The Journal of the Australian Institute of Environmental Health





...linking the science and practice of Environmental Health





The Journal of the Australian Institute 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.

Environmental Health is indexed in Ulrich's Periodicals Directory, the Australasian Medical Index, and APAIS

Aims

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

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The Journal of the Australian Institute of Environmental Health

Call for Papers

The Journal is seeking papers for publication.

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- **O** Environmental Health Vol. 4 No. 3 2004

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

GUEST EDITORIALS

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

RESEARCH AND THEORY

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

PRACTICE, POLICY AND LAW

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

REPORTS AND REVIEWS

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

Correspondence

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EDITORIAL

Readers will have noticed some changes with this issue of *Environmental Health*. The Journal will continue to be produced in the same format as before but distribution will be as an on-line publication.

The Journal will be accessible via the secured members and subscribers only section of the AIEH website thus the online Journal will afford members the benefits of a peer reviewed publication without incurring high production costs. It is intended that index and search functions will allow for instantaneous key word searches of current and archived materials. Articles on demand features will allow for the reproduction of individual articles. Multiple subscription access will be available also thus reducing costs in servicing new subscribers, and content syndication of Environmental Health articles will be available to other Environmental Health organisations worldwide. All past copies of the Journal will be made available online to members and subscribers with full index and search capabilities.

Heather Gardner, the inaugural editor of Environmental Health, has completed her contract and, in recognition of her work in outstanding establishing Environmental Health, Heather was made an Honorary Fellow of the Australian Institute of Environmental Health at the recent National Conference held in Melbourne. With the continued development of the Journal a new editorial team has been established. The new Editor of Environmental Health is Jim Smith and Heather Gardner has accepted the position of Associate Editor and will continue to assist the development of the Journal. Jaclyn Huntley is undertaking the role of Editorial Assistant. A major task of the team will be the marketing of the Journal.

As has been mentioned, the 31st National Conference of the Institute was held in Melbourne in October. The conference attracted average daily attendances of 200 delegates. The two keynote speakers were the Rev Tim Costello, CEO of World Vision and Mr Rob Gell (former Channel 9 weatherman) who gave thought provoking insights into sustaining communities and sustaining the natural environment.

The 5th National Indigenous Environmental Health Conference was held in Terrigal on 3rd and 4th November with attendance of many delegates from all over Australia. Addresses were made by Michael Jackson, Chair of enHealth Council on the achievements to date in environmental health. A number of speakers addressed many aspects of the diverse area of indigenous environmental health organised around the conference themes of environmental health workforce development; housing issues and planning; local, regional and national initiatives; community capacity building/partnerships; community food supply and nutrition; and environmental health services in communities.

On the subject of conferences the Queensland Division of the AIEH will conduct its Annual State Conference from 21-24th November 2004 on the Gold Coast. Refer to the AIEH website for details. The 36th Conference of the Asia-Pacific Academic Consortium for Public Health will be held 30th November to 3rd December 2004 in Brisbane. Further details can be obtained from the website www.apacph.org/conf2004.asp

The papers in this issue of the Journal reflect the increasing environmental health interest in air pollution. Hinwood et al. have researched the relationship between changes in daily air pollution and hospitalisations in Perth, also in Western Australia, Oosthuizen and Cross have studied respiratory hazards and the development of best practice protocols for occupational monitoring in open cut mining, and Pisaniello et al. have looked at the implications for public health of ambient air concentrations of lung carcinogens and fine particles in North West suburban Adelaide. Continuing with public health practice, Hogan et al. investigated a large outbreak of gastroenteritis in a residential educational institution. Oosthuizen provides an update in a report on a re-evaluation of the data on selected heavy metals in fish and sediment from the Swan River using the FSANZ standard. Of importance in this issue is the excellent book review section with reviews of books which also reflect current concerns with a major emphasis on water, enHealth's Rainwater Tank Guidelines, White's Controversies in Environmental Sociology, Steve and Elizabeth Hrudey's Safe Drinking Water, Rose's An Introduction to the Physics of Soil, Water and Watersheds, and also Australia's Health from the Australian Institute of Health and Welfare.

We look forward to the continued, indeed increased, success of the Journal in its new format and with its new editorial team. On a personal note from Heather, I would like to thank all the contributors and reviewers who have worked so hard and with such good will over the years, the Advisory Board and Editorial Committee, and the professional expertise and cheerful support of the printer and of the graphic designer. Thank you too to the Australian Institute of Environmental Health, who made it all possible, and for the award of the Honorary Fellowship, which has made me very proud. I look forward very much to working again with my colleague Jim Smith.

> Jim Smith LFAIEH Editor

Heather Gardner FAIEH (Hon) Co-editor



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Changes in Daily Air Pollution and Mortality in Perth: A Case Crossover Study

A.L. Hinwood¹, N. de Klerk², C. Rodriguez¹, T. Runnion³, P. Jacoby², L. Landau⁴, F. Murray⁵, M. Feldwick⁶ and J. Spickett⁷

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The monitoring of the common air pollutants in Perth has shown that concentrations exceed national standards for particles and photochemical oxidants (measured as ozone $[O_3]$) on occasions in winter and summer respectively. By contrast, concentrations of sulphur dioxide (SO₂), nitrogen dioxide (NO₂) and carbon monoxide (CO) are all below national standards. In this study, a time stratified case crossover design was used to investigate the relationship between changes in daily air pollutant concentrations and mortality in Perth. The data were analysed using conditional logistic regression to produce odds ratios. Mortality data were categorised into cardiovascular disease, respiratory disease, and 'other mortality' and were obtained for the period 1992 through 1998. Network air quality data and meteorological data were obtained for the same period.

The results showed significant relationships between cardiovascular mortality and NO_2 and O_3 concentrations. Increased odds ratios were also observed for O_3 concentrations and respiratory mortality, however, only for the 8-hour O_3 concentration was the increase statistically significant. A significant odds ratio was also observed for changes in CO concentrations and 'other mortality'. The data support the findings of other Australian and international studies and will assist in the evaluation of strategies designed to reduce air pollution in Perth.

Key words: Air Pollution; Mortality; Case Crossover; Cardiovascular Diseases; Respiratory Diseases

There are now numerous studies reporting significant relationships between air pollution and mortality (Dockery, Schwartz & Spengler 1992; Dockery et al. 1993; Health Effects Institute 2000; Katsouyanni et al. 1997; Laden et al. 2000; Pope, Hill & Villegas 1999; Samet et al. 2000; Schwartz 1993). The research demonstrates clear associations between mortality for a variety of causes and levels of air pollution (Mar et al. 2000; Ostro et al. 1996; Pope et al. 2002; Ponka, Savela & Virtanen 1998; Sunyer et al. 1996; Wong et al. 2001). In Australia, several studies using time series analysis have been conducted to investigate the relationship between daily ambient air quality and health outcomes including mortality. Studies conducted in Sydney and Melbourne have shown that increases in air pollution levels are associated with increased risks of daily mortality (Environmental Protection Authority 2000; Morgan et al. 1998). A positive association was observed between ozone and particulate matter (PM) and daily mortality for a study in Brisbane where the results were significant for those older than 65 years (Simpson et al. 1997).

Air Quality in Perth is monitored via a network of 13 ambient air quality stations with different parameters monitored at each station. Perth air quality is considered unsatisfactory for relatively short periods of time each year. The levels of photochemical smog (ozone) in Perth during summer regularly exceed national standards, and, during the winter months, particles are relatively high (Department of Environmental Protection [DEP] 2000). The episodes of unsatisfactory air quality are influenced by weather conditions that prevent air pollutants from dispersing rapidly. By contrast, concentrations of SO₂, NO₂ and CO are all below national and international standards in Perth with elevated levels being observed in specific areas impacted by point source emissions (DEP 2001).

Figure 1: Map of air quality monitoring sites in Perth as at 1/1/2000



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Given the results of national and international studies relating air pollution and health outcomes, it was necessary to establish whether such associations are observed in Perth where there is a different pollutant mix and meteorology and where measures are required to reduce air pollutants to acceptable levels. This study used the case crossover design to investigate the health effects of air pollution. The case crossover design measures the effects of transient risk factors for acute disease events. This design samples only cases with exposures for each subject during a designated period before the disease or hazard event and compares the distribution of exposures during a reference period.

The reference exposures should be representative of the expected distribution of exposures for follow-up times that do not result in a case (Levy et al. 2001). The study design may have advantages in controlling for time trends and seasonality compared to traditional time series analysis and controls for all measured and unmeasured variables which do not vary with time (Bateson & Schwartz 2001). It does not require modelling of all variables and all season and trend effects are removed. Factors such as age and sex can be estimated more easily as the event is the unit of analysis. The case crossover conditional logistic regression analysis is considered to be more robust and less prone to bias and confounding than the more standard time series regression analysis, however, bias may be introduced when the exposure in the reference periods is not equally representative of exposure in the hazard periods (Bateson & Schwartz 2001).

This study investigated the relationship between changes in daily ambient air pollutant concentrations and daily mortality, cardiovascular mortality, and respiratory mortality in the Perth metropolitan area between 1992 and 1998 using a time stratified design, with three or four control cases in the same month as the hazard.

Method

Study area and population

The study area encompassed the metropolitan Perth region according to the Australian Bureau of Statistics (ABS) Statistical Division.

There are several pockets of high industrial activity, the population is spread out and there is a high dependency on private motor transport compared with public transport use. The city is generally free from trans-boundary pollution events being subject to consistent wind from the Indian Ocean where the nearest land is thousands of kilometres away and offshore winds are from regions of very low population density.

Mortality data

Mortality data were made available by the Registrar General of Western Australia from the period 1 January 1992 to 31 December 1997. There are approximately 12,000 deaths recorded annually in Perth. Causes of death codes are made available on an annual basis by date of registration. Deaths registered in 1998 were checked for the date of death in case any 1997 deaths had been missed. Three groups of causes of death using the International Classification of Diseases version 9 (ICD-9) were analysed separately: cardiovascular (ICD9 codes 390 to 459), respiratory (ICD9 codes 460 to 529), and 'other causes' excluding accidents, poisoning, violence, cardiovascular and respiratory causes.

Air quality data

Daily ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), sulphur dioxide (SO_2) and particle concentrations measured by nephelometry (Bsp) have been measured since 1990. PM_{2.5} has been monitored since 1994 using a tapered element oscillating microbalance (TEOM). PM₁₀ was collected every 6 days by high volume sampler (HiVol) and was excluded for analysis.

The most complete data set with the highest number of days with co-located instruments were from the Caversham, Swanbourne and Queens Building sites and these were used to generate the daily air quality data for the pollutants investigated, with the exception of $PM_{2.5}$ which is discussed below. SO_2 was not included in the analysis due to the negligible SO_2 concentrations in the Perth metropolitan area, most being below the limit of detection.

Data aggregation

Data from Caversham, Queens Building and Swanbourne monitoring stations were aggregated in this study. The aggregate data were then compared with all available data across the network.

The averages for each pollutant and each method of generating a daily estimate were comparable with no statistically significant differences, indicating the use of the three sites was acceptable as a surrogate for data across the entire network.

The averaging times selected were based on the Australian National Environmental Protection Measure Ambient Air standards and were used for each pollutant in subsequent analyses. Daily temperature, wind speed and direction, dew point temperature and relative humidity were obtained by averaging the data from the monitoring stations. These data were compared with the Bureau of Meteorology averages to validate the use of data collected from individual fixed air monitoring stations.

Dispersion modelling was utilised to develop daily average concentration of $PM_{2.5}$ due to the limited number of particulate monitoring sites and the large variations in population density and subsequent particle concentrations. Measurements made at a mix of locations in the Perth region have shown that in winter, when high particulate concentrations are most common, there is a strong relationship between the density of population about each site and the particulate concentrations measured.

Measurement of particles and numerical modelling studies have confirmed that daily averages vary considerably across the region, being largest near centres of highest population density (Clench-Aas et al. 1999; Micallef & Colls 1999; Vinitketkumnuen et al. 2002).

Despite the inherent limitations in this approach (related to the limited knowledge of meteorological conditions and of the actual particulate emissions), it was considered that the modelled concentration distribution, scaled to match available measurements, would form a better basis for exposure estimation. The modelling work was conducted using a Gaussian plume dispersion model, with the particulate emission rate for each location presumed proportional to population density. Each day of a winter season was modelled separately, providing for each day a grid map of estimated daily average concentrations. From this map, modelled concentrations for each particulate measurement site were determined.

For a totally accurate model, these concentrations would have matched measurements, giving a ratio of one at each site. In practice, the ratios differed from one, and varied across the modelled region. However, it was possible to interpolate this set of ratios to develop a grid map of correction factors for the region. The best estimate for each model grid point was then taken as the product of the modelled concentration and the interpolated ratio of measured to modelled concentration. These estimates matched all measurements, and also varied between measurement sites in a manner consistent with model calculations. To enhance further the accuracy of exposure estimates, the relationship between particulate concentrations and optical backscatter coefficient (B_{sp}) was studied.

For the winter haze events, it was found that there was a linear relationship, which for B_{sp} over 2 was:

 $[PM_{2.5}] = 17.86 B_{sp} + 3.69 \mu g/m^3$

With this relationship, it was possible to use B_{sp} measurements to enhance the data set used in interpolations. Figure 2 shows the interpolated and modelled PM2.5 concentration contours for Perth.







Average PM_{2.5} concentrations µgm⁻³) for the Perth region, modelled for the period June-July 1997, and adjusted to match measured and estimated PM_{2,5} values at all monitoring sites.

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Statistical analysis

This study used a time stratified case crossover design. Individual deaths were considered and the cases were matched to exposure levels during referent days falling on the same day of the week in the same month as the day of mortality. This provided three or four control days depending on the number of days and cases in each month. Comparisons between cases and controls were then made using conditional logistic regression to produce odds ratios (Bateson & Schwartz 1999). The analysis was conducted using Proc PHREG in SAS V8 (SAS Institute Inc., Cary, NC). Before examining the effect of pollutants, temperature and humidity were examined. Variables indicating public holidays and day of the week were included. However, only the most significant effects were included in the pollutant models. Each pollutant was entered into its own predictive model for seven different lag periods, (0,1,2,3,0-1, 0-2, 0-3) days, and four different averaging periods (1hr, 4hr, 8hr, 24hr) where data were available. Each category of mortality data was analysed in relation to NO_2 , O_3 , particulate ($PM_{2.5}$ modelled and B_{sp}) and CO concentrations. Days for which no measures of a particular pollutant had been made were ignored in the analysis involving the specific pollutant. When days of missing data occurred randomly, as was the case in this data set, ignoring the data was not likely to introduce selection bias.

Results

From 1992 to 1998 the average number of daily deaths was 26.8 with an average of between 2.1 and 2.4 deaths attributable to respiratory disease (Table 1). By comparison 11.6 deaths were attributed to cardiovascular disease. Most of these deaths occurred in the age group greater than 65 years. Daily pollutant concentrations and meteorological parameter measures for the period are presented in Table 2. The association

Table 1: Daily number of deaths between 1992 to 1998

		N	lovember-A	pril			May-October			All year		
	Mean	SD	10 th	90 th	Mean	SD	10 th	90 th	Mean	SD	10 th	90 th
			centile	centile			centile	centile			centile	centile
All deaths	24.2	5.4	18	31	29.2	6.2	21	37	26.8	6.3	19	35
All deaths > 65 years	18.8	4.7	13	25	23.4	5.6	17	31	21.1	5.7	14	29
CVD deaths	10.2	3.4	6	15	12.9	3.8	8	18	11.6	3.9	7	17
CVD deaths > 65	8.8	3.1	5	13	11.4	3.6	7	16	10.1	3.6	6	15
Respiratory deaths	1.8	1.4	0	4	2.9	2.0	I	5	2.4	1.8	0	5
Respiratory deaths > 65	1.5	1.3	0	3	2.6	1.9	0	5	2.1	1.7	0	4

Table 2: Air pollution concentrations and meteorological parameters

	November-April				May-October			All year				
	Mean	SD	10 th	90 th	Mean	SD	10 th	, 90 th	Mean	SD	I Oth	90 th
			centile	centile			centile	centile			centile	centile
NO ₂ - 24h ave (ppb)	9.6	4.8	4.3	15.7	11.1	5.1	4.8	18.0	10.3	5.0	4.4	17.1
NO ₂ - Ih max (ppb)	24.7	11.1	12.4	39.2	24.9	8.9	14.4	35.7	24.8	10.1	13.3	37.5
03 - 8h max (ppb)	27.7	7.4	19.5	38.3	24.2	4.9	18.4	30.5	25.9	6.5	18.9	34.1
03 - 4h max (ppb)	31.1	9.4	20.8	45.0	26.6	5.0	21.1	32.3	28.8	7.8	21	39.5
O ₃ - Ih max (ppb)	35.0	12.4	22.1	53.7	28.3	5.6	22.4	34.0	31.6	10.2	22.2	46.I
B _{sp} - 24h ave bscat/10 ⁴	0.20	0.13	0.09	0.33	0.30	0.23	0.12	0.57	0.25	0.20	0.10	0.47
B _{sp} - 1h max (bscat/10 ⁴)	0.74	0.87	0.25	1.39	1.61	1.70	0.39	3.73	1.2	1.4	0.3	2.6
CO - 8h max (ppm)	2.2	1.3	0.8	4.2	2.4	1.2	1.1	4.2	2.3	1.3	0.9	4.2
*PM ₁₀ - 24h ave (ppb)	20.6	7.7	12.6	29.7	18.8	7.8	10.7	29.0	19.6	7.8	11.3	29.5
**PM _{2.5} - 24h ave (ppb)	8.6	3.8	4.9	13.1	9.7	4.7	5.1	16.2	9.2	4.3	5.0	14.5
Temperature - 24h ave	21.8	3.4	17.8	26.7	15.1	2.5	12.0	18.2	18.4	4.5	13.0	24.7
Humidity - 24h ave	56.2	15.3	35	75.5	71.4	12.2	53.5	85.0	64.0	15.8	40.5	82.5

* Available only weekly

** Modelled PM2.5 data

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		NO ₂ 24-hr	03 8-hr	B _{sp}	CO 24-hı
03	All year	-0.06			
	Warm	0.35			
	Cool	-0.11			
B _{sp}	All year	0.39	0.01		
	Warm	0.26	0.28		
	Cool	0.42	0.02		
C0	All year	0.57	0.00	0.35	
	Warm	0.57	0.16	0.24	
	Cool	0.55	0.00	0.37	
Temp	All year	-0.12	0.20	-0.27	
	Warm	0.22	0.51	0.04	0.04
	Cool	-0.19	0.04	-0.31	-0.11
Humidity	All year	0.18	-0.12	0.25	
	Warm	0.01	-0.31	0.18	0.26
	Cool	0.15	0.00	0.11	0.33

temperature, as well as CO concentrations and NO_2 and particulates were found.

The resultant odds ratio and 95% confidence intervals (CI) were plotted for particulate (B_{sp}), NO₂ O₃ and CO concentrations and each mortality category (Figures 3 to 6). The odds ratios and 95% CI for each mortality category and lag for 24hr averaging periods for particulates (b_{sp}), NO₂ and modeled PM_{2.5} is shown in Table 4. The results of analysis showed no significant associations between changes in daily particle concentrations as measured by nephelometry (B_{sp}) (1hr and 24hr) and respiratory mortality, cardiovascular diseases (CVD) mortality or 'other' mortality (Figure 3, Table 4). Small but significantly elevated

Table 4: Odds ratios and 95% confidence intervals (CI) by mortality category, per lag, per unit concentration of 24 hr average of Bsp, NO2 and PM2.5 (modeled)

Mortality	lag	B _{sp}	(Nephelometry)		NO ₂ (ppb)	PM	2.5 ("g/m ³)
Category	-	OR	95% CI	OR	95% CI	OR	95% CI
Respiratory							
. ,	0	0.9690	(0.7285 - 1.0367)	0.9922	(0.9836 - 1.0009)	0.9961	(0.9915 - 1.0023)
	I	0.8984	(0.7529 - 1.0719)	0.9953	(0.9867 - 1.0040)	0.9961	(0.9916 - 1.0015)
	2	0.9785	(0.8237 - 1.1624)	0.9971	(0.9887 - 1.0058)	1.0016	(0.9973 - 1.0022)
	3	0.9816	(0.8240 - 1.1694)	1.0019	(0.9932 - 1.0106)	1.0005	(0.9960 - 1.0023)
	0-1	0.8477	(0.6918 - 1.0386)	0.9916	(0.9817 - 1.0016)	0.9944	(0.9890 - 1.0023)
	0-2	0.8574	(0.6833 - 1.0759)	0.9912	(0.9803 -1.0024)	0.9963	(0.9902 - 1.0027)
	0-3	0.8587	(0.6689 - 1.1024)	0.9933	(0.9812 - 1.0055)	0.9969	(0.9901 - 1.0032)
Cardiovascul	ar Diseases	(CVD)					
	0	1.0536	(0.9740 - 1.0069)	1.0031	(0.9992 - 1.0069)	1.0003	(0.9982 - 1.0023)
	I	1.0089	(0.9325 - 1.0072)	1.0034	(0.9995 - 1.0072)	0.9995	(0.9975 - 1.0015)
	2	1.0017	(0.9264 - 1.0075)	1.0037	(0.9998 - 1.0075)	1.0002	(0.9982 - 1.0022)
	3	0.9918	(0.9165 - 1.0053)	1.0014	(0.9976 - 1.0053)	1.0003	(0.9983 - 1.0023)
	0-1	1.0414	(0.9509 - 1.0088)	1.0043	(0.9991 - 1.0088)	0.9998	(0.9974 - 1.0023)
	0-2	1.0338	(0.9340 - 1.0106)	1.0057	(1.0007 - 1.0106)	0.9999	(0.9972 - 1.0027)
	0-3	1.0240	(0.9159 - 1.0114)	1.0059	(1.0005 - 1.0114)	1.0002	(0.9971 - 1.0032)
Other							
	0	1.0044	(0.9304 - 1.0845)	1.0012	(0.9975 - 1.0049)	1.0005	(0.9985 - 1.0024)
	I	0.9444	(0.8736 - 1.0211)	1.0006	(0.9969 - 1.0042)	0.9983	(0.9963 - 1.0003)
	2	0.9614	(0.8899 - 1.0386)	1.0025	(0.9987 - 1.0062)	0.9989	(0.9968 - 1.0009)
	3	0.9671	(0.8958 - 1.0442)	1.0004	(0.9968 - 1.0041)	0.9992	(0.9972 - 1.0012)
	0-1	0.9668	(0.8842 - 1.0572)	1.0011	(0.9969 - 1.0054)	0.9991	(0.9967 - 1.0015)
	0-2	0.9494	(0.8587 - 1.0497)	1.0022	(0.9975 - 1.0069)	0.9985	(0.9958 - 1.0013)
	0-3	0.9396	(0.8419 - 1.04869)	1.0020	(0.9969 - 1.0072)	0.9982	(0.9952 - 1.0012)

Note: Each pollutant was entered into its own predictive model for seven different lag periods, (0,1,2,3,0-1, 0-2, 0-3) days.

between air pollutant concentrations, seasonality and meteorological parameters is shown in Table 3. As expected, significant correlations between O_3 and particulates and

odds ratios for CVD mortality in relation to changes in NO_2 concentrations (1hr and 24hr) were observed (Figure 4, Table 4). No significant relationships were observed for

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Respiratory Mortality Odds Ratio per Unit Increase in Neph(1 hr) 1.1 Odds Ratio (95% CI) 1.05 1 1 t T 0.95 0.9 0 L 2 3 +| +2 +3 Lag CVD Mortality Odds Ratio per Unit Increase in Neph(1 hr) 1.1 Odds Ratio (95% CI) .05 I 0.95 0.9 0 2 3 +| +2 +3 Т Lag Other Mortality Odds Ratio per Unit Increase in Neph(I hr) 1.1

Figure 3: Odds ratios plotted for each mortality category lag per unit increase of particle concentrations as measured by nephelometry (1 hr)



Figure 4: Odds ratios plotted for each mortality category, lag and per unit increase in nitrogen dioxide concentrations ppb (1 hr)



Respiratory Mortality Odds Ratio per Unit Increase in NO₂ (1 hr)

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Figure 5: Odds ratios plotted for each mortality category per unit increase in ozone concentrations ppb (8 hr)

Figure 6: Odds ratios plotted for each mortality category per unit increase in CO concentration (8hr)



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changes in $PM_{2.5}$ concentrations and mortality (Table 4).

The odds ratios for 1, 4 and 8 hr O_3 concentration and respiratory mortality were elevated with wide confidence intervals, with statistical significance observed for 8-hr O_3 concentration (Figure 5). Significant odds ratios were observed for changes in O_3 concentration (1hr, 4hr, 8hr) and CVD mortality with an estimated 0.2 to 0.45% increase in mortality for every 10ppb of O_3 .

Significant associations were also observed for changes in O_3 concentration (8hr) and CVD and respiratory mortality (Figure 5). A significant association was also observed for the relationship between changes in the 8-hr CO concentrations and 'other' mortality but not for other mortality categories (Figure 6).

Discussion

The results of this study suggest that changes in daily air quality in Perth may increase the risks of cardiovascular and respiratory mortality. The significant finding for O_3 concentrations provides an estimated 3% increase in mortality with every 10 ppb change in O_3 concentration. These results are supported by the recent publication by Gryparis et al. (2004) where a significant increase in the number of cardiovascular deaths was associated with both 1hr and 8hr O_3 concentration in Europe. Vedal et al. (2003) report similar findings.

Significantly elevated odds ratios for cardiovascular mortality in relation to NO_2 concentrations were also observed. Similar findings have been reported for other studies using different statistical methods (Dockery et al. 1993; Morgan et al. 1998; Simpson et al. 2000). The significant finding for CO and 'other mortality' is difficult to interpret as 'other' mortality consists of many diseases, which may not be associated with air pollution, and CO concentrations are considered relatively low in Perth. The finding may be a spurious one, particularly in view of the generally low point estimates for the different lags tested.

Other case crossover study design studies have been used to investigate daily mortality and air pollution. The Shanghai study reports a significant increased mortality associated with increases of PM10, SO2 and NO₂ concentrations (Kan, Chen & Jia 2003). The Seoul study found a significant increase in mortality associated with SO₂ concentrations and a non significant but elevated odds ratio for mortality associated with 1 hr O3 and particle concentrations (Lee & Schwartz 1999). Although we also found significant increased mortality associated with NO₂ and O₃ concentrations, it is difficult to compare previous study results with this study since we analysed mortality data into three different categories. In addition, the studies by Lee and Schwartz (1999) and Kan, Chen and Jia (2003) reported increased mortality associated with changes in SO_2 concentrations. One of the benefits of this study is the absence of SO₂ and the strength of the significant findings for the pollutants in the absence of SO_2 .

The limitations of this study are similar to those of time series studies: its inability to distinguish between mixtures of pollutants; its difficulty in determining independent effects from a range of interrelated, correlated factors; and the confounding of co-pollutant effects. One of the most significant problems is the limited exposure data available.

Conclusion

Our findings are consistent with the findings of other studies reported in the national and international literature. Since air quality in Perth has been associated with increased risks of cardiovascular mortality further development and implementation of the Air Quality Management Plan for Perth will contribute to reducing air pollutant concentrations and improving the health of Western Australians. A.L. Hinwood, N. de Klerk, C. Rodriguez, T. Runnion, P. Jacoby, L. Landau, F. Murray, M. Feldwick and J. Spickett

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Investigation of a Large Outbreak of Gastroenteritis in a Residential Educational Institution: Lessons for Public Health Practice

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In June 2002, a large outbreak of gastroenteritis occurred in a tertiary residential training facility in Southern New South Wales. Outbreaks of gastroenteritis in residential facilities are common in NSW, and can recur at the same premises from time to time. We undertook a detailed investigation of the outbreak to determine effective control measures, and to prevent ongoing transmission of gastrointestinal disease at the facility. We distributed questionnaires to all staff and students to determine a detailed food history, environmental exposures and symptoms. A casecontrol study was conducted to identify specific associations with illness. Drinking water, faecal and vomitus samples were submitted for laboratory analysis. Environmental investigations of food services and residential facilities were undertaken. Gastrointestinal symptoms were reported by 188 (24%) of 786 respondents. No statistically significant associations between illness and the consumption of particular foods or drinking water were found. Living at the facility was significantly associated with developing acute gastroenteritis (OR: 24.0, 95% C.I. 2.9-532.9). No pathogens were isolated from 7 faecal and 1 vomitus samples from cases and water sources at the facility. Soap and hand-drying equipment were not available in shared bathrooms and toilets. The outbreak ended after improved handwashing practices were promoted throughout the facility. The likely primary mode of transmission in this outbreak was person-to-person spread with the possible contribution from environmental contamination of bathroom areas. Reported symptoms were consistent with norovirus infection. Early identification and effective management of outbreaks in institutions can provide the basis for the implementation of long-term interventions to prevent their recurrence in the future. It is essential for the public health investigator to gather a broad range of data from epidemiological, environmental and clinical sources to inform effective interventions.

Key words: Norovirus, NLV, Handwashing, Outbreak, Data Collection

On the 19 June 2002, a tertiary training institution (Facility A) reported an outbreak of gastroenteritis to the Southern New South Wales Public Health Unit. Ten people had recently presented to the facility's clinic with acute gastroenteritis. Of these, two cases were hospitalised. Facility A had an on-campus residential population of approximately 700 people, and an extra 200 people attended during the day. Staff and students undertook a number of common activities, including attendance at meals, teaching sessions, and assemblies. Residents were accommodated in single rooms in 20 buildings of 2-4 storeys. Most buildings had 16 rooms on each floor, with shared bathroom facilities. Food was available from two outlets, which were serviced from a kitchen located on campus.

Outbreaks of gastroenteritis in institutions are common in New South Wales, and are notifiable to NSW Health by doctors and hospitals under the Public Health Act 1991. We investigated the outbreak to determine the possible cause, and to inform control measures.

Methods

Epidemiologic investigation

Descriptive epidemiology

The cases of self-reported gastroenteritis reported to the public health unit were interviewed using a questionnaire to collect descriptive epidemiological information and to develop a case definition. Menus were obtained from the institution for meals served by the onsite food outlets for the 16-18 June. We distributed a self-administered comprehensive questionnaire to all staff and students (n=900) to collect detailed information on age, sex, residence, symptoms, food histories and usage of drinking water fountains.

Case-control study

We undertook a case-control study to further characterise the outbreak, identify risk factors for illness including food and drinking water, and to determine effective control measures. We defined a case as a person who attended Facility A and reported onset of vomiting and/or diarrhoea plus at least one of nausea, abdominal cramps, fever, headache and myalgia between 16 and 25 June.

A control was defined as a person who attended Facility A but did not report any gastrointestinal illness between 16 June and 25 June. We numbered each returned questionnaire and randomly selected cases and controls using a random number generator. Data were analysed using Epi-Info version 6.04d (Centers for Disease Control and Prevention, Atlanta). Odds ratios and 95% confidence intervals were calculated by univariate analysis for each exposure. Stratified Mantel-Haenszel odds ratios were calculated for cases and controls living at Facility A.

Environmental investigations

We evaluated the food services of the institution on the 19 June for food handling and storage practices and reviewed leave records for food handlers and other kitchen staff for absenteeism due to illness. We obtained a sample of water from all public drinking water fountains on the campus for analysis of drinking water quality. We inspected the residential facilities and the common toilet and bathroom areas of the campus for handwashing facilities and to determine existing cleaning regimes.

Laboratory investigations

We requested that local general practitioners and the local hospital collect faecal or vomitus samples from patients presenting with gastrointestinal symptoms (vomiting and/or diarrhoea) for laboratory investigation. The samples were cultured for salmonella, campylobacter and shigella, examined by microscopy for parasites including Giardia and Cryptosporidium, and antigen tested for rotavirus and adenovirus. Samples were tested for norovirus (formerly Norwalk-like virus) reverse by transcription-polymerase chain reaction (RT-PCR), viral culture and direct electron microscopy (DEM).

Water samples obtained from drinking water fountains at Facility A were tested by standard methods for the presence of faecal indicator bacteria, such as faecal and thermotolerant coliforms, using standard methods.

Results

Epidemiologic investigations

Of the 900 questionnaires distributed to staff and students, 786 (87%) were returned. Of these 188 (24%) reported onset of gastrointestinal symptoms between 11 June and 25 June, of which 140 (18%) fitted the case definition. Forty eight individuals were reported with illness either by the health clinic of Facility A or from active hospital surveillance. (9%) cases presented to the local hospital with symptoms of gastroenteritis but none required admission.

Case-control study

The case-control study included 31 cases and 36 controls (Table 1). The majority of cases and controls were male (68% and 78% respectively). The median age for cases was 26 years, and 28 years for controls. Most (97%) cases lived on site, as did 56% of controls.





The first case of self-reported gastroenteritis occurred on 11 June, with the final cases experiencing the onset of symptoms on the 25 June (Figure 1). The peak incidence of 106 (56%) of cases occurred between the 17-19 June. The median duration of illness was 24.5 hours, with a range of 12 hours to 11 days.

Of the 140 cases, the median age was 26 years (range 18-54 years), 108 (77%) were students and 94 (67%) were male. Most (103/74%) cases resided at Facility A. Reported symptoms included diarrhoea (120/86%), headache (106/76%), abdominal cramps (96/69%), vomiting (78/56%) and fever (66/47%). Thirteen

Living at Facility A was strongly illness correlated with (Table 1). Attendance at dinner on the 17 June and breakfast on the 18 June were also associated with illness. Of the 260 possible menu items, only rump steak and orange juice consumed at the dinner meal on the 17 June were associated with illness (Table 1). The use of drinking water fountains at the facility was not significantly associated with illness. When stratified for living on site, no significant associations between attendances at meals, consumed food items, and consumption of drinking water were found.

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Characteristics	Cases (n=31)	Controls (n=36)	OR	CI	Living on site Cases (n = 30)	Living on site Controls (n = 20)	Adjusted OR	Adjusted Cl
Age (Yrs)	2//10 50	20/10 55			27/10 50	20/10 4/		
(riedian/kange)	20/19-50	28/19-55			21/19-50	28/19-40		
Sex (Male)	21 (68%)	28 (78%)	0.8	0.3-1.8	21 (70%)	18 (90%)	1.85	0.8-6.9
Student	24 (75%)	20 (56%)	2.7	0.8-9.3	23 (77%)	15 (75%)	1.1	0.2-4.9
Live Onsite	30 (97%)	20 (56%)	24.0	2.9-532.9	30 (100%)	20 (100%)		
Breakfast 16 June	9 (29%)	5 (14%)	2.5	0.6-10.4	9 (30%)	5 (25%)	1.3	0.3-5.7
Orange Juice	8 (26%)	3 (8%)	3.8	0.78-20.9	8 (27%)	3 (15%)	2.1	0.4-11.8
Lunch 16 June	11 (36%)	8 (23%)	2.1	0.7-7.5	12 (40%)	8 (40%)	1.0	0.27-3.75
Orange Juice	5 (16%)	4 (11%)	1.5	0.3-7.9	5 (17%)	4 (20%)	0.8	0.2-4.3
Breakfast 17 June	21 (68%)	15 (42%)	2.6	0.84-7.85	20 (67%)	15 (75%)	0.7	0.2-2.8
Orange Juice	12 (39%)	8 (22%)	2.2	0.7-7.5	12 (40%)	8 (40%)	1.0	0.3-3.8
Lunch 17 June	24 (75%)	17 (48%)	3.8	1.2-13.0	24 (80%)	16 (80%)	1.0	0.2-5.0
Orange Juice	14 (45%)	7 (19%)	3.4	1.0-11.87	14 (47%)	7 (35%)	1.6	0.4-6.2
Dinner 17 June	27 (88%)	15 (42%)	9.0	2.3-38.9	27 (90%)	14 (70%)	3.9	0.7-23.8
Rump Steak	20 (65%)	10 (28%)	4.7	1.5-15.5	20 (67%)	10 (50%)	2.0	0.5-7.6
Orange Juice	17 (55%)	8 (23%)	4.3	1.3-14.3	17 (57%)	8 (40%)	1.9	0.5-7.4
Breakfast 18 June	24 (75%)	12 (34%)	6.9	2.0-24.2	24 (80%)	12 (60%)	2.7	0.6-11.6
Orange Juice	17 (55%)	8 (22%)	4.3	1.3-14.3	13 (43%)	6 (30%)	1.8	0.5-7.1
Lunch 18 June	24 (75%)	17 (48%)	2.7	0.8-8.9	23 (77%)	17 (85%)	0.6	0.1-3.1
Orange Juice	13 (42%)	6 (17%)	3.6	1.0-13.2	12 (40%)	11 (55%)	0.6	0.2-2.0
Dinner 18 June	20 (65%)	15 (42%)	2.5	0.8-8.2	19 (63%)	15 (75%)	0.6	0.1-2.4
Orange Juice	12 (39%)	19 (53%)	1.4	0.5-4.5	6 (20%)	9 (45%)	0.3	0.1-1.0
Water Fountains	10 (33%)	14 (39%)	0.8	0.3-2.5	9 (30%)	9 (45%)	0.5	01-20

Table 1: Characteristics and exposures of cases and controls

Environmental investigations

Food services

The institution was serviced by a large cafeteria and a smaller café-style business. The food preparation and storage facilities of the cafeteria were used by both premises. The café-style premises prepared sandwiches, rolls and pastries. The larger cafeteria prepared three daily meals for over five hundred people, mainly residents. The food service facilities contained a servery for hot meals and refrigerated self-serve salad bars for side dishes and for the preparation of sandwiches. Beverages were dispensed from self-service refrigerated units. Both premises had an externally-audited food safety program in operation, involving the regular monitoring of storage temperatures, a cleaning schedule of surfaces and fixtures, and a pest control program. The food

handling practices of staff complied with the acceptable standard (Food Standards Australia New Zealand 2004), with handwashing facilities located in close proximity to food handling areas and serviced with soap and towels. The refrigerated self-service facilities lacked effective sneeze barriers and were not routinely supervised by staff.

No food handling staff member had reported illness or taken sick leave in the previous week. A staff member was absent from work on the 19 June with a gastrointestinal illness that developed in the late evening of 18 June. The staff member was not a food handler.

Residential and bathroom facilities

On inspection more than 50% of common bathroom facilities serving 700 residents

throughout Facility A, including those located near the food services and classrooms, did not have soap and hand towels. Facility A's management reported that bathrooms were cleaned daily, and soap was not routinely provided.

The drinking water for Facility A was supplied by a filtered and chlorinated municipal treatment system. No increases in the incidence of gastrointestinal illness were noted in the community immediately before or after the outbreak.

Laboratory investigations

Samples of 7 stools and 1 vomitus were submitted from 7 cases for analysis. Two of these cases were included in the case-control study. None of the enteric pathogens tested for were identified in any samples. Analysis of water samples from three drinking water fountains did not detect the presence of faecal indicator bacteria.

Interventions

Public health interventions began on the 19 June, with provision of soap and hand towels in all bathrooms. The public health unit distributed fact sheets to all staff and students, highlighting the importance of handwashing and the regular cleaning of bathroom facilities. We directed the food services management to upgrade the refrigerated self-service units to ensure compliance with legislative requirements (Food Standards Australia New Zealand 2004), and advised that drinking water fountains should not be used until sample results were known.

We recommended that Facility A incorporate a management plan for prevention and control of future outbreaks into their standard operating procedure. This included the ongoing provision of adequate handwashing facilities, disinfection of bathroom and common facilities, infection control strategies for outbreak management, and enhanced cleaning regimes to prevent transmission. Despite the fact that a causative agent was not isolated during this outbreak, the case information suggests a viral aetiology, and matches the clinical and epidemiological criteria for an outbreak of norovirus, formerly Norwalk-like virus (Centers for Disease Control and Prevention 2001; Hedburg & Osterholm 1993; Kaplan et al. 1982).

The case-control study identified that living on site was strongly associated with developing disease. The epidemic curve suggests a person-to-person transmission, although the lack of data available for other exposures, such as contacts between cases, attendance at meals, and eating at the café in preceding days does not allow for further analysis. The association hetween attendance at particular meals, and food items such as rump steak and orange juice, and the development of illness diminished when stratified for living on site.

The laboratory investigation of the case samples submitted for analysis failed to detect a pathogen. The drinking water fountains at the facility were tested in response to anecdotal concerns regarding water quality, however, no indications of faecal contamination were found, and the epidemic curve of disease, and absence of similar disease in the wider community served by the same water source, makes the possibility of drinking water contamination unlikely.

Noroviruses are a major cause of viral gastroenteritis in adults worldwide (Centers for Disease Control and Prevention 2001). Outbreaks due to norovirus may be due to a number of transmission routes, such as person-to-person, from point sources including contaminated food or water supplies, environmental contamination, and in some cases airborne transmission, has been suspected (Centers for Disease Control and Prevention 2001; Marks et al. 2000; Beller et al 1997; and Parashar et al. 1998). The environmental distribution and persistence and low infectious dose of norovirus strongly contributes to the transmission of disease during outbreaks

(Cheesbrough et al. 2000). Infection with norovirus does not confer long-term immunity, however, there is a suggestion that infection may lead to short-term immunity to the specific strain involved (Centers for Disease Control and Prevention 2001). Facility A has a dynamic population which fluctuates every few months with the recruitment and graduation of cohorts, and is therefore unlikely to significantly benefit from this immunity and may remain susceptible to future outbreaks. Handwashing using soap has been demonstrated as an effective means of reducing the microbiological contamination of hands, and the use of paper handtowels are considered preferable to hot air dryers (Montville, Chen & Schaffner 2002). Maintaining the supply of soap and handtowels to communal bathrooms is essential to prevent the occurrence of long-term transmission of norovirus across subsequent cohorts, as seen in a number of long-term hotel outbreaks (Kuusi 2002).

The staff and students at this facility participated in a significant number of common activities, including training and attendance at meals, which may have provided opportunities for the transmission of viral agents. Outbreaks of norovirus have been shown to cause considerable environmental and fomite contamination, particularly in areas where large amounts of virus may be excreted, such as toilets and bathrooms (Cheesbrough et al. 2000). The absence of soap at many of the communal bathrooms may have contributed to the transmission of disease by limiting the effectiveness of handwashing (Montville, Chen & Schaffner 2002). The bathrooms were reportedly cleaned daily, however, the potential for recontamination by vomiting and diarrhoea in these shared facilities may necessitate more frequent cleaning and disinfection to reduce ongoing transmission during an outbreak.

The investigation was limited by several factors, including the failure to isolate an

infectious agent and the difficulty in obtaining detailed information rapidly from a large population. Only 4% of cases submitted samples for laboratory analysis, despite requests by investigators. The need to implement public health control measures in a timely manner necessitated rapid data collection from the large population of Facility A. Consequently, the investigation was limited to a brief food history, demographic information, and symptoms. Investigators were unable to analyse the outbreak in relation to risk factors such as contact with ill cases and a food history taken over a longer period. The food histories were focused on the cafeteria, while limited data were collected from the smaller café on site. A selection bias in the case-control study became apparent during the analysis. Cases and controls differed significantly in regard to gender, whether they were staff or students, and whether they lived at Facility A. Gender differences were unlikely to affect the likelihood of exposure, however, being a resident of Facility A would substantially increase the frequency of contact with the communal bathrooms. More cases than controls lived on site. When living on site was adjusted as a confounder in the stratified analysis, 44% of the controls were necessarily excluded from the analysis, compared to 3% of cases.

In this outbreak investigators relied upon the range of available epidemiological and clinical data to determine the likely applying bv aetiology established epidemiological criteria (Hedburg & Osterholm 1993; Kaplan et al. 1982). This outbreak is therefore an example of a multifactorial approach to disease investigation and highlights the importance of collecting broad а range of information for analysis. We found that this methodology was useful and reinforces the crucial importance of environmental, epidemiological, and laboratory collaboration.

Conclusion

The provision of basic hygienic services, such as soap and handtowels, should be essential in common bathrooms, particularly in residential institutions. Outbreak response plans, incorporated into existing institutional operating plans, may be a useful way of rapidly identifying and controlling outbreaks as they arise. Response plans may include the implementation of rigorous cleaning regimes during outbreaks, the maintenance of a sickness log for staff and students, a prepared communication strategy for the rapid dissemination of information, and protocols for liaison with health agencies.

Although we suspect that the primary vehicle of transmission during this outbreak was person-to-person and environmental contamination, future investigations of this type may consider obtaining a variety of samples for analysis, including environmental samples, to assist in the understanding of such events. In response to the frequency of gastrointestinal outbreaks associated with residential and institutional facilities, NSW Health are developing guidelines for use by public health authorities and institutions to ensure that outbreaks are quickly identified and controlled.

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Investigation of a Large Outbreak of Gastroenteritis in a Residential Educational Institution: Lessons for Public Health Practice

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Occupational Hygiene Monitoring of Respiratory Hazards: A Case Study

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Minara Resources, located at Murrin Murrin in Western Australia, extracts nickel and cobalt from lateritic ores. Employees may be exposed to a range of potentially hazardous substances. The aim of this preliminary investigation was to identify hazards, develop air monitoring protocols, describe exposure patterns, quantify risks and recommend appropriate interventions to protect workers. Engineering controls have been put in place to control exposure to dusts including nickel and cobalt in the refinery. In this study there were occasional excursions due to, for example, sweeping cobalt dust instead of vacuuming. This study has highlighted areas where nobalt exposures need to be reduced. Gas exposures, particularly to hydrogen sulphide are of concern in specific areas of the plant. It is during maintenance tasks that the highest exposures may occur. Where possible deposition of dust should be engineered out, but good housekeeping practices are also necessary to reduce cobalt levels. Because it is highly unlikely that all potential gas exposures can be controlled through engineering means during maintenance, there are gas emission alarms and a stringent respiratory protection program. All employees and visitors to the site wear personal hydrogen sulphide monitors with audible alarms and an escape respirator. Respiratory protection is worn in areas where workers are at risk of exposure to nickel and cobalt dust. Further engineering controls have been budgeted and are to be implemented. Dust reduction strategies such as enclosing certain operations, provision of showers and change rooms on site and tarring of roads in the plant area need to be investigated. Respiratory health surveillance and urinary nickel and cobalt biomonitoring will be implemented for all workers.

Key words: Nickel; Cobalt; Dust Exposures; Respiratory Hazards

The Minara - Murrin Murrin site consists of open cut mining pits and a major chemical processing plant. The products produced at Murrin Murrin are nickel and cobalt briquettes. As a consequence of the complicated chemical process used to extract nickel and cobalt from the ore, there is potential for employees and contractors to be exposed to a wide variety of chemical hazards. Hygiene monitoring as required by the West Australian Department of Industry and Resources has been conducted at Murrin Murrin since the plant was first commissioned in July 1999. Due to the complex nature of the Murrin Murrin operation it was decided that a more refined monitoring protocol is necessary to adequately quantify worker exposures and recommend interventions (Wing & Cross 2003).

Significance and Purpose of the Study

The mining and processing of nickel and cobalt are high-risk activities. Workers are exposed to a variety of chemicals and dusts that could have both acute and/or chronic effects. Various carcinogens are also present in this work environment (Nickel Producers Environmental Research Association 1997). The purpose of this study was to design and implement a comprehensive best practice occupational hygiene-monitoring program for Murrin Murrin.

Process Overview

The Murrin Murrin operation is located in a dry and dusty region of Western Australia on the edge of the Gibson Desert. Workers operate on a 'fly in, fly out' schedule. Other than a mine village located approximately 10 km from the plant there are no other permanent settlements in close proximity to the plant. The Murrin Murrin refinery operations are not fully enclosed, thus the facility is subjected to the prevailing environmental conditions which are often windy and dry.

Mining

The process at Murrin Murrin commences with the mining of ore in several open cut pits. The ore is transferred to the refinery where it is stockpiled and fed into the processing plant.

Utilities

Electrical power is generated using both steam and gas turbines. Furthermore potable and demineralised water, hydrogen sulphide gas, sulphuric acid, oxygen, nitrogen and hydrogen are produced for use in the plant.

Ore leach

Ore is screened to remove large rocks and is then crushed in a mill. Water is added to produce slurry which is fed into a series of autoclaves containing sulphuric acid. The autoclaves operate at a temperature of approximately 250°C and 40 atmospheres pressure. During this process metals are extracted from the ore into solution. The autoclaved slurry is fed through settling tanks where solids are removed and pumped to the tailings dam as waste. Hydrogen sulphide gas is added to the solution.

Refinery

Hydrogen sulphide gas facilitates the precipitation of metal sulphides which are

autoclaved in the presence of oxygen. During the solvent extraction phase nickel and cobalt is separated into two streams and iron and zinc is removed. Both streams are autoclaved in the presence of hydrogen and ammonia which reduces cobalt and nickel into metal powders. The powders are fed into pug mills and a sintering furnace to produce nickel and cobalt briquettes (Anaconda Operations 2002).

Potential Health Effects

Workers at Murrin Murrin are potentially exposed to a range of contaminants that can impact on their health, in particular the respiratory system.

Nickel dusts

Nickel can be classified as follows:

- Metallic (elemental and alloyed) which is currently classified as not a suspected carcinogen.
- ii) Oxidic (including hydroxides, carbonates, complex Ni-Cu oxides, forms of nickel oxide, etc) which have been linked to respiratory cancers.
- iii) Sulphidic (including the subsulphide) which is carcinogenic.
- iv) Soluble (including sulphate and chloride) which is suspected to be a cancer promoter rather than an initiator.

Murrin Murrin is known to create metallic, soluble and sulphidic forms of nickel during the refining process (Wing 2003).

Cobalt dusts

Exposure to cobalt dust has been linked to asthma, pulmonary function changes and myocardial effects. The American Conference of Governmental Industrial Hygienists (ACGIH) classified cobalt as an A3 - Confirmed animal carcinogen with Jacques Oosthuizen and Martyn Cross

unknown relevance to humans, while the International Agency for Research on Cancer (IARC) classify it as 2B - Possibly carcinogenic to humans (Wing 2003).

Sulphuric acid mist

Sulphuric acid, which is manufactured on site, is corrosive and exposure to sufficient concentrations causes skin, eye, respiratory and gastrointestinal irritation. Exposure to high concentrations causes chemical burns on exposed areas of the body. Inhalation of sulphuric acid mist causes irritation, coughing, bronchitis, ulceration, nose bleeds, lung tissue damage, chemical pneumonitis, pulmonary oedema and death. Sulphuric acid mists has been classified as a Group 1 carcinogen (carcinogenic to humans) by the IARC (International Agency for Research on Cancer 2003a), and as an A2 carcinogen (suspected human carcinogen) by the ACGIH (Wing 2003).

Hydrogen sulphide

Hydrogen sulphide gas can be detected at very low levels due to its pungent odour. Low level exposures cause eye irritation and conjunctivitis. Acute exposures to high concentrations cause paralysis of the olfactory sense (ability to smell). At high levels of exposure pulmonary oedema, cessation of breathing and death will occur. Chronic low level exposure or repeated high level exposures cause headache, fatigue, dizziness, irritability, and loss of libido (Wing 2003).

Sulphur dioxide

Acute health effects associated with Sulphur dioxide include eye and upper respiratory tract irritation as well as narrowing of the airways, runny nose and coughing. Chronic exposure can cause permanent pulmonary impairment (Wing 2003).

Ammonia

Ammonia causes irritation of the respiratory tract and eyes (Wing 2003).

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Methods

Design

The National Occupational Health and Safety Commission's (NOHSC) guideline, 'Guidance Note for the Assessment of Health Risks Arising From the Use of Hazardous Substances in the Workplace' (NOHSC 1994) was used to identify and review all chemical hazards present on site. The review, facilitated by an occupational hygienist was conducted with a multidisciplinary team including engineers, process chemists and departmental teams (Wing & Cross 2003).

- 1. All chemical and dust exposure areas on site were identified and described in terms of the process, tasks and the form the contaminants are in at various stages of the operation. To ensure that this inventory was as accurate as possible, operators and supervisors from the various areas were involved.
- 2. Literature and codes of practice were reviewed in order to identify shortcomings in the existing sampling strategies.
- 3. An intensive monitoring program was implemented.
- 4. Data were analysed and assessed.
- 5. Results were communicated to management and all employees.
- 6. Appropriate control measures were recommended.

Sampling methods

 Respirable Dust was sampled according to AS 2985-1987 -Workplace Atmospheres - Method for sampling and gravimetric determination of respirable dust (AS 2985 - 1987).

- Inspirable Dust was sampled according to AS 3640-1989 Workplace Atmospheres Method for sampling and gravimetric determination of inspirable dust (AS 3640 1987).
- Organic Vapours were sampled according to AS 2986 - 1987 -Workplace Atmospheres - Organic Vapours - Sampling by Solid Adsorption Techniques (AS 2986 -1987).
- Asbestos Fibres were sampled according to the guidance note on the membrane filter method for estimating airborne asbestos dust. (NOHSC 1988).

Samples requiring laboratory analysis were dispatched to an external National Association of Testing Authorities (NATA) accredited laboratory. Most of the monitoring was conducted across a full shift (ie. as close as possible to 12 hours) and was conducted on operators potentially exposed to the contaminants under consideration. Shifts and individuals to be monitored were selected randomly.

Occupational hygiene data dating back to the first samples taken shortly after the plant was commissioned were aggregated and descriptive statistics were used to establish a baseline and identify areas of concern. The total number of samples taken to achieve statistical significance was determined according to the methodology of Mulhausen and Damiano (Mulhausen & Damiano 1998). Sample numbers required depended upon the mean measured exposure levels, range and standard deviation of the samples and the prescribed exposure standard. In some cases it was not possible to collect sufficient samples due to time and resource constraints.

Discussion

The following limitations were identified:

- 1. Sampling was conducted randomly and environmental factors such as wind and rain could have influenced results.
- Changes in production or maintenance requirements could have impacted on normal operations thus making sampling less representative of normal working conditions.
- 3. In some instances samples may have been tampered with 'salted' to intentionally produce a high reading.
- In some cases it was not possible to collect sufficient samples to achieve statistical significance given time and budgetary constraints of the project.

Ore leach

Inspirable dust was reported at a mean concentration of 3.06 mg/m^3 , SD 3.42, which is well within the occupational exposure standard (OES) of 7 mg/m³. One sample exceeded the OES with a result of 9.3 mg/m³. The mean iron concentration was 0.81 sd 0.74, which was well below the OES of 1. However, 1 sample exceeded the standard of 0.9 mg/m³ with a value of 1.6 mg/m³.

All asbestos, arsenic, nickel, cobalt, copper, mercury, zinc and chromium samples were below their respective OESs. Samples of sulphuric acid mist and hydrogen sulphide were also all within the OES.

Refinery

The refinery is considered to be the area within the plant where most potentially dangerous exposures could occur. Jacques Oosthuizen and Martyn Cross

Table	1: A	gg	rega	te s	amples	for
obera	tors	in	the	Ore	Leach	area

Insp	irable dust (mg/m³)	lron (mg/m³)
OES	7	I
26/11/ 00 - 18/03/04	n = 32	n = 27
Mean	3.06	0.81
SD	3.42	0.74
Number over OES	Ι	Ι

Respiratory protection is worn in areas of the refinery that have been demarcated as respirator zones. All samples of copper, iron, silica, VOCs, benzene, ethyl benzene, toluene, xylene and sulphuric acid mist were within their respective OESs.

There were some excursions of inspirable dust, respirable dust, nickel, cobalt, cristobalite and silica above the OES, however, these were measured in areas where respiratory protection is worn. detector and respirator at all times. This detector alerts the wearer to concentrations exceeding 10pmm. When this occurs, the wearer is required to don their respirator and evacuate the area. In this way, individuals are protected from acute health effects arising from high exposures (e.g. if a valve or tank leaks). Insufficient research has been conducted to determine adequately the risks posed to employee health by constant lowlevel exposure to hydrogen sulphide.

Calcrete plant

The calcrete plant presents a significant respiratory hazard with the mean inspirable dust level (12 mg/m³) as well as 4 out of 12 samples exceeding the OES of 7 mg/m3. The maximum concentration measured was 110 mg/m³. Three calcium carbonate samples out of 33 exceeded the OES with an upper value of 30 mg/m³.

Table 2: Aggregate samples for Refinery operators

	Inspirable dust (mg/m ³)	Respirable dust (mg/m ³)	Nickel (mg/m³)	Cobalt (mg/m³)	Cristobalite (mg/m³)	Silica (mg/m³)
OES	7	3.5	0.9	0.05	0.1	0.14
18/12/00 - 02/04/04	n = 213	n = 31	n = 201	n = 228	n = 6	n = 8
Mean	0.93	2.93	0.17	0.02	0.8	2.24
SD	1.06	3.56	0.31	0.03	0.99	4.16
Number over OES	5	8	15	37	I	2

OES = occupational exposure standard, SD = Standard deviation

Utilities

Inspirable dust, sulphur, lime, respirable dust, quartz, christobalite, nickel, cobalt sulphuric acid mist and Sulphur Dioxide samples were found to be within the OES. TWA (time weighted average) samples of hydrogen sulphide and ammonia were within the OES, except for a few short term 'excursions' with a peak H_2S value of 55 ppm and ammonia reached a peak of 97 ppm.

A hierarchy of respiratory protection is used in the H_2S areas. When the levels are unknown an airline respirator or self contained breathing apparatus is used. At levels below 300 ppm a full faced respirator is used. Everybody working at Murrin Murrin is required to carry a personal gas Respirable dust, silica, nickel and cobalt samples were below the OES.

Table 3: Calcrete Plant operators

	Inspirable dust (mg/m³)	Calcium Carbonate (mg/m³)
OES	7	10
05/10/99 - 10/04/04	n = 36	n = 33
Mean	12	5
SD	21.9	8
Number over OES	12	3

OES = occupational exposure standard, SD = Standard deviation

Mining

All asbestos, cristobalite, silica, copper, iron and nickel samples obtained from the

mining environment were below the relevant OES values. 4 out of 32 inspirable, 3 out of 135 respirable dust, and 4 out of 70 cobalt samples exceeded the OESs, however, all mean sample values were below the OESs.

Table 4: Mining

	0		
	Inspirable dust (mg/m³)	Cobalt (mg/m³)	Respirable dust (mg/m³)
OES	7	0.05	3.5
01/03/00 - 29/03/04	n = 32	n = 70	n = 135
Mean	5.2	0.05	0.63
SD	4.07	0.06	0.79
Number ove	r OES 4	4	3

OES = occupational exposure standard, SD = Standard deviation

Laboratory

Five out of 36 respirable dust samples, with a maximum value of 7.6 mg/m³ and 1 cobalt sample with a value of 0.06 mg/m³ exceeded the OES. However, 6 out of 12 Inspirable dust samples exceeded the OES (mean 23.73, SD 34.33, Max 110 mg/m³).

All nickel, aAsbestos, cristobalite and quartz samples were below the OES.

Table 5: Laboratory

	Respirable dust (mg/m³)	Inspirable (mg/m³)	Cobalt dust (mg/m³)
0ES	3.5	7	0.05
14/03/04	n = 36	n = 12	n = 28
Mean	2.21	23.73	0.02
1.81	34.33	0.02	
Number over	OES 5	6	Ι

OES = occupational exposure standard, SD = Standard deviation

Recommendations

Ore leach

In the ore leach area intermittent dust exposures above the OES occur and alternative dust control strategies should be considered. As an interim precautionary measure respiratory protection for workers should be maintained. Routine dust monitoring of this area should take place on a three-monthly cycle.

Refinery

Refinery workers are potentially exposed to nickel and cobalt dust. Therefore a number of measures should be undertaken:

- additional engineered dust reduction measures should be considered
- monthly random personal dust monitoring must continue
- health promotion interventions aimed at improving personal hygiene and understanding of strategies to reduce exposure should be implemented
- strict enforcement of respiratory protection measures must be maintained
- biological monitoring and health surveillance of refinery workers, particularly for cobalt exposure and respiratory function must be implemented
- dust monitoring should be conducted monthly.

Calcrete operation, mining and laboratory

- Dust control measures need to be upgraded
- respiratory health surveillance and respiratory protection needs to be implemented and maintained
- monthly dust monitoring must be implemented.

General

• Current early warning systems for gas exposures must be maintained and regular monitoring of gas exposures should continue.

- Maintenance and shut down tasks appear to pose the greatest risk to employees in terms of exposures to hazardous substances. The current protocols of job risk assessment and implementation of adequate safety precautions and enforcement of protective equipment protocols must be maintained at the current high standard. Contractors must be subjected to the same training and induction as Minara employees and they must abide by the same code of conduct and policies.
- Tarring roads and providing concrete walkways will significantly reduce wind blown dust and prevent the transportation of contaminated dust back to the mine village by vehicles
- The provision of change rooms, lockers and showers as well as laundering facilities at the workplace will further reduce contamination of rooms and bedding at the village. This will improve general hygiene of the operators thus reducing the risk of accidentally ingesting hazardous substances. This is particularly important for refinery workers.
- The Murrin Murrin site is unique in that there are multiple agents that impact upon the respiratory system. Workers may therefore be exposed to acceptable levels of contaminants if considered in isolation, but not much is known regarding the additive or synergistic effects of

the combined exposures. Health surveillance, particularly of the respiratory system is recommended.

Conclusion

Occasional exceedances of OESs for nickel and cobalt are known to occur within the packaging sheds in refinery. Therefore, it is compulsory to wear respiratory protection in these areas. Respiratory protection is also required in the laboratory sample preparation area and the calcrete plant due to the presence of dust. Additional controls have been implemented since this study. It is predictable that hydrogen sulphide gas will be emitted during maintenance work, and due to agitator seal failures. Gas emission monitors have been strategically placed for early detection and a strict respiratory protection program is in place. All personnel on site have adequate training and area specific inductions, which are evaluated.

Through the implementation of extensive and on-going occupational exposure monitoring, personal exposure levels have been quantified and can be benchmarked for future reference. The implementation of a rigorous, targeted health surveillance program will allow for the integration of the environmental and health data.

Minara Resources are in a position to embark upon the establishment of a comprehensive epidemiological surveillance program where exposure profiles can be related to individual worker health data. This will enable the development of 'best practice' protocols in the management of the respiratory health of Murrin Murrin workers.

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Ambient Air Concentrations of Lung Carcinogens and Fine Particles in North West Suburban Adelaide: Implications for Public Health

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The North Western region of Adelaide is a shared residential and industrial area with elevated lung cancer mortality up to twice that of metropolitan averages. This study aims to quantify current airborne carcinogen concentrations in the region, compare them with health based exposure guidelines, and use them as an indicator of potential future respiratory risk. Carcinogens and fine particulates with potential industrial sources in the region were monitored in five locations. Average concentrations were within guidelines. However, diesel exhaust particulate and polycyclic aromatic hydrocarbons were elevated at sites in proximity to heavy vehicle traffic. Diurnal variations in PM_{2.5} included weather and traffic-related short term peaks at most sites, and other peaks potentially related to industrial activity. The data suggest that there is not a significant public health risk at present. However, these results are unlikely to be indicative of previous exposures relevant to contemporary lung cancer cases due to changes in industrial activity since 1970. Further studies should investigate the health significance of the observed short-term peaks.

Key words: Lung Carcinogen; Air Pollution; Exposure; Monitoring

The North Western (NW) region of metropolitan Adelaide, Australia has lung cancer mortality rates ranging from slightly higher to more than twice that expected from state metropolitan averages (Figure 1) (Glover et al. 1996). This high mortality has prompted a case control study, The North West Adelaide Lung Cancer Study (Whitrow et al. 2003), to investigate the risk factors associated with lung cancer in the region. The area under investigation is a region of approximately 10km by 15km, with an 88km² residential area and population of 100,000 (10% of metropolitan Adelaide population). The region is

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bordered to the West by the St Vincent Gulf, to the North by vacant land and some residential homes, and to the East and South by residential areas. The Port River is located in the Mid-North of the area. The river is the site for a cluster of industry that uses the river to transport goods by ship into and out of metropolitan Adelaide. Due to the concentration of air polluting industry in this area, a key factor investigated by the case control study was residential proximity to air polluting industry.

There are 205 industries licensed to carry out 'prescribed activities' in the North West Adelaide Lung Cancer Study geographic

Figure 1: North West region of metropolitan Adelaide



area, comprising 33% of licensed industry in metropolitan Adelaide. Licensed industries are allowed to carry out a variety of polluting activities and regularly report their emissions to the South Australian Environment Protection Authority (EPA). Some industries within the study area are known to have either currently or historically produced lung carcinogens such as asbestos and polycyclic aromatic hydrocarbons (see Table 1).

Table 1: Lung carcinogens (IARC rating 1* and 2A**) and potential sources in the North West of Adelaide

Carcinogen	Potential Source
Asbestos	Abandoned asbestos cement manufacturing factory
Crystalline Silica	Cement manufacturer
Formaldehyde	Foundries, industrial vehicles
Polycyclic Aromatic Hydrocarbons (PAH)	Industrial vehicle traffic, power station, fuel depots
Diesel Exhaust Particulate	Industrial vehicle traffic

* known to be carcinogenic

** probably carcinogenic

For many decades there has been widespread community concern regarding pollutant emissions in the North Western suburbs, and its association with not only lung cancer but also other respiratory illness with elevated incidence in the area, for example, asthma and colds (Glover et al. 1996; Pilotto et al. 1999). Evidence that respiratory health effects are associated with air pollution concentration exists in other industrial and residential areas in Australia, particularly asthma symptoms and residential proximity to operational power stations (Halliday et al. 1993; Henry et al. 1992). However, it is not known conclusively if the air quality in the NW region is poorer than that of other regions, particularly with regards to ambient concentrations of carcinogens. The South Australian EPA conducts periodic sampling for a range of pollutants; however, this sampling is not focused on carcinogenic agents.

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The present study was undertaken in conjunction with the Lung Cancer Case Control Study. It was not designed to provide epidemiological exposure data. Rather, its purpose was to measure contemporary levels of airborne carcinogens in the study's region and to compare these levels to both a control residential site outside of the study boundaries and to national and, where available, international health based exposure guidelines. Given the ongoing co-location of a diverse range of industry and residential homes, we wanted to quantify current levels of airborne lung carcinogens in the area to use this information as an indicator of future respiratory health risk.

Methods

A global literature search and research of local industrial activity were undertaken to identify a list of carcinogens, potentially associated with industrial sources in the NW area, to be included for environmental sampling (Table 1). In addition, we monitored for particulate matter with a mass median equivalent aerodynamic diameter of 2.5μ m (PM_{2.5}) and conducted short term monitoring of ultrafine particulate matter. Fine and ultrafine particulate matter have not been classified as carcinogenic, but are useful indicators of pollution levels, and are able to act as carriers for carcinogenic substances (Pope et al. 2002).

Monitoring locations

Five locations were selected as monitoring sites based on their proximity to industry or to a major traffic intersection, and on lung cancer mortality figures (See Figure 1, site numbers correspond to the list of sites below). These sites were readily accessible to our vehicles transporting the monitoring equipment, and appeared secure from vandalism and tampering.

The five site locations selected for sampling were:

- 1. Osborne residential area closely adjacent to an industrial area (including a soda ash production plant)
- Birkenhead residential area very closely adjacent to industrial area (including a cement works) with high volume of heavy vehicle traffic
- 3. Pt Adelaide high volume, heavy vehicle traffic intersection with some nearby residences
- Mile End high volume traffic intersection, away from North West industrial area
- 5. Kidman Park control residential area with little industry and no heavy vehicle traffic.

Precise monitoring locations were chosen based on criteria in the *Guide for the Siting of Sampling Units* developed by the Committee on Methods for Examination from the Air Standards Association of Australia (Standards Australia 1987a).

Sampling duration and timing

Air sampling was predominantly carried out in March 2002. This period was considered 'mid season' and was chosen to avoid temperature extremes and increase the likelihood of dry conditions (metropolitan Adelaide had been in drought conditions for approximately six weeks prior to this). The sampling is representative of typical conditions rather than a worst-case scenario.

Field work

SKC PCXR universal air sampling pumps were used for low flowrate measurements of asbestos, crystalline silica and diesel exhaust particulate. External high capacity 6V lead acid batteries were fitted to each pump. Prior to sampling all pumps were calibrated at 2.0 L/min, except for respirable crystalline silica, which was 1.9 L/min. Flowrates were checked at the end of each sampling interval.

Asbestos

Airborne asbestos was sampled using 25 mm Nuclepore 0.8 µm polycarbonate filters and anti-static cowls (NOHSC 1988), with subsequent scanning electron microscopy (SEM) by the Centre for Electron Microscopy and Microstructural Analysis, University of Adelaide. Samples were examined using a Philips XL30 field emission gun SEM fitted with an EDAX energy-dispersive spectrometer. The microscope was operated at an accelerating voltage of 20 kV with the spot size set to 3. Non-overlapping regions (10244 µm²) of the filters were examined in a grid pattern. Particles were identified using backscattered electron (BSE) imaging mode in order to distinguish inorganic particles from organic material. An estimate of the number of asbestos particles on a given filter was obtained by searching 20 regions for particles with a fibrous morphology and determining the elemental composition of these particles using EDX spectroscopy (NIOSH 1994).

Respirable crystalline silica

Respirable crystalline silica was sampled using 25 mm *Nuclepore* 0.8 µm polycarbonate filters in miniature cyclones (Standards Australia 1987b). Quartz content was determined by MPL Laboratories, using an in house infrared spectroscopic method (WILAB 4.0) adapted from NIOSH Method 7603 (NIOSH 1998a).

The crystalline silica content of asbestos SEM samples (see above) was also estimated using automated particle counting software (iDXac) from 4 regions of each filters. The iDXac software was set to detect all particles with dimensions over 0.5 µm in size in BSE images.

Formaldehyde

Formaldehyde was sampled using 2,4dinitrophenylhydrazine-coated silica gel passive samplers with an effective sampling rate of 25 ml/minute. Samples were analysed by HPLC with UV detection (NIOSH 1998b).

Polycyclic aromatic hydrocarbons (PAH)

Inhalable PAHs were sampled using the EHL sampler (Kirton 1993; Standards Australia 1989). The analytes were the US EPA 16 priority PAHs and analysis was conducted by the BHP Environmental Health Laboratory, using in house methods (EHL 9 GC/MS for PAH).

Diesel exhaust particulate

Diesel exhaust particulate was monitored using 37 mm quartz filters, in accordance with NIOSH Method 5040 (NIOSH 1998c). Analysis was conducted at the University of Sydney.

Fine particulate (PM_{2.5}) by Dust Trak[™]

 $PM_{2.5}$ levels were sampled using a TSI Instruments Dust TrakTM with 2.5µm inlet. The Dust TrakTM determines $PM_{2.5}$ concentration using light scattering detection. Concentration data, recorded at 1min intervals, were periodically downloaded to a laptop computer at the site. Environmental enclosures (Dust TrakTM Environmental Enclosure Model 8520-1) were used for fixed monitoring locations.

PM_{2.5} by Tapered Element Oscillating Microbalance (TEOM)

 $PM_{2.5}$ sampling at the Osborne site (1) was also carried using a Tapered Element Oscillating Microbalance in a caravan located in the driveway of the residence. A Ruprecht and Patashnick Series 1400A ambient particulate monitor was used. Total flow was 16.67 L/min, with a sample flow of 3L/min. The sample inlet and filter were maintained at 50C. Correction was made for semi volatiles and daily temperature variation. A similar TEOM unit was located at an EPA reference site approximately 6 km West of the Adelaide central business district.

Ultrafine particulate

Short term sampling of ultrafine particles was carried out with a TSI Instruments P-TrakTM which counts particles between 0.02 and 1µm. The P-Trak[™] predominantly measures diesel exhaust and combustion sources (Levy, Dumyahn & Spengler 2002). The P-Trak sampling was undertaken in 15 sites (in addition to the 5 key sites) for short (5 to 15 minute) periods both within and external to the study area.

Meteorological measurements

The prevailing weather conditions at Site 5 were recorded using a portable weather station (Model Number 102083, Climatronics Corporation, Bohemia, NY), [supplied by MEA instruments; Datalogging was with a Unidata Australia Starlogger Model 6004C]. The Bureau of Meteorology supplied other meteorological data.

Figure 2: Typical diurnal variation of PM_{2.5}

Results

Table 2 illustrates the ambient air concentrations of the carcinogens under investigation. The only detectable asbestos was found at Site 4 (a busy metropolitan traffic intersection), all other values being less than the detection limit for the SEM method. Similarly, respirable crystalline silica levels were less than the limit of detection using infrared spectroscopy. However, when particles greater than 0.5 microns were examined using SEM, the highest values of crystalline silica (quartz) were found at Sites 1 and 2 (in close proximity to industrial sites). Formaldehyde levels were well below national and international criteria. Concentrations of PAH including naphthalene were very low and in particular benzo-a-pyrene was not detected in any of the samples. Acenaphthene, fluorene, phenanthrene, benz(a)anthracene and chrysene were detected in small amounts. Apart from naphthalene, no PAHs were detected at the control site (5). As expected, the diesel exhaust particulates concentrations as expressed as elemental carbon were higher at sites 3 and 4, reflecting the higher density of diesel powered vehicle traffic.



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Substance		Units I	2	3	Site 4	5 (Control)	EPA reference site	Standard
Asbestosa	fibres/ml	<0.0002	<0.0002	<0.0002	0.00035	<0.0002		
Crystalline Silicaª	µg∕m³ b	<0.5	<0.5	<0.5	<0.5	<0.5		
	particles/m ^{3 c,d}	52709	29934	14069	17744	12821		
Formaldehyde ^e	µg/m³	5.1	3.8	4.2	2.3	3		100f 188
PAHe: Naphthalene Higher order ^h	μg/m³ μg/m³	0.25 0.005	0.24 0.039	0.74 0.056	0.55 0.027	0.19 <0.001		i
Diesel Exhaust particulatee, ^j	µg/m³	0.8	0.44	1.82	2.06	0.27		k
PM2.5	µg/m³ ™	7.6					6.3	25 "
	µg/m³ •.p	10.1 (9.0-11.0)	4.3 (1.8 - 6.3)	q	.7 (8.6-20.1)	8.1 (6.6-13.1)		
	µg/m³′		12					
	µg/m³ ⁵	15.2	15.4					

able 2: Ambient air concentration	s of lung	carcinogens in	the North	West of Adelaide
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Table 2 Legend

a Averaged over 4 days (25/2 to 1/3/02)

b By infrared spectroscopy

c By scanning electron microscopy d Particles greater than 0.5 m size

e Averaged over 4 days (11/3 to 15/3/02)

f WHO Goal 30 minute average

g NEPM 24 hr average

h Acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(123, cd) pyrene, dibenzo(ah)anthracene, benzo(ghi)perylene (Standard US EPA PAH set)

Proposed NEPM is 0.3 ng/m³ for benzo(a)pyrene as an annual average

Elemental carbon

k NEPM emission guidelines only m TEOM average of 2 days 14 and 15 March 2002

n NEPM Goal 25 g/m³ daily average, 8 g/m³ annual average p Dust Trak average of 4 days (min - max)

q No measurements

Dust Trak 14 March 2001

s Dust Trak 15 March 2001

With regard to PM_{2.5}, the measured concentrations did not exceed the proposed 24-hour National Environmental Protection Measure (EPHC 2003) of 25µg/m^{3.} The highest 4-day average was found at site 2 (industrial). Average Dust Trak[™] data for site 1 are somewhat higher than TEOM data, although there was a strong temporal correlation. Site 1 TEOM results were similar to that of an EPA reference site.

Diurnal variation of PM2.5 was monitored and it was found that peaks often occurred early morning and late afternoon, and in some cases late evening (see Figure 2 for a typical profile). All of the recorded peaks were less than 100µg/m³, as a 1-minute average.

Ultrafine particulate concentrations were generally low (typically 15 000 particles per cm³), and were highly reflective of diesel vehicle traffic. By comparison, site 3 (adjacent to a bus depot) had elevated concentrations, averaging 47 000 particles per cm³, and ultrafine particles in the central business district of Adelaide averaged 50 000 particles per cm³, potentially due to a combination of topography (high rise buildings) and vehicle density.

Dusts deposited on filters collected at each site were subjected to SEM. It was found that they constituted mainly aluminosilicates, calcite and gypsum (each higher at Sites 1 and 2), halite and other common soil minerals.

Discussion

This study has demonstrated that air quality with respect to specific respiratory carcinogenic substances in the North Western suburbs of metropolitan Adelaide is satisfactory according to national and international health based guidelines.

With one exception, airborne asbestos was not detectable. This is consistent with other studies of environmental asbestos nonoccupational exposure to mineral fibres (IARC 1989), and is to be expected given the lack of operational contemporary sources. Unlike occupational situations, there is a lack of data on airborne crystalline silica in the general environment. The significance of the observed low levels of quartz is thus unclear at this stage. Airborne formaldehyde was present at very low levels, again consistent with EPA data (Environment Protection Authority 2003). Across the sites PAHs and diesel exhaust particulate data were closely related, possibly suggesting a principal contribution local traffic particularly from as concentrations were highest at high density traffic sites.

PM_{2.5} data from TEOM and Dust Trak[™] instruments indicate relatively low average concentrations, with some variability during the day that appears to be due to a combination of local and mesoscale meteorology (Manins et al. 2001), motor vehicle traffic and, in the case of Sites 1 and 2, some industrial contribution. The instantaneous absolute concentrations may exceed 25ug/m³, which, if sustained, could be associated with respiratory health effects in susceptible individuals, particularly illness with short latency such as asthma and colds. For example, a previous Australian clustered randomised control study demonstrated respiratory health effects (sore throat, colds) in children from short-term exposure to high concentrations of indoor nitrogen dioxide (Pilotto et al. 1997). The interpretation of our data should be tempered by the limited amount of real time monitoring at the sites. However, based on

the current guidelines the data do not suggest an appreciable health risk. The diurnal variations were seen across the various sites in Adelaide and historical real time nephelometry data appears to parallel current PM_{2.5} measurements (South Australia Air Quality Branch 1992). The natural tendency is for air pollution to rise in the early morning (katabatic airflow), subside, and then reappear in late morning or afternoon due to the emergence of anabatic onshore airflow. Thus а distinguishing feature in the case of NW Adelaide may be industrial contributions superimposed on normal meteorological and traffic related trends.

From Table 2 it can be seen that the Dust Trak[™] PM_{2.5} values for Site 1 are approximately 30% higher than the corresponding TEOM values (10.1 vs 7.6 $\mu g/m^3$). It appears from the literature that the Dust Trak[™] oversamples by a factor of about 2 (Chang et al. 2001; Levy, Dumyahn & Spengler 2002). However, use of the environmental enclosure may result in some losses to the inlet of the Dust TrakTM impactor due to the presence of tubing, thus probably reducing the typical Dust TrakTM oversampling issue. Our reported values are essentially consistent with the other studies, which have found the Dust Trak[™] device to be reasonably precise compared with referenced methods (Chang et al. 2001; Levy, Dumyahn & Spengler 2002; Yanosky, Williams & MacIntosh 2002) and having the benefits of being relatively cheap and easy to set up. It is important though, that side-by-side sampling be conducted to accurately determine the extent of over sampling.

The monitoring of ultrafine particles was for very short periods of time (5 to 15 mins) due to the limitations of the monitoring equipment. Guidelines for airborne concentrations of ultrafine particles have not been developed. However, we were able to see short term fluctuations (<10sec) in recordings when buses were present in close (<100m) proximity, and higher averages in areas with greater traffic density and in built up areas (central business district).

The location of air sampling equipment in private residences has allowed for a more thorough investigation of localized air pollution. In addition we have investigated a range of ambient lung carcinogens, some of which are not routinely monitored (i.e. asbestos, crystalline silica). However, the generalisability of our investigations could be limited by short and long term weather patterns and the short duration of monitoring. This sampling campaign was conducted during a dry period (6 weeks of no rain), but strong winds were not observed, and there were no bushfires or dust storms. As such, resuspension of settled dust may not be as relevant as otherwise might be expected. However, if industrial production was a dominant contributor to ambient concentrations of lung carcinogens this would have been evident in our data. Somewhat higher values of PM2.5 were observed at the industrial sites (compared to control Site 5) but these were still well below NEPM guidelines. In general, these data are consistent with other measurements conducted by the SA EPA during the entire year (across multiple weather patterns) (Environment Protection Authority 2003).

In the context of current lung cancer cases in the NW Adelaide region, it is likely that the extent of industrial air pollution in the 1960s to '80s was greater than current values. There is good evidence of this from the documented changes in the industry arising from clean air regulations introduced in 1972 and Australian design rules in the 1970s (Department of Public Health 1976; South Australia Air Quality Branch 1992). Examples of industry change in this period include closure of an insulation factory, an oil-fired power station, and the changeover from coal and oil to natural gas for firing of industrial kilns and furnaces. Improvements in emission control during this 30-year period include taller chimneys and particle filtration. Thus it is possible that past environmental exposures may have contributed to existing incident cases of lung cancer.

The NW Adelaide Lung Cancer study investigated the high mortality rate of lung cancer in the region using epidemiological methods. This case control study did not find residential exposure to air pollution (as measured by proximity to industrial sources) to be associated with lung cancer, rather finding cigarette smoking to be the dominant factor (dose response relationship evident, odds ratio of 9.25 for 40 pack years with confidence interval 4.62-18.55, unpublished data).

Overall, our air sampling and epidemiological data suggest that environmental exposures to lung carcinogens probably represent a minor contribution to respiratory cancers in the NW of Adelaide. The health significance, including non-cancer endpoints, of observed short-term outdoor peaks remains unclear in our study and in the literature. Further research should explicitly investigate this phenomenon.

Acknowledgments

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REPORTS AND REVIEWS

A Re-evaluation of Mercury Related Health Risks Associated with Fish Consumption in the Swan River, WA

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During 2001 - 2002 a study was conducted by Oosthuizen and Boyce (2002) to determine levels of cadmium, lead and mercury in Swan River sediment and fish and to quantify the associated health risks for regular consumers of river fish. The risk assessment was conducted using World Health Organization (WHO) guidelines and US Environmental Protection Agency (EPA) prescribed reference doses. Since Food Standards Australia New Zealand (FSANZ) recently published their own exposure guidelines (FSANZ 2004) the 2002 evaluation was re-evaluated using the local guidelines. The 2002 evaluation suggested that adults could safely consume one fish meal per day, children one fish meal every three or four days and pregnant women one meal per month. In terms of the FSANZ standards there is no need to publish a fish consumption guideline as the risk for all populations associated with mercury in the Swan River fish is negligible.

Key words: Mercury; Fish; Health Risks

A study was conducted by Oosthuizen and Boyce during 2001 - 2002 to determine levels of selected heavy metals in Swan River (Perth) sediment and fish. These data were used to quantify health risks for regular consumers of river fish. This health risk assessment was conducted using World Health Organization (WHO) guidelines (WHO 2001) and the US Environmental Protection Agency (EPA) prescribed reference doses (US EPA 2002).

The results of long term epidemiological studies done in a number of locations such as the Seychelles Island and Faroe Islands were published during 2004, these and other related publications prompted the development of a fish consumption standard by Food Standards Australian New Zealand (FSANZ) in 2004. The FSANZ standard is reported as a PTEI or provisional tolerable weekly intake (FSANZ 2004) rather than a daily intake rate as with the US EPA reference dose (US EPA 2002).

It was therefore decided that a reevaluation of the Swan River data using the FSANZ standard would be appropriate.

Methods

Design

During October 2001 to June 2002 fish samples were collected to quantify levels of mercury in river fish and to evaluate the risk that regular fish consumers may be exposed to (Oosthuizen & Boyce 2002). These data were re-evaluated in terms of FSANZ (2004) guidelines.

Sampling methodology

Twenty three adult fish were purchased from regular anglers. Various species of edible fish of legal size were sampled (Oosthuizen & Boyce 2002).

Laboratory analysis of samples

All fish samples were analysed for total mercury content and it was assumed that 100% of the mercury was present in the form of methyl-mercury therefore depicting a worst case scenario. All samples were transported to the School of Natural Sciences analytical laboratory of Edith Cowan University, where they were analysed for mercury, cadmium and lead concentration.

Filleted fish (skin and scales removed) were air dried at 30°C for 48 hours. Approximately 0.5g (in duplicate) of the dried fish samples was weighed and transferred to separate 50 mL acid washed digestion tubes. Standards in the range 0.2 -5.0 ppb Hg were prepared in 5% nitric acid. Concentrated sulphuric acid (4 mL) and concentrated nitric acid (4 mL) were added to the tubes containing the standards and the samples. The tubes were heated to 120°C for 3 hours. Excess hydrogen peroxide (30%) was added upon cooling and the samples were then reheated for 30 minutes at 80°C. Upon cooling, 12% hydroxylamine hydrochloride (5 mL) was added and the solutions made up to volume (25.0 mL).

Mercury in the samples was determined by hydride generation with the aid of a Varian AA20 fitted with a vapour generator (VGA76 module) using stannous chloride (10%) as the reductant (Oosthuizen & Boyce 2002).

Analysis of data

Mean fish mercury levels were used to estimate the potentially absorbed dose for adult males, females and children. Dose was expressed in µg mercury per kg body weight. These data were compared to US EPA and FSANZ standards. Hazard quotients were calculated in order to estimate the risk to which people could potentially be subjected.

Results

Table 1 is a summary of results obtained from fish samples taken from the Swan River. A risk assessment using the US EPA RfD of 0.0001 mg/kg/day or 0.7 µg/kg/week was conducted by Oosthuizen and Boyce (2002). This assessment was based upon the assumption that one fish meal is consumed daily. The size of an average fish meal was estimated as being 200g. In this study the body weight of adult males was estimated as 75 kg, females 60 kg and children 19 kg. Table 1:Summary of mercury in fish data collected from the Swan River during the period 18 January - 14 February 2002

n	23 µg/g	
max	0.063 µg/g	
mean	0.029 µg/g	
standard deviation	0.0017 µg/g	
% exceeding 0.05 µg/g	9% (2)	

Note: 0.05 μ g/g is the limit imposed on the sampled species of fish by both FSANZ (2004) and WHO (2001). 16 samples were below the level of detection.

The hazard quotients in Table 2 indicate that the predicted dose for adult males would be within acceptable limits. Adult females could safely consume one fish meal per day and children one fish meal every three to four days.

Table 2: Mercury risk prediction for daily fish consumption (Swan River) based upon US EPA criteria, 2002

Body weight E	ody weight Estimated daily dose mg/kg based on a fish meal of 200g/day and an estimated mean fish mercury level of 0.029 μ g/g.		
Adult male (75kg)	0.00008 mg/kg	0.8	
Adult female (60kg	g) 0.0001 mg/kg	I	
Child (19 kg)	0.0003	3.1	

(Oosthuizen and Boyce 2002)

FSANZ (2004) published mercury in fish criteria levels for women of childbearing age as 1.6 µg/kg body weight/week and 3.3 µg/kg body weight/week for the general population over 2 years of age. The average weight of women of childbearing age was estimated to be 66 kg and the general population 67 kg. Children between 2 and 6 years of age were estimated as being 19 kg on average. A fish meal (single serving) was estimated as 150 g for adults and 75g for children under 6 (FSANZ 2004).

In terms of the revised risk assessment, based upon the FSANZ criteria, consuming fish from the Swan River on a daily basis presents no mercury related health risk. Furthermore maximum permissible weekly consumption (table 4) was calculated in accordance with the methodology reported by FSANZ (2004).

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Table 3: Mercury risk prediction for daily fish consumption (Swan River) based upon FSANZ (2004) criteria

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Body weight	Estimated weekly dose µg/kg based on a daily fish meal of 150g/day (adult) or 75g (child) and a mean fish mercury level of 0.029 µg/	Hazard quotient based on a PTWI of 1.6 for women of childbearing age, and 3.3 for the general g. population
Women of childbearing age (66kg)	0.46 µg/kg	0.3
General population (67kg)	0.45 µg/kg	0.1
Child 2- 6 (19 kg)	0.8	0.2

As can be seen from the results of the "maximum serves" calculation, it is highly unlikely that anybody would consume even close to a dangerous amount of fish from the Swan River. Hg residue. The PTWI is published as a wet weight fish Hg residue value. Since most mercury levels were found to be very low, 16 out of 23 below the level of detection, this would not affect the conclusions of the study.

There are anecdotal reports that suggest that some families living along the upper reaches of the Swan River rely quite heavily upon river fish as a source of food, therefore, it is important to ensure that the fish is safe to consume in large quantities (one meal per day).

The assessment conducted in 2002 reported that women of childbearing age could safely consume one fish meal per day and children one fish meal every three to four days. Pregnant women were advised not to consume more than one fish meal per month

Table 4: Mercury risk prediction for weekly fish consumption (Swan River) based upon the FSANZ PTWI (FSANZ 2004)

	Women of childbearing age	General population	Children (19 kg) PTWI
	(66 kg), PTWI 1.6 µg/kg/week	(67 kg),PTWI 3.3 µg/kg/week	3.3 µg/kg/week
Total permitted weekly intake	1.6 x 66 = 105.6 µg/week	3.3 x 67 = 221.1 µg/week	3.3 x 19 = 62.7 µg/week
Estimated non-fish intake 0.09% of total	0.94 µg/week	1.14µg/week	3.1 µg/week
Amount that can be safely consumed in fish	105.6 - 0.94 =	221.1 - 1.14 =	62.7 - 3.10 =
	104.66 µg/week	219.96 μg/week	59.6 μg/week
Maximum amount of	104.66 μg/week	219.96 μg/week	59.6 μ g/week
Swan River fish that can be	/0.029 μg/kg =	/0.029 μg/kg =	/0.029 μ g/kg =
consumed per week	3609g of fish per week =	7585g of fish per week =	2055g of fish per week =
per week	24 serves per week	50 serves	27 serves per week

Adapted from FSANZ 2004.

Discussion

A limitation of this study related to the experimental method needs to be considered. The laboratory method used to analyse the fish samples was based upon dry fish weight as a precautionary measure. However, in view of the revised assessment using the PTWI published by FSANZ, 2004 there is deemed to be no risk associated with regular consumption of fish taken from the Swan River.

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Safe Drinking Water: Lessons from Recent Outbreaks in Affluent Nations London

Steve E. Hrudey & Elizabeth J. Hrudey

IWA Publishing, 2004, 486pp. 1 84339 042 6 (hardback)

On 26 August 2004, the WHO/UNICEF Joint Monitoring Programme on Water Supply and Sanitation (JMP) issued its report, Meeting the Millennium Development Goals (MDG) Drinking Water and Sanitation Target: A Mid-term Assessment of Progress. Although the major emphasis of the report is on the 'silent emergency' of global populations who still do not have access to safe drinking water, it sounds a note of optimism on progress toward meeting the goal of reducing the number of people who do not have access to safe drinking water to 800 million, or 25% of world population, by 2015. The report notes that about half the population of the world now drinks piped water. This is perhaps the greatest public health success story of the 20th Century.

But as the Hrudeys' book amply informs us, we cannot assume, as we usually do, that if water comes from a utility through a pipe, it is invariably safe to drink. As we have seen emergent and re-emergent infectious disease of the air, blood, and mosquito-borne varieties, so too has the developed world experienced what to most will be a surprisingly large number of infectious disease outbreaks over the past 30 years in which the mode of transmission has been piped water, supplied by a utility and monitored and treated for our consumption. This book chronicles these water-borne outbreaks, tells us why they occurred, how they were dealt with, and develops several themes on how to prevent them.

The book provides a compilation of cautionary tales, based around the Hrudeys' theme of 'Converting Hindsight into Foresight'. The first part of the book is a primer on waterborne infectious disease pathogens, including a history of the dawning of appreciation of water as a vehicle of transmission of disease and development of the science of bacteriology and the technology of water treatment. Here are all the old familiar tales, told well and truly, setting the record straighter than it is usually presented in basic epidemiology or public health texts. The praise of famous men includes the stories of Snow, Koch, and even Fillipo Pacini, the *real* discoverer of cholera and its pathogenic waterborne potential, thirty years before Koch. Max von Pettenkopfer, Koch's nemesis in his own time, also gets a brief but rightful place in the chronicle.

A capsule summary of important waterborne pathogens follows. These summaries are not much longer than those in Benenson's, sorry, Chin's, no, make that Heymann's Control of Communicable Disease Manual (Heymann 2004), yet are eminently more focused on the type of information that will be useful to the scientist or engineer charged with keeping these wee beasties out of drinking water. Crossreferences from this section to the case studies that make up the balance of the book make this an effective tool for the reader wishing to understand the mode of action of these pathogens and their potential for mayhem on population health.

Chapter 3 is a brief but comprehensive technical review of water treatment and purification technologies, nicely augmented by illustrative figures and tables. The next chapter, which at 300 pages makes up well over half the book, is devoted to comprehensive analyses of every major waterborne disease outbreak in the developed world over the past 30 years. Here are to be found detailed analyses of the familiar stories, including the Milwaukee cryptosporidiosis outbreak of 1993, the

Sydney non-outbreak of crypto in 1998, and the Walkerton, Ontario outbreak (E coli 0157:H7 accompanied by Campylobacter jejuni) of 2000. The authors do an excellent job of presenting the cases to be made regarding the latter two. As Steve Hrudey spent a sabbatical with the Cooperative Research Centre for Water Quality Treatment in Adelaide in 1999 (Don Bursill, CEO of the CRC, provides the Foreword for the book), and served with the Research Advisory Panel to the Walkerton Inquiry, he knows whereof he speaks. The reports in this weighty chapter tell who did what when, what was done wrong, and what was done right. They are thoroughly referenced, painstakingly methodical, and extensively cross-referenced.

The Hrudeys' work strives to be exhaustive, and succeeds, perhaps excessively so. While this book should become the standard reference text in the field, no one is likely to read it cover to cover. The authors could have accomplished a thoroughly usable book that tells all the tales necessary for lessons to be learned by focusing on the biggest and/or most representative outbreaks of a type. In its attempts to be encyclopedic, it will come across as ever more dated when future readers note the absence of documentation on outbreaks that undoubtedly will occur.

And this is the real lesson of the book, that water supplied by a public utility in modern industrialised nations is still capable of causing human disease and death in the absence of due diligence. In the closing chapter, the Hrudeys make the case for how and where this due diligence needs to be applied. They advocate a systems thinking approach in order to nurture a prevention ethic and avoidance of complacency, emphasising accountability on the part of those officially charged with providing safe drinking water to the people's tap.

There is not much company on the shelf for this book. Hunter and Waite's *Drinking Water and Infectious Disease (2002)* would fit. As with all CRC publications, it is both comprehensive and pricey. So too would the (US) National Research Council's voluminous *Drinking Water and Health* (1980) series, if anyone still has their copies. Started in 1978, the series gushed to Volume 9 in 1988 before trickling to a halt. It is now long out of print, though the information it contained is not that dated.

The Hrudeys deserve praise for a comprehensive work that must have taken a very large effort on their parts. Their book should be read by every manager of any major water supply utility, and should have a place as at least a reference text in any undergraduate or postgraduate environmental health course.

More information on the publication is available from: www.iwapublishing.com

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Environmental Health Vol. 4 No. 3 2004 55

Controversies in Environmental Sociology

Rob White (Ed)

Cambridge University Press, 2004, 300pp. ISBN 0 521 60102 9, \$49.95 (paperback)

Controversies in Environmental Sociology will be of considerable value to environmental health practice, in all the diverse areas of that broad field. Many environmental health publications cover the standard biophysical issues of air pollution or food safety and so on. This book offers insights into the social contexts in which these issues are set, and in which environmental health practitioners have to operate. It will be useful in all aspects of environmental health program design and management, giving a deeper understanding of the many social issues that arise in the complex field.

The insights provided by the book are timely. Environmental Health as a profession has only recently come to accept that environmental health practitioners have a responsibility for reducing the social causes and effects of environmental degradation, as well as being experts in the biophysical conditions. The book's division into three sections, Social Perspectives, Social Trends and Social Issues, is a practical division in terms of the social context of environmental health practice. Each section reflects the plain language style that makes the book accessible throughout. The book is an outstanding success in presenting important topics that are only too often buried in dense sociological jargon, in most cases in clear and readily understood terms.

The core theme of 'Controversies' recognises that responses to environmental health issues will always be debated among the social groups that they affect. The material within each of the three sections is varied, and sometimes difficult to connect. The connecting thread suggested for responding to a controversy is the three steps of 'see, judge, and act'. This is a good guide to practical use of the material, but is not necessarily carried through in all the chapters.

The first section, Social Perspectives,

addresses the environmental implications of religion, social theory, ecofeminism, ecocentrism and the risk society in turn. These are all keys to understanding environmental health decisions, but it would have been good to have them connected in some way. The examples chosen represent frameworks that everyone brings with them to any environmental controversy. We will all, including the environmental health practitioner themselves, bring our belief system, ideas about the world, social role (in this case gender) and our attitude to risk. The influence of religion is traced through the positions held on environmental themes by the major formal belief systems, Christian, Buddhist, Moslem and New Age, and the modern belief in the market economy. There is a considerable difference in approaching the environment as under human control, a segment of an interrelated world outside human control, a gift from God, and a threat to a market resource. Yet all these positions exist in most communities.

The four chapters in the next section, on social theory, discuss the ongoing debates on the nature of 'social nature', that is, the human allocation of meaning to the natural world. The strength of this section is that it does not divide the debates into empty oppositions of either or, but explores the interactions between the different positions. The first three debates selected for discussion are the social versus the scientific construction of the environment, feminism as an issue of social justice against ecofeminism as the women's identification with nature, and animal versus human rights. The fourth is perhaps the most important chapter of the book from the perspective of environmental health practice. It explores the important work of Ulrich Beck who writes that western society has become a society driven by a concern for risk, rather than towards social or religious ideals. He suggests that we have become a risk-averse, rather than an adventurous people.

Beck identifies the unique aspects of risk in our time as follows: we now accept that risks are created by the whole of society, not a malignant god or an unknown agency; risks have the capacity to be global and so are a global responsibility affecting everyone on Earth; and risks such as radiation are no longer immediately apparent to the senses in the manner of chimney smokestacks and so need constant external monitoring. His conclusion is that the management of risk is both everybody's problem and a matter of professional expertise. The resulting debate is about who does the management, government controls delegated by society, or the network of people who make up the society. As with the other debates, the author of this chapter looks at the interaction between the poles of the debate. He identifies as a source of the confusion apparent in response to risk today, the changing pattern of social approaches to risk in general, as well as a range of expert opinion on how risk is to be managed. This chapter is somewhat 'hard going', but its material lies at the core of environmental health practice today.

The chapters in the second section of Controversies in Environmental Sociology examine the consequences for environmental health of social issues which are only too apparent: population increase and its dispersal; growing inequity between human and environmental needs; the dilemmas in using sustainable and unsustainable technologies; the diverse pressures to bridge local and global scales; citizens' growing rights and responsibilities; and the emergence of environmental advocacies. The third section explores a range of social influences on biophysical environmental health issues, by the media, agriculture, health impact assessment, social impact assessment and the law, respectively.

While this menu of social trends and their related issues is a familiar one, the value of this book lies in its orientation towards matters of central concern in environmental health. I thoroughly recommend *Controversies in Environmental Sociology* to the designers of any formal or informal educational program in Environmental Health. I also recommend it to inquiring individual practitioners who are wondering why the field has become so complex and unpredictable. Sections of this book may be hard going at times, but well worth the effort to discover that it isn't our profession that is confused, it is the state of the world.

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An Introduction to the Environmental Physics of Soil, Water and Watersheds

Calvin Rose

Cambridge University Press, 2004, 453pp. ISBN 0 521 53679 0, \$99.00 (paperback)

This is a very readable book which deals with the principles of environmental physics underpinning the behaviour of water during its passage through the watershed, as an integral part of the hydrologic cycle.

While it does not attempt to give advice on management strategy per se, the reader is provided with sufficient perspective on physical processes to enable the application of what is said to the sustainable management of catchments. Given the climatic mood-swings which seem to have become the norm for our Region, and the resultant need to carry out catchment protection with all the tools available, a book which gives an understanding as to why and how things happen, and when they are likely to occur, is a very useful acquisition.

A strength of the book is the author's presentation of the physics in digestible portions, well-linked through relevant examples to various aspects of watersheds. The examples are based on highly practical situations which are likely to be encountered by the environmental manager, scientist or engineer almost on a daily basis. Even in the parts where the author delves into deeper physical waters it is reassuring to note that the common themes of the hydrologic cycle and the catchment are never far away and the reader re-emerges from the quick dip into more sophisticated physics, to a familiar part of the landscape.

While a knowledge of integral calculus or mathematical modelling are normally required in texts dealing with water or soil physics, the author has found clever ways of approaching the issues from a different angle, and the level of skill required to read the text seldom demands more than a basic knowledge of arithmetic or algebra.

To ensure that the knowledge gained can be applied in a functional way the author has provided exercises at the end of each chapter with fully-worked solutions available through a Cambridge publications site on the world-wide web. The book thus offers considerably more value than its 400odd pages suggest.

The book is divided into clearly defined sections, the reader initially being introduced to the Earth as a solar-energy driven mill, in which aquatic and other forces grind down primordial rock to produce soil as the base material for the attachment of plant and animal life. The reader is taken through all processes in a series of well defined steps in which physics is clearly linked to relevant environmental events.

Areas covered include the mechanics of soil integrity and its implication in land subsidence, forces behind water capillarity and their role in maintaining the shape and location of ground water, the dynamics of fluids and the moulding of landscape through erosion, the nature of wave motion and its role in estuarine structuring, the forces behind evapotranspiration and their place in water-balance accounting, the nature of heat flux in maintaining soil temperature, the influence of particle physics in subterranean retention and flow of water, physical determinants of rainfall patterns across catchments, friction considerations in the overland movement of water, forces involved in sediment transport and deposition, physical equilibria in maintaining groundwater level, the role of charge in pollution mobility and soil salinity, and plume physics relating to subsoil water flow in agricultural settings.

This is clearly a book written by an environmentalist with an understanding of the importance of physics and not by a physicist looking for a convenient place to hang a set of physical theories. The book is recommended for all readers with an interest or role in catchment management, or in the management of the aquatic and terrestrial environments in general. It allows the reader to come to grips in a painless way with physical theory underlying the dynamic nature of the catchment landscape.

Many of us approach physics with a trepidation born of having been taught the subject at some time or other as being a mere extension of advanced mathematics. This book provides an opportunity for shedding the aversion and re-engaging with physics as a useful, applied science with capacity to open new areas of perception and understanding for those engaged in sustainable watershed management.

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Guidance on Use of Rainwater Tanks

enHealth Council

Department of Health and Ageing, Australian Government, May 2004 (paperback)

Rainwater tanks have been a highly successful source of harvesting water supplies for isolated properties, small communities and in rural areas of Australia for over 200 years. Increased interest in rainwater tanks has arisen because of widespread drought conditions, predictions of worldwide shortages of fresh water as populations continue to grow, and increased water restrictions. The number of agencies offering incentives for installation of domestic tanks has also increased.

Used correctly, rainwater tanks are an effective way to take the pressure off our limited water resources, and at the same time, help manage stormwater run-off. In general, rainwater tanks can provide a valuable water source for flushing toilets, use in washing machines, watering gardens, and for washing cars.

There are many factors that need to be considered to ensure that the rainwater you harvest can be used to its full potential. Atmospheric pollution, animal droppings, insects, roofing materials and paints, soil and vegetative matter have the potential to affect water quality from both rural and urban roof sources.

In some jurisdictions, the compulsory installation of rainwater tanks, often in conjunction with energy efficient design ratings for new developments, is required even when suitable town water supply is provided. Therefore, planning authorities might be responsible for developing appropriate specifications, in order to achieve the effective harvesting of water and to provide assurance that installations meet reasonable health and safety standards.

As a result of this increased interest, enHealth Council, led by Dr David Cunliffe of the South Australian Department of Health, has revised its Guidance on Use of Rainwater Tanks, originally produced in 1998 by its precursor group, the National Environmental Health Forum. This 2nd edition provides a valuable resource to Environmental Health Officers, Building Surveyors, and Sustainability Planners in both the assessment of potential health risks and contamination sources. the development of preventative maintenance and health risk management procedures, and tools to assist in tank specification and installation so that comprehensive advice can be prepared for dissemination to the public.

Guidance on Use of Rainwater Tanks is a comprehensive document of 72 pages covering a multitude of issues, including suitability for drinking and mosquito control. A new section on "Preventative measures and corrective action" is founded very much on a risk-based approach, in line with the drinking water guidelines (in press), and the national water recycling guidelines under development. The framework identifies both health risks and aesthetic quality risks and provides corresponding preventative measures, monitoring and corrective actions in an easy to use format.

Tables for the calculation of water harvest based on the average annual rainfall, and quantity of water required, can provide an estimate of the roof and tank sizes required to ensure a reasonable margin of supply security. For example, based on a moderate 600mm of annual rainfall, a family of four in West metropolitan Melbourne currently using 400 litres per day of water, living in a house with a roof size of 150 square metres (16.5 sq), would require a tank size of 25,000L to guarantee a 90% chance that 60% of their daily water needs would be supplied by a rainwater tank. As many households currently use 800 to 1000 litres per day in summer, it is clear that it is unlikely that complete water self-sufficiency will only be attained by the most fastidious fraction of the community.

Guidance on Use of Rainwater Tanks is published by the enHealth Council 2004 (ISBN 0 642 82443 6). Hard copies of enHealth Council publications can be ordered from

Email:nmm@nationalmailing.com.au Tel:02 6269 1000 Fax: 02 6260 2770. Reports and **Reviews**

The document can also be downloaded from either the enHealth Council website or the Department of Health & Ageing website.

http://enhealth.nphp.gov.au/council/pubs/ documents/rainwater_tanks.pdf

http://www.health.gov.au/pubhlth/publica t/document/metadata/env_rainwater.htm

A model brochure suitable for public information is also provided and can also be distributed without alteration and is available at:

www.health.gov.au/pubhlth/publicat/docu ment/env_rainwater_brochure.pdf

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Public Health Law and Regulation

Christopher Reynolds with Genevieve Howse

The Federation Press, Sydney, 2004, 336 pp. ISBN 1 86287 512 X, \$55.00 (paperback)

For most people the idea of reading a legal textbook rates 'up there' with watching grass grow. However, for those of us involved in environmental health, a new textbook on public health law should be of particular interest, as a key element that defines the role of an environmental health officer is the duty to administer various complex pieces of public health legislation.

As legal textbooks go, this new textbook on *Public Health Law and Regulation* is relatively easy to read. The authors have been mindful of their audience and state that the aim of the text is to 'impart some of the useful information that public health students and practitioners who seek to understand the legal system would want to know'. It provides a comprehensive overview of past, present and future issues for Australian public health law, and takes the view that legislation and legal processes powerfully influence public health practice, and in general this influence is positive.

The text consists of the following chapters: (1) public health law - scope, structure and issues, an introduction; (2) tools for regulation - parliament, courts and administration; (3) the framework of public health law; (4) ideas about regulation; (5) a risk-based approach to regulation; (6) environmental health and the health of the environment - the environment protection and planning systems; (7) food law; (8) communicable disease; and (9) drugs.

In particular, Chapter 1 discusses public health and the law, answers the question of 'what is public health law?', introduces some key issues in public health law, discusses health and legal issues for Indigenous Australians, and looks at public health and human rights. Chapter 2 goes on to discuss issues such as legal frameworks, statutory interpretation, the court system and enforcement of legislation. Chapter 3 then discusses the public health roles and laws of the various levels of government in Australia, including new issues for and modern approaches to public health legislation. Chapter 4 looks at how public health influences the law and discusses other issues such as command and control structures, regulation versus self regulation, consumer choice, taxation and litigation. Chapter 5 completes the first section on general issues by discussing the increasing use of risk-based approaches in public health law. The remaining chapters discuss in detail the following specific types of public health health law: environmental and environmental protection, food safety and standards, communicable disease control, and the regulation of drugs and poisons.

To help illustrate many of the legal issues and concepts, a large number of excellent examples and case studies are provided throughout the text. Many of these examples are related to environmental health issues, for example, tobacco control, food safety and HACCP, environmental protection standards and ISO 14000, planning laws, and legal issues for the investigation of a Legionella outbreak. As each of the chapters present various perspectives on public health law, many of

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the topics illustrated through the examples are discussed from a range of perspectives throughout the text. This technique is particularly insightful and is most beneficial for the reader.

Overall, this text continues and expands on the excellent previous publications by the same authors (eg. *Public Health Law in Australia*), and it updates and clarifies many of the complex legal issues that we currently face. This text presents what can be very detailed information in a very readable but thoughtful manner, and provides much food-for-thought in regard to future public health issues that will need to be addressed through legislation. As such, this text will be of great benefit to students and practitioners, and provides policy-makers with a compelling argument for the positive role that can be played by public health law.

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Australia's Health 2004

Australian Institute of Health and Welfare

Australian Institute of Health and Ageing, Australian Government, 2004, 528 pp. ISBN 1 74024 382 X, ISSN 1032-6138 \$55.00 (paperback)

Australia's Health 2004 is the ninth edition of the AIHW's biennial health report to the nation. This report can be considered as providing a 'report card' on the health of Australians and on health services. For 2004, the report gives Australia a 'grade' of 'very good' to 'very good plus'. This is due to continuing improvements in life expectancy, falls in the prevalence of many diseases and health conditions, and improved survival from these conditions. In addition, health services continue to be of high quality and are widely available and accessible. However, the report identifies some major areas for improvement, such as obesity and physical activity, and highlights the fact that the health of particular sections of the community, notably Aboriginal and Torres Strait Islander people, has not improved and these people groups remain significantly disadvantaged.

This report presents a compilation of key health statistics and analysis that is based primarily on work by the AIHW, with many of the topics addressed in more detail in other AIHW publications. For some topics, data from other agencies have also been used (eg. National Notifiable Diseases Surveillance System for Communicable Disease Data).

In general, Australia compares well with other member countries of the OECD, with rankings across most categories being in the top or middle one-third. Some of the interesting data include:

Life Expectancy:

- Average life expectancy (at birth in 2002) was 82.6 years for females and 77.4 years for males.
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- The life expectancy of Australians is among the best in the world, ranking 4th for males and females in 2002.
- Life expectancy for Aboriginal and Torres Strait Islander peoples is 20 years lower than for other Australians.

Ill Health:

- Cardiovascular disease is the leading cause of death for both males and females, despite a substantial drop in death rates since the late 1960s.
- Around one in five Australian's had a cardiovascular problem in 2001 and about 1.1 million have a disability as a result.
- Cancer ranks second as an overall cause of death, but overall death rates fell between 1992 and 2002.
- Lung cancer was responsible for the most cancer deaths in 2002.
- Injury death rates have fallen dramatically over the past few decades, however, injury remains the leading cause of death for people under the age of 45.
- Diabetes prevalence has more than doubled in the last two decades, and is estimated to impact around 1 million adults.
- Asthma affects 14 to 16% of children and 10 to 12% of adults.

Health Risks:

- One in two adults in 1999-2000 had blood cholesterol levels considered to be high.
- Even though the prevalence of high blood pressure (among 24 to 65 year

olds) has more than halved in the last two decades, it still affects over 3 million adults.

- Obesity prevalence among adults has doubled over the last two decades, and now one in five are obese.
- 20% of adults smoked daily in 2001 compared to 70% of men and 30% of women in the 1950s.
- In 2000, more than one in two adults did not undertake leisure-time physical activity at levels recommended for health benefits.
- Between 1997 and mid 2002, there was a marked improvement in child vaccination rates, but coverage was a little below the targets for some specific age-groups.

Health Resources:

- Health expenditure represented 9.3% of GDP in 2001-02, compared with 8.7% in 1998-99 and 8.1% in the early 1990s.
- \$66.6 billion was spent on health services in 2001-02, with the highest health expenditure for cardio-vascular diseases (\$5.4 billon).
- Spending on health by members of the public (ie. out of their own pocket) has grown at a faster rate (7.7%) than funding by governments (5.7%) over the period 1997-98 to 2001-02.
- The rate of health expenditure for people over 65 years was three times that for the whole population (in 2000-01).
- Expenditure on pharmaceuticals grew, in real terms, at an average of 11.9% per year between 1997-98 and 2001-02.

Health Services:

• There were 6.4 million admissions to public and private hospitals in 2001-02.

- There has been a shift in hospital use from public to the private sector, eg. from 1997-98 to 2001-02 the proportion of hospital admissions in public acute hospitals fell from 67.4% to 61.7%; and for the same period, admission rates for private hospitals rose by 35.7% whereas admission rates only rose by 5.2% in public hospitals.
- There was a clear trend towards more day-only hospital admissions, with the rate going from 46.3% in 1997-98 to 52.3% in 2001-02.

As can be seen from the above data, the report provides a comprehensive overview of the health status of Australians as assessed by a large range of measures. As this sort of data can be daunting to interpret, the report uses a defined 'conceptual framework' through which to present the information. The main elements of this framework are determinants, health and wellbeing, interventions, and resources and systems. These elements are weaved through the following eight chapters that are supported by a series of appendices: introduction, health of Australians (this is the largest determinants chapter), of health, population health, health resources, health services, national health information and its development, and health of older Australians.

From an environmental health perspective, the sections on communicable diseases (section 2.7), health behaviours (section 3.3, eg. dietary behaviour, physical tobacco smoking, alcohol activity, consumption, and vaccination status), environmental factors (section 3.5), and Aboriginal and Torres Strait Islander peoples (section 4.4) are particularly interesting.

Overall, this report should be viewed as the premier source of summary information on national patterns of health and illness, determinants of health, the supply and use of health services, and expenditure on

Reports and **Reviews**

health services. When used in conjunction with local data (eg. state-specific health determinants data such as Health Determinants Queensland 2004), these reports provide critical information for planning of many public health services and interventions. The AIHW is to be congratulated for producing such a readable and rich source of information, and it more than adequately showcases the AIHW's mission of 'better health and wellbeing for Australians through better health and welfare statistics and information'.

Note: A number of interactive databases and a full range of AIHW publications are available at no cost through their website: <www.aihw.gov.au>

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Vaccines: Preventing Disease and Protecting Health

Ciro A. de Quadros (Ed)

Pan American Health Organization, Washington, DC, 2004, 398 pp. ISBN 92 75 31596 5, 165 \$40 (paperback)

The dramatic decline in communicable diseases and the major role of vaccines in this endeavour, ranks as one of the greatest public health achievements of the 20th century. However, with a number of infectious diseases recently emerging or reemerging, the world has been forced to reevaluate the current prevention efforts for these diseases. Given this background, the Pan American Health Organization held an international conference on vaccines in November 2002 with the purpose of reviewing the current 'state of the art' knowledge and discussing future issues for vaccine development and immunisation. The text, Vaccines: Preventing Disease and Protecting Health, was born out of this conference and represents a collection of papers that contain the most up-to-date information available by the world's foremost experts in the field.

The 46 chapters discuss a range of topics including: a reflection of the progress in communicable disease control made achievable through the use of vaccines; detailed descriptions of a large range of diseases and the current role played by vaccines in their control; discussions of cutting edge research in the field; future issues for the control of specific diseases through the use of vaccines; challenges and opportunities for vaccine development; vaccines and terrorism; regulatory and safety issues; and the role of vaccines in public health programs including discussions on reforms for the current funding and delivery systems.

This text provides extensive and detailed information on a large range of vaccinerelated topics, and it does so in a very readable manner. To enhance the written content, there are also a large number of well-constructed figures, graphs and tables throughout the text that provide excellent illustrations. The list of authors is impressive and represents the world leaders in this field.

Overall, this text represents an exceptional collection of papers on the role of, and future challenges for, vaccines in preventing disease and protecting public health. It is highly recommended reading for all public health practitioners who are involved in communicable disease control activities, and the PAHO should be congratulated for providing practitioners with such a valuable resource.

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