describes the process used to develop a forward functional service plan for Health Services within the City of Swan, Western Australia (WA). The City of Swan is the largest metropolitan local government area within metropolitan Perth, and is one of the fastest growing urban corridors in Australia. Historically, in Health Services, environmental health priorities had been identified through a range of systems including environmental health complaints and past experience. Health Services acknowledged that these planning indicators were not based on evidence and were inward looking. In late 2004, the City's Health Services committed to the development of a new forward functional service plan (FFSP) to encourage innovative and contemporary service planning. This article does not attempt to provide the results of the planning process, but merely attempts to describe the processes that resulted in the ratification of the Health Services FFSP. The aim of the planning process was to provide a plan that would help facilitate the development of new partnerships, would ensure good program management, included an evaluation process, and would enable capacity building of internal staff. In addition the plan provided a clearer rationale for what programs are provided and why, is sustainable for reviewing and developing in the future, and flexible to changing the selected priority program areas in the future, a 'plug in and play approach'.*

**Key words**: *Environmental Health Planning; Local Government; Evidence Based Planning*

The Local Government Authority of the City of Swan (CoS) is located in the north east of the Perth metropolitan area approximately 16 kilometres from the central business district. Swan covers an area of 1042 square kilometres amounting to approximately 20% of the Perth Metropolitan Region. Ranging from the urban and industrial suburbs, through the regional and historic centres, to the rich Swan Valley and rural areas, the City hosts a diversity of economy, culture and environment (CoS 2005).

The current population of the City is 91,354. However, the average growth rate is 3.2% and the population of the City is projected to be 145,000 by 2021 (ABS 2004). The City includes large amounts of land marked for future urban development under the Metropolitan Region Scheme, and it is anticipated that development of this land will accommodate a significant proportion of growth for metropolitan Perth and a doubling of the population of the City of Swan over the next 30 years. The median age of the population of the City is 31 years of age. The Swan region also has the largest proportion of Aboriginal people of any local government area in WA, and a heritage of migrant settlement.

## The Role of the Health Services Unit in the City of Swan

Local government in Western Australia forms the third tier of government in a federal system of states and a central, national government. As such the City's primary administrative relationships are with state government departments, and to a lesser degree with federal agencies. Local government environmental health departments in Western Australia administer a wide range of legislation, including that relating to food, environmental nuisance, noise, recreational water safety and large public events. Beyond the regulatory role, environmental health teams influence behaviour change through education and promotion interventions as well as ensuring health concerns are clearly placed on the agenda in strategic and local planning matters. The City of Swan's Health Services unit also takes on a community advocacy role on particularly significant local or regional environmental health issues, regardless of whether there is a direct regulatory role. This has included issues such as contaminated sites, regional air pollution issues, local government responses to pandemic influenza, and acid sulphate soils.

The role of Health Services in the City of Swan is to manage known environmental health risks so as to promote an environment that is supportive for the health and wellbeing of the community. In 2004, the City's Health Services committed to the development of a new forward functional service plan (FFSP). The FFSP was seen as an expansion of the vision and mission statements. The Health Services FFSP lies between the Corporate Plan and the Unit's annual business plan. The primary difference between this plan and a business plan is the integration of futuristic options for program areas and service delivery. The FFSP identifies ways to improve how Health Services will plan, implement and evaluate their identified program areas. The FFSP belongs to every team member in Health Services and is designed to be a proactive guide for action and a reference tool.

The primary environmental health programs managed by Health Services at the City of Swan include the following:

- Food safety program - food premises and production of food;
- Public buildings risk assessment and public safety;
- Pollution control program including local pollution and contaminated site issues;
- Environmental noise program;
- Safe Water Program - chemical/ microbiological/administrative safety issues in public swimming pools;
- Health nuisance management/ complaint resolution;
- Onsite effluent disposal approvals;
- 24-hour call out capacity;
- Large public event risk management program;
- Mosquito Control Program; and
- Range of other services from pest control advice to health and safety in lodging houses.

### *Previous processes used to identify environmental health priorities*

Historically, Health Services identified environmental health priorities through systems such as assessment of current workload, complaint and approval statistics, risk management processes to identify the potential risk to public health, the type and number of premises in the City, corporate or community priorities and through the National Environmental Health Strategy. There was also an element of tradition, where some services were simply provided based on historical precedents. While this approach to business planning had served Health Services well in the past, it was an essentially inward looking process. This resulted in Health Services being constrained by 'traditional' environmental health risks to the exclusion of contemporary risks. As such, Health Services struggled for identity within the organisation and, more importantly, within the broader community.

The staff in Health Services sought to change this modus operandi and aimed to identify clearly environmental health priorities based on evidence. In the climate of changing public health issues, increasing expectations of the community and resource contraction, the ability to identify program priorities clearly and with accountability, was paramount. In addition, staff were experiencing pressure from external drivers, such as contained resources in a rapidly growing local government authority, and continued expansion in roles and expectations of services by others, including councillors and members of state government. The devolution of powers and the introduction of new legislation, the changing public health risks from nuisances to modern issues such as pollution and non-communicable diseases, and the social determinants of health have all contributed to this increased pressure. The development of a FFSP aimed to address these concerns, to be inclusive of new approaches to the planning of services, and to provide a sustainable planning framework for future changes.

## Key Elements Required in New Forward Functional Service Planning Approach

From an early stage in the planning for the FFSP some key elements were identified. These included a process that was able to be clearly documented and followed, was ultimately endorsed by the organisation, and validated by other stakeholders, that encouraged the introduction of a range of stakeholders to help determine outcomes, and that helped to facilitate the development of new partnerships. Additional elements, such as ensuring that the process provided good program management to agreed programs, was able to be evaluated, and that bridged the gap from the bottom of the corporate strategic plan to the annual business plan. It also needed to cover operational daily activities and enable capacity building of internal staff so that they are able to implement, evaluate and review the plan. All of these were considered important.

The team also believed that a sense of ownership of the plan by those charged with implementing it was important. The provision of a clear rationale for what programs are currently provided and why included a process that involved evidence-based decision-making, and ensuring that the plan was sustainable and flexible to changes. The possibility for such changes needed to encompass corporate plans and approaches and be flexible to changing the selected priority program areas in the future, a 'plug in and play approach'.

It was also important to note the corporate imperatives or givens that were implicitly applied to all Health Services program areas and service delivery. These givens included those elements that were not within the control of Health Services. These were things such as ensuring the FFSP was based on the current Corporate Strategic Plan and the values and principles included within it (Place Based Management, Best Value Approach, Self Help, Sustainable Development and Business Excellence). The plan needed be reviewed easily to align with the next review of the City of Swan Strategic Plan.

As environmental health has a largely statutory base, it was also important to ensure that the core statutory role which applies to the majority of work in environmental health as it relates to local government in the Health Act and other legislation, was integrated into the plan:

- Ensuring the integration of the City's approach to customer service into all services provided; and,
- Ensuring the Health Services Risk Management Policy was considered inherent in all program and service planning and implementation.

## Methodology

The development process for the FFSP was designed as a staged process with full involvement from the Health Services staff to ensure the transferring of capacity. A consultant was contracted to lead the process. The basic steps of the FFSP are described below but can be expressed as:

1. Literature review
2. Formation of a cross functional team (CFT)
3. Appreciative Inquiry Workshop
4. Development of a matrix for identifying priority program areas
5. Development and administration of a program area service issues survey
6. Analysis of data from the service survey
7. Drafting of the FFSP
8. Development of an evaluation framework
9. Ratification by Council.

### *Literature review*

A literature review of similar environmental health planning processes undertaken in Australia and overseas was conducted. This review focused on a range of issues including links to local, regional, state, national and international environmental health policies, examples of other environmental health planning tools, and case studies from around the world. This review provided evidence about quality practice in the area of environmental health planning. An internal policy analysis from the City of Swan identified links between the Health Services' core business and other corporate activities.

## Formation of a Cross Functional Team

A cross-functional team (CFT) was established to lead this process in partnership with the consultant. The roles of the CFT included participating in the planning, development, implementation and dissemination of the FFSP. Internal Officers from the City of Swan and external stakeholders were members of the CFT.

### *Appreciative Inquiry workshop*

Appreciative Inquiry (AI) is an approach to organisational development and change that grows out of social constructionist thought (Reed 2006). The AI approach offers a process and potential to explore positively, collectively imagine, collaboratively design and jointly commit to a path forward. All staff from Health Services attended this full day workshop. Outcomes included:

- The development of a mission and vision;
- The identification of core tasks;
- The provision of a participative dimension to complex planning;
- The obtainment of support for structural changes and for working collaboratively across council departments;
- The identification of ways to engage communities and external stakeholders in matters ranging from policy development to resource allocation; and

- The identification of the existing key tasks that the Health Services Unit was currently addressing.

### *Development of a matrix to identify priority program areas*

Following the analysis from the AI workshop, it was clear that for the FFSP to be achievable, existing core tasks needed to be prioritised to a smaller number. To facilitate this task, a priority program matrix was developed. A number of amendments were made to the matrix as it developed and discussion was held regarding the program areas to be included.

In the absence of any corporate process to delineate between discretionary and non-discretionary services, the priority program matrix framework was considered to be the best approach to prioritising work programs. Each program area was defined for ease of use for external partners, and to ensure internal staff held a consistent understanding of the areas.

A series of horizontal indicators were developed in response to issues and policies that were relevant to both the City of Swan and external agencies. Indicators contribute to the overall environmental health goals and support the goal through following key applications, such as advocacy, accountability, system management, quality improvement and research (Anderson, Brown & McColgan 2003). The indicators used in this process included:

- Statutory requirement of CoS;
- CoS Corporate priority;
- CoS Place Planning priority;
- Department of Health (DoH) priority;
- Reporting requirement to DoH;
- Potential partnerships;
- No other jurisdiction covering this issue; and
- Supporting Public Health Unit data /epidemiological data.

Internal policy direction, such as place planning and links to the City's Strategic Plan, were considered imperative to include. This was to ensure that the FFSP was aligned with corporate direction. The priorities of external agencies such as the national environmental health priorities, the Western Australian DoH environmental health priorities and local Public Health Unit issues were considered to be useful to include in the decision making process. Again, this was to ensure a close association with regional, state and national directions. Reporting requirements and legislative imperatives were critical to include, as they are core business for Health Services. Finally, the listing of potential partners who might provide assistance with program area strategy implementation was considered useful in identifying areas of intersectoral significance and avenues for sharing scarce resources (Nutbeam 1996).

The weighting system was the final validation technique for selecting the priorities. Its use resulted in an improved understanding of the risks associated with environmental health. The weighting variables were:

- High risk associated with magnitude of worst likely effect;
- Large distribution of risk across the population of Swan; and
- Multiple risks across all areas of the CoS.

Each program area was allocated a weighting of 1,2 or 3, with 3 being the highest risk weighting. The matrix was purposely designed to be user friendly. A tick was placed in each column where action had occurred in that particular vertical descriptor. For example, if Swan had

***Table 1: An example of the environmental health priority matrix***

| Service Issue | Weighting 1, 2 or 3 | City of Swan priority | enHealth Priority | WA Env Health Priority | City of Swan Place Planning | Reporting imperative to DoH | PHU data | Legislative requirement | Jurisdictional coverage | Potential for partnerships |
|---|---|---|---|---|---|---|---|---|---|---|
| Pollution management | 2 | ✔ | ✔ | ✔ | ✔ | | | ✔ | | |

developed a formal partnership with the Public Health Unit in a program area, a tick would be placed in the partnership column. One additional column was added to allow for any qualitative comments to be made about a program area, such as a local political imperative. An example of this matrix appears in Table 1.

The primary purpose of the priority matrix was to provide a rationale for why the issues listed were core tasks for Health Services. The rationale is evidence, statutory, corporate priority and best practice based. It provided the staff with a tool to assist in determining its priorities and level of service provided, taking into account legislative requirements, policies of the CoS, stakeholders views, and state and national environmental health agendas.

The matrix was piloted with four members of Health Services to test for reliability and validity before being validated by the remainder of Health Services and the Cross Functional Team. The results of the prioritisation process were eight priority programs that had the strongest links to statutory requirements, corporate planning and priorities, and environmental health priorities for the community.

### *Development and administration of a program area service issues survey*

In addition to the priority matrix, a service survey was developed. The aim of this survey was to flesh out the service issues associated with each of the priority program areas. This process also signified all the ingredients needed to develop and manage a best value service. The service survey comprised eighty questions under the following categories:

- Service planning;
- Service delivery;
- Community needs;
- Community education;
- Resources;
- Supportive policy environment;
- Data management;
- Identity;
- Evaluation; and
- Partners.

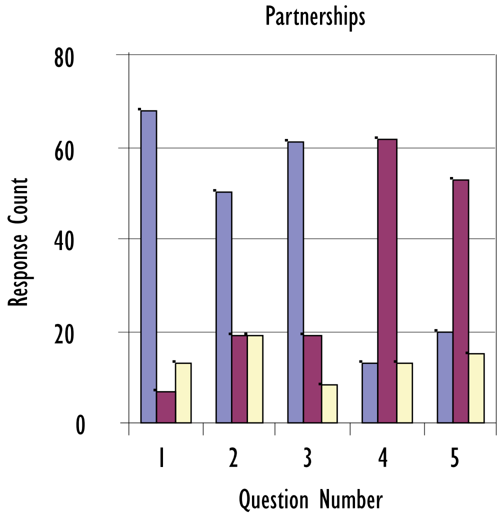
The survey was administered through an excel spreadsheet. All questions were quantitative. A team meeting took place prior to staff completing the survey to ensure all staff members were aware of the process, their obligations, and the process to resolve or clarify any issues. Each staff member completed the survey for each program area. One of three responses was used to answer the questions. These were: 'no', 'to some extent' or 'yes'. The surveys were completed over a two-week period. Staff did not discuss their responses until all surveys were complete.

## Data Analysis

A team of Heath Services staff together with the consultant analysed the data from the service survey. Descriptive statistics were used to indicate response rates and aggregated data. The data highlighted both weaknesses and strengths in each of the program areas and indicated areas to be examined in the FFSP. Graphs were commonly used to illustrate findings.

An example of the type of data (including the questions asked across all priority service issues) resulting from this phase is illustrated in Figure 1.

***Figure 1: An example of the data resulting from the partnership service issue questions across all priority areas***




Questions Asked:

1. Do you work with external partners on this issue?
2. Do you work with internal partners on this issue?
3. Have you accessed the relevant government organisation to gain assistance/share info with this issue in the past 3mths?
4. Have you gained assistance from/shared information with any NGOs on this issue in the past 12 months?
5. Have you gained assistance from/shared information with the community on this issue in the past 12 months?

The results were categorised using the 10 headings from the service survey rather than under individual program areas. The decision to illustrate the data in this way was based on the considerations that Health Services did not want to develop a series of silos for program areas. It wanted: to facilitate cross team collaboration on program areas; to ensure that service issues were a 'best fit' with current political imperatives and planning processes; to provide a service matrix for ensuring good program management, into which any future program area could be inserted and evaluated against; and wanted to focus on the outcome of effective and efficient program management and service delivery of program areas, whether by Health Services, other internal functional areas, external agencies or any combination of above.

### *Drafting of the FFSP*

It was decided that to ensure ownership and improved implementation of the FFSP, Health Services would write the FFSP. Therefore, all Health Services staff members were given a refresher on program management prior to the commencement of the writing process. The purpose of this training was to ensure that all staff had the basic understanding of the process and framework to be used. The consultant conducted the training and supplied a proposed template for drafting the plan. When the templates were complete for each priority program area, the team workshopped the proposed strategies to ensure consistency, approval and achievability.

The basis of the final FFSP is the 10 service plan templates based on the 10 service survey issues. Within each of these templates, the eight program areas are addressed. Each service plan template addresses the key program area 'weaknesses' identified from the service survey as well as the key 'good' program management needs identified. In light of the fact that this is the first FFSP, many of the activities identified relate to strategic program management needs across all program areas. It is expected that in future reviews and revisions of the plan a greater percentage of actions will be specific to particular program areas. Some initial discussion about which services might be able to be outsourced in the future has commenced and this issue will be a focus for the first revision of the FFSP.

### *Developing an evaluation plan*

The evaluation framework was developed as a practical, non-prescriptive tool, designed to summarise and organise essential elements of program evaluation. It provides a common frame of reference for conducting evaluations across all program areas. Adhering to the steps and standards of the framework will allow an understanding of each program's context and will improve how program evaluations are conceived and conducted.

The framework will assist in answering the following questions for Health Services at the City of Swan:

- What is the best way to evaluate? ;
- What are we learning from evaluation? and
- How will we use the learning to make Health Services efforts more effective?

The evaluation plan proposes that evaluation should be integrated into routine program operations and the emphasis is on practical, ongoing evaluation that involves all program stakeholders, not just evaluation experts. The six-step framework provides a starting point to tailor an evaluation for a program area at a particular point in time. The steps are interrelated and might be encountered in a nonlinear sequence. The evaluation framework is underpinned by four key principles which are utility, feasibility, propriety and accuracy.

## Ratification by Council

Following review and final comments from the Cross Functional Team (CFT), the plan was formally endorsed by the CFT in February 2006. It was also agreed by the CFT to refer the plan to the following additional stakeholders for final comment and endorsement including:

- City of Swan - Community Services;
- City of Swan - Corporate Services;
- Environmental Health Directorate - Department of Health; and
- Pollution Response Unit - Department of Environment.

In addition, the plan was endorsed by the City's Executive Managers' group and was presented to Council at a briefing session and before Council for endorsement at an Ordinary Meeting.

## Conclusion

The development of the FFSP for Health Services at the City of Swan aimed to allow the staff to move beyond traditional approaches to environmental health and seek innovative strategies in dealing with established, new and emerging environmental health issues. This plan succeeded in identifying a range of evidence based environmental health management solutions to meet the needs of the residents of the City of Swan. A partnership and intersectoral approach was used to progress the plan and subsequently ensured that many views were sought and heard. A key component to the success of the plan was the use of the Health Services staff members. This strategy not only ensured that the plan was relevant to core business but also allowed for capacity to be built and enhanced among staff.

In the short term, this FFSP will be used to build upon existing actions and achievements and seek to firmly ground environmental health within a supportive policy and decision making framework. The objectives and strategies listed in this Plan are key elements to its success.

In the longer term, the FFSP will be reviewed to reflect and address issues as they emerge. This process of continuous improvement will ensure that environmental health in the City of Swan remains at the forefront of decision making processes and is managed in a best practice environment.

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Back to TOC

# Food Safety: Temperature Data Analysis and the HACCP System

**John Robson and Roli Varma**

***Robson Laboratories, Australian Capital Territory***

In the food service industry, rethermalisation units provide a cost-effective method of preparing meals having both hot and cold food items. These two compartment units are able to maintain cold meals at cool room temperatures on one side and the other to reheat previously cooked food to an acceptable temperature in approximately 50 minutes. The application of these units has proved successful where large quantities of food are served.

The findings of a recent study (National Risk Validation Project, Ref: Food & Safety Hygiene, November 2002) assessed high-risk food businesses as being consistently linked with food-borne illness outbreaks. The three most frequently occurring hazards related to:

- Faulty temperature control
- Contamination via inadequate handling, such as poor hygiene, and
- Contaminated raw material.

As a consequence of these findings the Australian and New Zealand Food Regulation Ministerial Council (ANZFRMC) has made the implementation of food safety programs mandatory in the high-risk food sectors. Compliance with these program requirements is to commence two years after amendments to the Food Standards Code being gazetted.

These amendments were gazetted on 5 October 2006 as The Australia New Zealand Foods Standards Code - Amendment No. 88 - 2006, Standard 3.3.1, *Food Safety Programs for Food Service to Vulnerable Persons*. Implementation of the Hazard Analysis Critical Control Point (HACCP) system as a means of establishing food safety is regarded as being essential in situations where potentially hazardous food is served to vulnerable populations such as in hospitals and nursing homes. A Critical Control Point in a system or process is "a step at which a control can be applied and is essential to prevent or eliminate a food safety hazard or reduce it to an acceptable level".

One of the most important Critical Control Points related to food management is temperature. Therefore, verification of acceptable temperature control of food, delivered to vulnerable populations, is extremely important to facilitate compliance with the new food standards and food safety management systems. The effectiveness of HACCP food safety systems is assessed during the Auditing and Accreditation process.

Examples of typical questions asked during the auditing are:

- Have the Critical Control Points for each significant hazard been identified and transferred to the Hazard Audit Table?
- Have Critical Limits been established for each preventative measure?
- Have monitoring procedures been developed for each preventative measure?
- Is the frequency of monitoring sufficient to provide a high level of assurance that the process is under control?
- Are monitoring records kept and reviewed by the appropriate personnel?

The HACCP system was developed in 1996 and is preferred by food safety professionals around the world. It is widely viewed as critical to food safety, as it helps prevent food contamination, by identifying potentially unsafe links in the food processing chain. It is a system that manages the risk associated with food safety aspects of production.

This system is based on seven principles identified in the Codex Guidelines for the Application of Hazard System adopted by the 20th Session of the Joint Food and Agricultural Organisation and World Health Organization Codex Alimentarius Commission 1993. It involves:

- Examining and analysing every stage of a food-related operation to identify and assess hazards;

- Determining the 'Critical Control Points' at which action is required to control the identified hazards;

- Establishing the Critical Limits that must be met at, and procedures to monitor, each critical control point;

- Establishing corrective procedures when a deviation is identified by monitoring; and

- Documentation of the HACCP system and verification procedures to establish that processes are working correctly, i.e., an approach to process quality control and food safety use in the food industry.

During a HACCP audit, an auditor might ask questions on particular aspects of your control system and its application. Auditors will be primarily looking to see that systems conform to the Codex requirements (Codex Alimentarius Commission, 1996: Annex 1 to Appendix II - ALINORM 97/13, pp. 66-76), in the application of the principles and the following of developmental steps. Compliance documentation will be necessary.

During 2003 and 2004, work was undertaken within the Food Services Department of Canberra Hospital to assess the potential to simplify the presentation of the data generated by temperature loggers monitoring their kitchen rethermalisation units. A software program was developed to simplify and enhance the presentation of the collected data. Originally, 2-3 hours were required to process, present and analyse the data. Our program has reduced this time to less than 10 minutes. A prototype of the software program was installed in 2004.

Due to the application's success, the Food Services Department of Canberra Hospital has confirmed the long-term implementation of the software application to generate the required food safety Management Reports.

With the increasing importance of computer technology and its application in the field of Quality Control, our initial assessment of this innovative software highlighted significant time and associated cost savings. As well as this reduced labour cost, the software also provides the essential time-history temperature information, which fulfils the requirements of the recently introduced mandatory food safety programs.

A unique feature of the software is that it produces an exception report, indicating potential non-compliances (See Figure 1). An additional feature of the software is archiving of raw data on a nightly basis, so that where reports have indicated a concern, the data may be reviewed to reveal precise trends in temperature change. Further, during power failures, the battery within the data logger permits the continuation of temperature records and data storage for more than a day, ensuring valuable data are not lost.

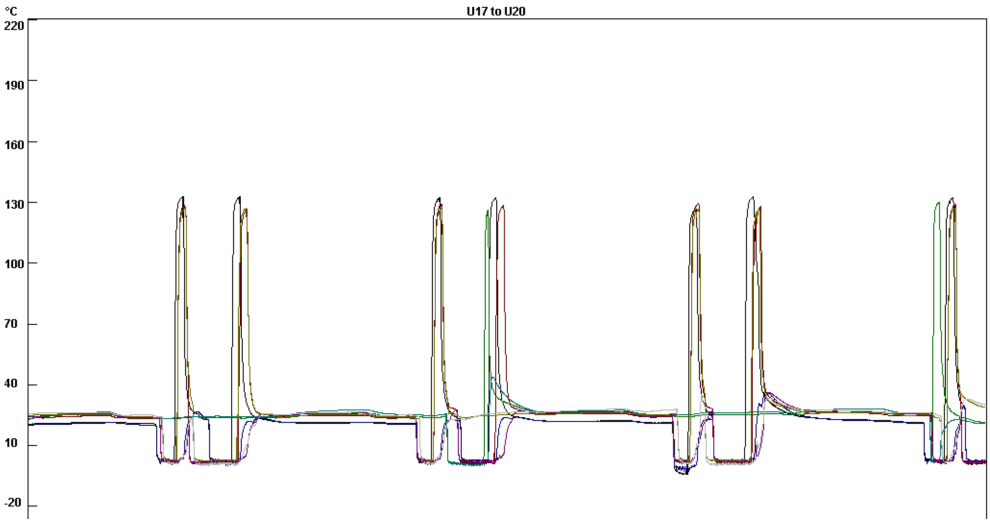
This particular monitoring system completed its first calibration compliance testing in January 2006.

***Figure 1: Top - Raw Data at 2-minute intervals, Middle – Previous presentation of data requiring individual graph analysis, Bottom – Software Application providing exception report.***

***Raw data - down loaded from temperature logger***

| Date | U17 Hot °C | U17 Cold °C | U18 Hot °C | U18 Cold °C | U19 Hot °C | U19 Cold °C | U20 Hot °C | U20 Cold °C |
|---|---|---|---|---|---|---|---|---|
| 21/11/2006 16:37:37 | 91.9 | 2.4 | 130.6 | 2.0 | 1.8 | -0.7 | 26.0 | 27.0 |
| 21/11/2006 16:39:37 | 102.8 | 2.7 | 130.8 | 1.5 | 1.5 | -0.6 | 26.0 | 27.0 |
| 21/11/2006 16:41:37 | 106.9 | 1.8 | 130.9 | 1.6 | 1.9 | -0.7 | 26.0 | 27.0 |
| 21/11/2006 16:43:37 | 113.9 | 2.1 | 131.2 | 1.5 | 1.8 | -0.3 | 26.0 | 27.0 |
| 21/11/2006 16:45:37 | 120.6 | 2.6 | 131.4 | 1.5 | 2.0 | -0.4 | 26.1 | 27.0 |
| 21/11/2006 16:47:37 | 127.5 | 2.1 | 131.4 | 1.2 | 2.1 | -0.3 | 26.1 | 27.0 |
| 21/11/2006 16:49:37 | 128.5 | 2.0 | 131.5 | 1.6 | 2.1 | -0.3 | 26.1 | 27.0 |
| 21/11/2006 16:51:37 | 129.1 | 2.5 | 131.4 | 1.6 | 2.4 | -0.1 | 26.0 | 27.0 |
| 21/11/2006 16:53:37 | 129.8 | 2.8 | 131.5 | 1.8 | 2.2 | 0.1 | 26.0 | 27.0 |
| 21/11/2006 16:55:37 | 130.5 | 2.1 | 131.5 | 1.8 | 1.9 | 0.3 | 26.0 | 27.1 |
| 21/11/2006 16:57:37 | 131.2 | 2.4 | 129.5 | 2.0 | 1.8 | 0.6 | 26.0 | 27.1 |
| 21/11/2006 16:59:37 | 131.8 | 2.5 | 125.2 | 1.6 | 1.7 | 0.6 | 26.0 | 27.0 |
| 21/11/2006 17:01:37 | 132.1 | 2.4 | 121.0 | 1.4 | 1.5 | 0.7 | 26.0 | 27.0 |
| 21/11/2006 17:03:37 | 132.3 | 2.5 | 101.6 | 1.7 | 1.6 | 0.6 | 26.0 | 27.0 |

***Graphed raw data - Individual graph analysis required***




***Section of management exception report - analysis (automated)***

**Management Report - Page 2 of 2**

**DINNER: Data Logging Records - 21/11/2006**

| Temp Range | HOT | 0 to 4 (-20ms) | 0 to 4(start) | 69 to 110 | 117 to 129 | 123 to 130 | 124 to 132 | 119 to 132(end) |
|---|---|---|---|---|---|---|---|---|
| Temp Range | COLD | 0 to 4(-20ms) | 0 to 4(start) | 0 to 4 | 0 to 4 | 0 to 4 | 0 to 4 | 0 to 4(end) |
| 52046 | Unit | 16:10 | 16:30 | 16:40 | 16:50 | 17:00 | 17:10 | 17:20 |
| DEL - 4 | Hot17 | 2.3 | 2.3 | 103.0 | 128.5 | 131.8 | 133.2 | 133.5 |
| | Cold17 | 2.1 | 2.5 | 2.6 | 3.2 | 3.3 | 3.8 | 3.2 |
| | | 15:45 | 16:05 | 16:15 | 16:25 | 16:35 | 16:45 | 16:55 |
| 9G - 5 | Hot18 | 0.8 | 1.7 | 114.4 | 127.6 | 130.2 | 131.4 | 131.5 |
| | Cold18 | 1.4 | 1.5 | 1.5 | 2.2 | 2.8 | 3.7 | 3.1 |
| | | 16:45 | 17:05 | 17:15 | 17:25 | 17:35 | 17:45 | 17:55 |
| 6E - 1 | Hot19 | 2.0 | 1.7 | 70.5 | 120.7 | 125.7 | 127.4 | 127.9 |
| | Cold19 | 2.5 | 1.6 | 2.7 | 3.1 | 3.8 | 3.6 | 4.2 |
| | | 16:40 | 17:00 | 17:10 | 17:20 | 17:30 | 17:40 | 17:50m |
| 13B-2 | Hot20 | 26.2 | 26.1 | 26.1 | 26.02 | 26.0. | 26.0 | 226.0 |
| | Cold20 | 27.1 | 27.1 | 27.1 | 27.1 | 27.1 | 27.0 | 27.0 |

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Back to TOC

# Public Health in Action: Practising in the Real World

## Jan K. Carney

*Jones and Bartlett Publishers, Massachusetts, 2006, 277 pp, ISBN 0-7637-3447-0 (paperback)*

If you are looking for a textbook on basic public health principles and methods, then this book is not for you. However, if you are looking for a book that provides a wealth of considered and practical advice on how to practice public health professionally in the real world, then this is a book for you. The author has had a career in both high-level academic and public service positions, particularly as the health commissioner for the state of Vermont, USA, and this combination of experience comes through in the content, style and arrangement of the text.

The text is organised in an unusual way in that it consists of 58 very short chapters (each around 3 to 4 pages long), with each chapter describing a specific public health issue or activity that the author has been associated with and the lessons learnt from that experience. Therefore, the text is basically a collection of short stories that are arranged into four parts: Fundamentals, which includes basic concepts for public health practice; Issues, which includes examples of public health issues, how they were addressed, and the tensions at play; Strategies, which discusses various approaches that have been successful in managing public health issues; and Challenges, which discusses future challenges for public health and key approaches that may be used to address these. Environmental health practitioners will be familiar with many of the issues discussed as they relate to public health management at a local level. The issues discussed include: public health planning, disease outbreak investigation and management, management of lead and other environmental health issues, tobacco control, effective interaction with the public and colleagues, and the day-to-day pressures of being a public health practitioner.

Initially, I was a little sceptical about the approach taken by the author, but the more I read, the more I was able to relate to the stories. I was particularly impressed by the way in which the experience of the author was communicated in an easy to read but insightful way. In some ways, it is like having a long conversation with a veteran public health manager in which a lifetime of experience is shared with clarity and a purpose to instruct. For example, one memorable quote is "the way in which you as a public health official communicate when there is not a crisis, and your ongoing relationships with laboratories, hospitals, doctors, nurses, and within your own department will determine how well and quickly you can respond when a crisis *does* occur" (p. 9). The chapters combine to provide a vivid account of the tight-rope that public health practitioners constantly walk "to balance short-term crises with progress on longer-term issues, to find new and better ways of solving problems, and to communicate more clearly and effectively" (p. ix).

If there is one criticism of the book, it is that there are no figures, tables or other inclusions to break-up the text and make it easier to digest. However, the short well-written chapters ensure that the topics are covered quickly and concisely and the lessons are revealed without much delay.

Overall, this text fills a niche in that it provides experience-based advice on how to be a more effective practitioner in the public health arena. It is therefore highly recommended for advanced public health students as an introduction to the real world, and will be of great value to public health professionals who are looking for a text they can relate to and which provides practical advice that is not available in other texts.

Back to TOC

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# Preventing Disease through Healthy Environments

**A. Pruss-Ustun and C. Corvalan**

*World Health Organization, Geneva, 2006, 104 pp, ISBN 92 4 159382 2*

On June 16, 2006, the World Health Organization released the report, *Preventing Disease through Healthy Environments: Towards an Estimate of the Environmental Burden of Disease*. This report is the most comprehensive and systematic study ever undertaken on the contribution of environmental hazards to a wide range of diseases and injuries. By focusing on the environmental causes of disease, and how various diseases are influenced by environmental factors, the report provides a new understanding of the interactions between environment and health. In addition, the report identifies how much death, illness and disability could be realistically avoided every year as a result of better environmental management.

The report identifies that as much as 24% of global disease is caused by environmental exposures that can be avoided. It estimates that more than 13 million deaths annually are due to preventable environmental causes, with nearly one third of death and disease in the least developed regions due to environmental causes. Well-targeted interventions are identified as being able to prevent much of this environmental risk, with over 40% of deaths from malaria and an estimated 94% of deaths from diarrhoeal diseases, two of the world's biggest childhood killers, able to be prevented through better environmental management.

The four main diseases influenced by poor environments are diarrhoea, lower respiratory infections, various forms of unintentional injuries, and malaria. Measures which could be taken now to reduce this environmental disease burden include the promotion of safe household water storage and better hygiene measures; the use of cleaner and safer fuels; increased safety of the built environment, including a more judicious use and management of toxic substances in the home and workplace; and better water resource management.

Diseases with the largest total annual health burden from environmental factors, in terms of death, illness and disability or Disability Adjusted Life Years (DALYs) are identified as:

- Diarrhoea (58 million DALYS per year; 94% of the diarrhoeal burden of disease), largely from unsafe water, sanitation and hygiene.

- Lower respiratory infections (37 million DALYs per year; 41% of all cases globally), largely from indoor and outdoor air pollution.

- Unintentional injuries other than road traffic injuries (21 million DALYs per year; 44% of all cases globally), with this classification including a wide range of industrial and workplace accidents.

- Malaria (19 million DALYs per year; 42% of all cases globally), largely as a result of poor water resource, housing and land use management which fails to curb vector populations effectively.

- Road traffic injuries (15 million DALYS per year; 40% of all cases globally), largely as a result of poor urban design or poor environmental design of transport systems.

- Chronic Obstructive Pulmonary disease (COPD) (12 million DALYs per year; 42% of all cases globally), largely as a result of exposures to workplace dusts and fumes and other forms of indoor and outdoor air pollution.

- Perinatal conditions (11 million DALYS per year; 11% of all cases globally).

Most of the same environmentally-triggered diseases also rank as the biggest killers outright – although they rank somewhat differently in order of lethality. Diseases with the largest absolute number of deaths annually from modifiable environmental factors include:

- 2.6 million deaths annually from cardiovascular diseases
- 1.7 million deaths annually from diarrhoeal diseases
- 1.5 million deaths annually from lower respiratory infections
- 1.4 million deaths annually from cancers
- 1.3 million deaths annually from chronic obstructive pulmonary disease
- 470,000 deaths annually from road traffic crashes
- 400,000 deaths annually from unintentional injuries.

**Back to TOC**

Overall, this report confirms the significant contribution that the environment makes to the global burden of disease and illness. It highlights how this burden of disease is substantially borne by children and the poorest societies in the world, but it emphasises that the environmental risk factors can be modified by well established and cost-effective interventions. The underlying message of the report is that there are millions of unnecessary deaths each year that result from environmental conditions that can be easily and effectively managed by existing and relatively cheap preventive measures. However, to be effective, these measures require global partnerships and strengthened collaboration between the health and non-health sectors.

The report can be accessed from: http://www.who.int/quantifying_ehimpacts/publications/preventingdisease/en/index.html

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# The Business Case for Early Action

## Australian Business Roundtable on Climate Change

*Australian Business Roundtable on Climate Change, 2006, 25 pp, ISBN 0-85802-136-6*

In April 2006, the Australian Business Roundtable on Climate Change released a seminal report titled *The Business Case for Early Action*. The Australian Business Roundtable on Climate Change (Roundtable) consists of six of the largest businesses in Australia (BP Australia, Insurance Australia Group, Origin Energy, Swiss Re, Visy Industries and Westpac) in collaboration with the Australian Conservation Foundation. The Roundtable was formed to undertake new research to advance the understanding of business risks and opportunities associated with climate change.

*The Business Case for Early Action* is a summary report based on the findings of two independent research projects that were funded by the Roundtable. For the first project, the Roundtable commissioned CSIRO to quantify climate change impacts on Australia. This research, outlined in CSIRO's report *Climate Change Impacts on Australia and the Benefits of Early Action to Reduce Global Greenhouse Gas Emissions*, confirmed that the economic impacts are potentially significant and widespread, affecting a wide range of industries. Of particular concern are impacts on two of Australia's largest export earners - agriculture and tourism. These impacts are predicted to have flow-on effects for the whole economy. Examples of these impacts are:

- The $32 billion tourism industry is highly climate dependent. For example, the Great Barrier Reef supports a $1.5 billion industry but with a 2-3°C increase in temperature, 97% of the Reef could be bleached;
- The $17 billion of exports from the livestock industry face risks from more heat stress, more pests and disease; national livestock carrying capacity is expected to fall by 40% if temperatures increase by 2°C; and
- A 2°C increase in temperature would reduce water flows in the Murray-Darling Basin and to Melbourne, by about 15%. Based on a 20% reduction in Australian irrigation allocations, GDP is projected to fall by around $750 million in 2009/10.

The Report also identified that there will also be constraints on other water-dependent industries such as power generation and process industries. How Australian industries and economic systems cope with these impacts depends not only on the extent and rate of climate change, but also on their capacity to adapt. The CSIRO report concluded that reducing global greenhouse gas emissions will reduce the rate and magnitude of climate change, thereby allowing industries more time to adapt. Therefore, acting early to cut emissions not only reduces damage, but buys time. CSIRO further concluded that reducing emissions in developed countries by 60% or more by 2050 as part of an international response would prevent some of the worst-case scenarios of climate change in Australia.

The Roundtable then commissioned the Allen Consulting Group to provide economic modelling into what it will cost Australia to substantially reduce emissions in line with the CSIRO findings. Based on international calls to limit temperature increases below dangerous levels, and specific targets adopted by a number of countries, it was agreed to model a 60% reduction on year 2000 emissions by 2050. This compares with the current Kyoto Protocol average of a 5% decrease and a target for Australia of an 8% increase (based on 1990 levels) by 2012.

This research is ground-breaking as it is the first time that the economic viability of achieving such a goal in Australia has been tested and published. Two trajectories were specified as alternate pathways, as part of an international response, to meet the same total emission reductions over the time period. One was an early action scenario with a carbon signal introduced in 2013, and the other was a delayed action scenario which assumed that the carbon signal would be delayed until 2022. The base case was specified as no global carbon price and no global action post-2012, which equates to no further action beyond the programs already existing in 2005. Under the model, this scenario would result in greenhouse gas emission levels in Australia in 2050 being 80% higher than current levels. However, as this is a reference case only, it does not factor in the economic impacts of climate change on Australian industry and is an unlikely international or domestic response. Overall, the research found that it is possible to deliver significant reductions at an affordable cost and endorses the case for early action.

The following summarises the recommendations of the Report:

- The Roundtable supports government calls for a collaborative approach to climate change as demonstrated by the Asia-Pacific Partnership on Clean Development and Climate and recent COAG initiatives. However, the Roundtable believes that more needs to be done.

- The Roundtable developed an integrated package of recommendations which it believes will create the necessary investment conditions to enable Australia to reduce greenhouse gas emissions while maintaining strong economic growth. These recommendations complement current government efforts to develop and deploy breakthrough technologies that will deliver the necessary deep cuts to greenhouse gas emissions in the long term.

- Australian business is looking to the Australian Government to implement a policy framework that will accommodate the fine balance between uncertainty about future international agreements and advancing scientific knowledge of climate change. Achieving such an outcome would also enhance our ability to influence negotiations for international action. Australia has a major role to play in these negotiations, driven by our vulnerability to climate impacts and our economic reliance on greenhouse-intensive fossil fuels.

The Report states that:

> *The research and the recommendations outlined in this report are designed to provide a timely contribution to framing Australia's policy response. The Roundtable strongly believes that the recommendations contained in the report are in the national interest. Long-term competitiveness will be enhanced by leading the development of market-based solutions to the climate change challenge. [They conclude with] By acting early, we will all benefit (p. 21).*

The various Roundtable reports can be accessed at: www.businessroundtable.com.au

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Back to TOC

Environmental Health

The Journal of the Australian Institute of Environmental Health

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Back to TOC

**Environmental Health**

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# Guidelines for Contributors

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Back to TOC