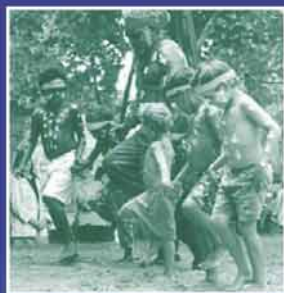


Volume 5  
Issue 4  
2005

# Environmental Health

*The Journal of the Australian Institute of Environmental Health*



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



## Environmental Health

The Journal of the Australian Institute of Environmental Health



ABN 58 000 031 998

### Advisory Board

Ms Jan Bowman, Department of Human Services, Victoria  
Professor Valerie A. Brown AO, University of Western Sydney  
and School of Resources, Environment and Society,  
Australian National University  
Associate Professor Nancy Cromar, Flinders University  
Mr Waikay Lau, Chief Executive Officer, Australian Institute of  
Environmental Health  
Mr Bruce Morton, Vice President, AIEH  
Mr Jim Smith, Australian Institute of Environmental Health  
Dr Ron Pickett, Curtin University  
Dr Thomas Tenkate, Queensland University of Technology

### Editorial Team

Mr Jim Smith, Editor  
Associate Professor Heather Gardner, Associate Editor  
Ms Jaclyn Huntley, Assistant Editor

### Editorial Committee

Dr Ross Bailie, Menzies School of Health Research  
Dr Dean Bertolatti, Curtin University of Technology  
Mr Hudson H. Birden, Northern Rivers University Department of Rural Health,  
Faculty of Medicine, University of Sydney  
Dr Helen A. Cameron, Department of Health and Ageing, Canberra  
Mr Peter Davey, Griffith University  
Dr Chris Derry, University of Western Sydney  
Ms Louise Dunn, Swinburne University  
Professor Howard Fallowfield, Flinders University  
Mr Ian Foulkes, The Chartered Institute of Environmental Health, London  
Mr Stuart Heggie, Tropical Public Health Unit, Cairns  
Ms Jane Heyworth, University of Western Australia  
Professor Steve Hrudey, University of Alberta, Canada  
Professor Michael Jackson, University of Strathclyde, Scotland  
Mr Ross Jackson, Maddocks, Melbourne  
Mr Steve Jeffes, TAFE Tasmania  
Mr George Kupfer, Underwriters Laboratories Inc, Illinois, USA  
Professor Vivian Lin, La Trobe University  
Dr Bruce Macler, U.S. Environment Protection Agency  
Dr Anne Neller, University of the Sunshine Coast  
Professor Peter Newman, Murdoch University  
Dr Eric Noji, National Center for Infectious Diseases, Atlanta, USA  
Dr Dino Pisaniello, Adelaide University  
Dr Scott Ritchie, Tropical Public Health Unit, Cairns  
Professor Rod Simpson, University of the Sunshine Coast  
Dr Peter Stephenson, Batchelor Institute, NT  
Dr Melissa Stoneham, Queensland University of Technology  
Ms Isobel Stout, Christchurch City Council, New Zealand  
Ms Glenda Verrinder, La Trobe University Bendigo  
Dr James M. Wilson, ISIS Center, Georgetown University Medical Center,  
Washington, USA  
Dr Amanda E. Young, Center for Disability Research, Massachusetts, USA

Environmental Health © 2005

## Environmental Health

The Journal of the Australian Institute of Environmental Health

ISSN 1444-5212 (Print), ISSN 1832-3367 (Online)

### *Linking the science and practice of environmental health*

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

Copyright is reserved and requests for permission to reproduce all or any part of the material appearing in *Environmental Health* must be made in writing to the Editor.

All opinions expressed in the journal are those of the authors. The Editor, Advisory Board, Editorial Committee and the publishers do not hold themselves responsible for statements by contributors.

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

Correspondence to: Jim Smith, Editor, P O Box 225 Kew, Victoria, 3101, Australia.

**Cover Design by:** Motiv Design, Stepney, South Australia

**Design & typeset by:** Mac-Nificent, Northcote, Victoria



**Environmental Health © 2005**

ISSN 1444-5212 (Print), ISSN 1832-3367 (Online)

---

## Environmental Health

---

The Journal of the Australian Institute of Environmental Health

ISSN 1444-5212 (Print), ISSN 1832-3367 (Online)

*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, PANDORA 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

### Correspondence:

Jim Smith  
Editor, *Environmental Health*  
P O Box 225  
Kew, Victoria, 3101  
AUSTRALIA

### Editorial Team:

Heather Gardner  
Email: [gardner@minerva.com.au](mailto:gardner@minerva.com.au)  
  
Jaclyn Huntley  
Email: [Jaclyn@infocusmg.com.au](mailto:Jaclyn@infocusmg.com.au)

Telephone: 61 3 9855 2444  
Fax: 61 3 9855 2442  
Email: [jim@infocusmg.com.au](mailto:jim@infocusmg.com.au)  
Website: [www.aieh.org.au](http://www.aieh.org.au)

**For subscription and memberships details visit our website: [www.aieh.org.au](http://www.aieh.org.au)**

## Call for Papers

### The Journal is seeking papers for publication.

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

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

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

#### Aims

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

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

#### GUEST EDITORIALS

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

#### RESEARCH AND THEORY

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

#### PRACTICE, POLICY AND LAW

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

#### REPORTS AND REVIEWS

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

#### Correspondence:

Jim Smith

Editor, *Environmental Health*

PO Box 225 Kew, Victoria, 3101, AUSTRALIA

Guidelines for Authors can be obtained from the Editor

Telephone: 61 3 9855 2444

Fax: 61 3 9855 2442

Email: jim@infocusmg.com.au

---

## CONTENTS ENVIRONMENTAL HEALTH, VOLUME FIVE, NUMBER FOUR, 2005

### EDITORIALS

<i>Jim Smith</i> .....	11
<b>The Development of the Tasmanian Bachelor of Environmental Health</b>	
<i>Ron Fry</i> .....	13

### ARTICLES

#### RESEARCH AND THEORY

<b>Respiratory Health of Underground Coal Miners in Newcastle</b>	
<i>Zhen Hai Gu, He Wang, Paul Thomas, Richard Henry and Rui Wang</i> .....	14
<b>Intervention of Ticlopidine on Silica-Induced Inflammatory Reaction and Apoptosis</b>	
<i>He Wang and James Leigh</i> .....	23

#### PRACTICE, POLICY AND LAW

<b>Rural Health Systems Change</b>	
<i>Peter Harvey</i> .....	33
<b>Environmental Health Impact Assessment (EHIA) in the Western Pacific Region</b>	
<i>J. T. Spickett and J. Graham</i> .....	42
<b>Faecal Source Tracking in Surface Waters: A Review of Faecal Indicator Microorganisms and Current Methods</b>	
<i>W. Ahmed, R. Neller and M. Katouli</i> .....	51

#### REPORTS AND REVIEWS

<b>Occupational Health and Safety: International Influences and New Epidemics</b>	
<i>Reviewed by Jeffery T. Spickett</i> .....	69
<b>Environmental Health: From Global to Local</b>	
<i>Reviewed by Thomas Tenkate</i> .....	71

■ SUBSCRIPTION FORM

■ GUIDELINES FOR CONTRIBUTORS

---

## CONTENTS ENVIRONMENTAL HEALTH, VOLUME FIVE, NUMBER ONE, 2005

### EDITORIAL

<i>Jim Smith</i> .....	9
------------------------	---

### ARTICLES

#### RESEARCH AND THEORY

##### **A Study of Associations between Cigarette Smoking, Nearby Industry and High Lung Cancer Mortality in North West Adelaide**

<i>Brian J. Smith, Melissa J. Whitrow, Louis S. Pilotto, Dino L. Pisaniello, Adrian Esterman and Pam Selim</i> .....	11
--	----

##### **Air Pollution and Cardiopulmonary Diseases in Australia: A Review of Epidemiological Evidence**

<i>Jessica Howie, Shilu Tong, Ken Verrall, Rod Gerber and Rodney Wolff</i> .....	23
--	----

##### **The Management of Crowds and Other Risks at Outdoor Music Festivals: A Review of the Literature**

<i>Cameron Earl, Elizabeth Parker and Mike Capra</i> .....	37
--	----

#### PRACTICE, POLICY AND LAW

##### **Planning and Management for Public Health Impacts at Outdoor Music Festivals: An International Study**

<i>Cameron Earl, Elizabeth Parker and Mike Capra</i> .....	50
--	----

##### **A Country's Hidden and Untapped Resource: Exploring Attitudes, Beliefs, Perceptions and Knowledge of Hygiene in Kandahar, Afghanistan**

<i>Kelly Monaghan</i> .....	62
-----------------------------	----

##### **Food Security and Permanent Residents of Caravan Parks**

<i>Andrea Bryce, Catherine Donoghue, Beverley Allen and Suzanne Stokes</i> .....	73
--	----

#### REPORTS AND REVIEWS

##### **Health Team "Foxtrot" Support to Banda Aceh**

<i>Brad Adams</i> .....	84
-------------------------	----

##### **Digital Detection of Wastewater in Stormwater**

<i>Callum Morrison, Skye Scott and Jim Smith</i> .....	88
--	----

##### **Sustainability and Health: Supporting Global Ecological Integrity in Public Health**

<i>Reviewed by Vivian Lin</i> .....	95
-------------------------------------	----

---

## CONTENTS ENVIRONMENTAL HEALTH, VOLUME FIVE, NUMBER TWO, 2005

### EDITORIAL

<i>Jim Smith</i> .....	9
------------------------	---

### LETTER TO THE EDITOR

<i>Senator the Hon. Robert Hill</i> .....	11
---	----

### ARTICLES

#### RESEARCH AND THEORY

<b>Listeriosis Awareness among Pregnant Women in the Loddon-Mallee Region of Victoria</b> <i>Paul Jackson, Maria Sheldon, and Dianne Katscherian</i> .....	15
---	----

<b>Comparison of the Biologically Effective UV in the Shade for Three Action Spectra</b> <i>D.J. Turnbull and A.V. Parisi</i> .....	26
--	----

<b>Improving the Feedback of Housing Information to Indigenous Communities</b> <i>K.I. Wayte, R.S. Ballie, and P. Stephenson</i> .....	36
---	----

#### PRACTICE, POLICY AND LAW

<b>A Study of Temperatures Achieved during Underground Cooking of Pork</b> <i>David Sellars and Stuart Heggie</i> .....	48
--	----

<b>Mobile Phone Use among Full time Students of Edith Cowan University: A Pilot Study</b> <i>Jacques D. Oosthuizen</i> .....	55
---	----

<b>The Bellevue (WA) Chemical Fire 2001: An Environmental Health Management Review</b> <i>Annemarie J. B. M. De Vos and Jeffery T. Spickett</i> .....	62
--	----

#### REPORTS AND REVIEWS

<b>The Public Health Response to a Case of Hepatitis A in a Food Handler at a Large Sydney Community Club</b> <i>Deshanie Sathanandan, Graham Burgess, Leena Gupta, Jenny Lane, and Patrick Maywood</i> .....	77
--	----

<b>Communicable Disease Epidemiology and Control: A Global Perspective, 2nd Edition</b> <i>Roger Webber - review by Thomas Tenkate</i> .....	81
---	----

<b>A Small Dose of Toxicology: The Health Effects of Common Chemicals</b> <i>Steven G. Gilbert - review by Thomas Tenkate</i> .....	82
--	----

---

## CONTENTS *ENVIRONMENTAL HEALTH*, VOLUME FIVE, NUMBER THREE, 2005

### EDITORIALS

<i>Jim Smith</i> .....	11
<b>Environmental Health University Education in Australia: Market Forces, Threats and Opportunities</b>	
<i>Thomas Tenkate</i> .....	13
<b>Climate Change and Population Health in Australia: What Do We Know and What Can We Do?</b>	
<i>Peng Bi</i> .....	19

### ARTICLES

#### RESEARCH AND THEORY

<b>Use of the SF36 Health Survey to Compare the Health and Wellbeing of Residents Living along Roads with Different Levels of Commercial and Non-commercial Traffic</b>	
<i>Craig Hansen and Anne Neller</i> .....	22
<b>Dust and Biological Monitoring During a Vanadium Catalyst Change-out: Is it the Dust Levels or the Hand to Mouth Contact?</b>	
<i>Martyn Cross</i> .....	32
<b>Interpretation of Drinking Water Monitoring Data for Environmental Health Professionals</b>	
<i>Daniel Jalba, Samantha Rizak and Steve E. Hrudey</i> .....	40
<b>Study of heavy metals in the Shar Chi River (Urmia Iran)</b>	
<i>H. Nanbakhsh, A. Hamzehzadeh &amp; L. Gollzadeh</i> .....	50

#### PRACTICE, POLICY AND LAW

<b>Dust, Distance and Discussion: Fieldwork Experiences from the Housing Improvement and Child Health Study</b>	
<i>Emma Kowal, Phil Donohoe, Katrina Loneragan, Harold Ulamari and Ross Baillie</i> .....	59
<b>Public Opinion of a Proposed Wind Farm Situated Close to a Populated Area in New Zealand: Results from a Cross-sectional Study</b>	
<i>Charmaine A. Watts, Philip J. Schluter and Roger Whiting</i> .....	73

The year 2006 promises to be exciting, with the International Federation of Environmental Health (IFEH) World Congress in Ireland rapidly approaching. It was encouraging to hear that the Australian Institute of Environmental Health (AIEH) has been able to organise cheaper individual airfares for those of you lucky enough to be travelling to Ireland in June. Make sure you pay your delegate registration fees by the 15th of April to avoid the late fee charges.

While you are attending the Conference, please report back to the Journal on what you have learnt or experienced to keep the rest of us up to date with what has been covered. I'm sure many of our readers would be interested in your commentaries on interesting seminars, workshops and people met during the conference. Such experiences could be emailed as a Letter to the Editor, or even as a report if you have time to expand your contribution. Please email your submissions to: [journal@aieh.org.au](mailto:journal@aieh.org.au).

This issue of *Environmental Health* covers a variety of research and environmental health issues from Queensland to Tasmania, and from Newcastle to the Western Pacific Region. From the South, we examine rural health systems changes with Peter Harvey. With over 20 years working in the rural health field, Harvey uses his experience and research to explore health reform in rural communities and proposes several strategies to improve the health care system in rural South Australia.

If we travel further South we reach Tasmania, where Ron Fry introduces us to the development of the Tasmanian Bachelor of Environmental Health. Those of you following recent Guest Editorials on education and environmental health practitioners will be excited with the

progress in the field of environmental health education.

We explore the respiratory health of underground coal miners in Newcastle, Northern New South Wales. Zhen Hai Gu et al. research the associations between respiratory impairment and underground coal mining, discovering a protective effect from smoking cessation and calling for strengthened anti-smoking campaigns in these mines.

Wang and Leigh also research industrial hygiene and respiratory health hazards through their study of the intervention of ticlopidine on silica-induced inflammatory reaction and apoptosis.

North from Newcastle leads us to Queensland where Ahmed, Neller and Katouli provide us with a review of faecal indicator microorganisms and current methods of faecal source tracking in surface waters. Their paper provides a critical evaluation of faecal indicators, and shows the advantages and limitations of current methods for tracking the sources of faecal contamination.

While focusing on Queensland, it is a good time to note that the AIEH Queensland Branch is currently accepting nominations for the 2006 Environmental Health Excellence Awards. Be recognised as an Environmental Health achiever and a leader in your field. Please see the AIEH website for more information.

Back to the current issue, our attention turns to an article on Environmental Health Impact Assessment (EHIA) in the Western Pacific Region. Spickett and Graham cover the links between EHIA and the need for supportive legislation to achieve public health goals in the region.

Those of you in Western Australia should

also note the upcoming Western Australia State Conference running from the 4th - 5th May 2006. The Conference will cover where environmental health fits in the community, and what makes the profession of environmental health tick. Please see the AIEH website for more information.

Make sure you also take time to browse the Book Review section in this issue of *Environmental Health*. Texts reviewed include:

*Environmental Health: From Global to Local*; and

*Occupational Health & Safety: International Influences and the 'New' Epidemics*.

Many thanks to Thomas Tenkate and Jeffrey Spickett for their time in reviewing these texts. If you would like to contribute

to the Book Review section of *Environmental Health*, feel free to contact us. If you have a particular book in mind that you would like to review, please contact us to organise a copy of the text from the publishers.

*Environmental Health* has also recently put out a Call for Papers for our 2006 Journal issues. All those interested in contributing research, practice initiatives, discussions of policy or legal and regulatory change, and reports or reviews to the Journal in 2006 should contact the Assistant Editor, Jaclyn Huntley at [journal@aieh.org.au](mailto:journal@aieh.org.au).

We look forward to receiving your papers in 2006.

*Jim Smith*  
*Editor*



## The Development of the Tasmanian Bachelor of Environmental Health

Great News for Tasmanian Environmental Health Practitioners.

As many of you would be aware, the issue of establishing a Bachelor of Environmental Health (BEH) to meet the needs of environmental health professionals in Tasmania has been on the agenda for many years.

Importantly, following recent meetings between senior representatives of the Australian Institute of Environmental Health (AIEH), the University of Tasmania (UTas), TAFE Tasmania, the Local Government Association of Tasmania (LGAT) and the Department of Health and Human Services (DHHS), agreement has been reached to establish an appropriate course at UTas commencing in 2007.

Among other benefits, the establishment of a BEH in Tasmania will:

- meet current and future national minimum qualifications as recognised by AIEH and other States and Territories;
- align with other States and Territories in order to facilitate portability;
- increase the profile and the credibility of the profession in Tasmania;
- improve the capacity of the profession to meet new challenges; and
- significantly increase the number of graduates above current levels.

Over the past year a working group has been meeting on a regular basis and progress with issues, such as subject content, method of delivery, specialist subject delivery, and practicum content, are now approaching the status required for submission to the AIEH for accreditation, and to UTas for its approval processes.

Naturally, consultation is very important to achieving a successful outcome and in this regard practitioners, employers and other stakeholders are being actively consulted throughout the process to ensure graduates are 'employment ready'. Representatives from related agencies, including primary industry, environment, energy and resources, are also included to ensure that issues, such as on-site waste disposal, air and water quality, noise control and environmental management, are appropriately supported in the course.

In order to keep all stakeholders informed and to seek active input to the process, degree bulletins are being issued on a regular basis. As the process moves forward, further workshops will be conducted to enable stakeholders to express their views and to provide detailed feedback on course content and related matters.

*Ron Fry  
AIEH, Hobart City Council, Tasmania*

**Back to  
TOC**

---

## RESEARCH AND THEORY

### Respiratory Health of Underground Coal Miners in Newcastle

<sup>1</sup>Zhen Hai Gu, <sup>1</sup>He Wang, <sup>2</sup>Paul Thomas, <sup>2</sup>Richard Henry and <sup>3</sup>Rui Wang

<sup>1</sup>*Department of Public Health, University of Adelaide, South Australia,* <sup>2</sup>*School of Medicine, University of New South Wales,*  
& <sup>3</sup>*Shandong Institute of Labour Health and Occupational Disease, Jinan, China*

*Underground coal miners are exposed to a mixture of airborne hazards and their respiratory health may be affected even if exposures to individual hazards are below the exposure standard. The aims of the research were to assess the association of respiratory impairments and exposure to underground coal mining. Two hundred and ten (210) miners in Newcastle coal mines and 95 unexposed subjects in the same area were recruited. Demographic information, occupational history, respiratory abnormalities, smoking profile and health history was obtained by questionnaire survey prior to lung function measurement. Data were analysed by univariate, bivariate and multivariate statistics. It was found that respiratory abnormalities did not occur more often in underground coal workers compared with controls, and associations between parameters of lung function and exposure were not demonstrable after adjustment. The study results might be an indication of continuing effective hazard control in these mines.*

**Key words:** Coal Mining; Respiratory Symptoms; Lung Function

Airborne hazards in coal mining environments include a number of potentially hazardous substances such as coal dust, diesel exhaust and quartz (World Health Organization 1986). These substances are generated from the extraction and processing of coal minerals (Coplu et al. 2005) and the primary route of exposure is inhalation during the course of employees' work (Driscoll et al. 2004). The main health effects of this mixed exposure are non-malignant respiratory diseases such as asthma, chronic obstructive pulmonary disease (COPD), silicosis, and coal workers' pneumoconiosis (CWP) (Driscoll et al. 2004). Although incidences of respiratory diseases in coal mining have been reduced dramatically over the past 30 years due to markedly improved mechanisation and standards of industrial hygiene, the mixed exposure might still lead to compromised respiratory health and reduced working capacity among coal mining workers in an insidious way.

Researchers have investigated the relationships between exposure, respiratory abnormalities, and lung function in mining workers. A cross-sectional study concluded that occupational exposure to coal dusts was associated with a small mean deficit in lung function among British coal miners (Lewis et al. 1996). Attfield and Hodous (1992) also demonstrated that coal dust exposure was related to reductions in Forced Expiratory Volume in one second (FEV<sub>1</sub>) among US coal miners with no radiographic evidence of pneumoconiosis. Another longitudinal study in America showed that exposure to coal mineral dusts was associated with greater prevalence of respiratory symptoms and accelerated loss of lung function (Beeckman 2001). A prospective study in 1954-1982 on the effect of nitrogen oxides, which is a component of diesel exhaust, at nine British coal mines indicated that long-term exposure to low concentrations of nitrogen

oxides was associated with increased susceptibility to respiratory infections (Jacobsen 1988).

Australia is the largest exporter of black coal in the world, and the coal mining industry is a major source of export income for the Australian economy (McKay, Lambert & Miyazaki 2001). There are more than 20,000 employees in the Australian coal mining industry (Minerals Council of Australia [MCA] 2001). These workers are potentially exposed to many airborne hazards such as coal mineral, diesel exhaust, silica, methane, and radon. These substances might have combined effects on coal miners' respiratory health, and the effects might develop in an insidious way leading to irreversible lung diseases. Based on these concerns, a historical cohort study was conducted at underground coalmines in the Newcastle area. Underground coal miners were selected since this group was exposed to relatively high levels of mixed hazards. Data obtained were analysed to examine if there were differences in respiratory abnormalities and lung function between underground coal miners and unexposed controls. Better understanding of this issue could assist in assessing lung health of those coal miners, and provide health workers and policy makers with more information relevant to effective prevention of respiratory illness in the coal mining industry.

## **Methods**

### **Subjects**

This investigation had been approved by the Human Research Ethics Committee of the University of New South Wales when it was initiated. The calculation of sample size (Fang 2003) revealed that approximately 200 underground coal miners and 90 control subjects were needed to detect the difference of lung function between underground coal miners and a normal population with a 95% level of confidence.

Computer randomisation of employee ID was utilised to select both exposed and unexposed subjects. Two hundred and ten (210) Australian male underground coal miners working in the Newcastle coal mining area were selected. Ninety five (95) male employees were recruited as control subjects who worked in companies at Newcastle. This study was restricted to males because there were very few exposed female coal miners.

### **Questionnaire**

An interviewer-administered questionnaire survey was conducted for all participants. Three registered nurses were employed to collect the information including demographic details, employment history, respiratory abnormalities, smoking profile, and other possible confounders such as family history of asthma, medical history of tuberculosis (TB), and renal diseases. According to the British Medical Research Council (BMRC) questionnaire (BMRC 1986), questions about respiratory symptoms were modified using the temporal relationship of respiratory abnormalities with work. Chronic bronchitis was defined as cough and phlegm production on most days for at least three months for a year and for no less than two successive years. Smoking profile included smoking status (non-smokers, ex-smokers and current smokers), smoking duration, and cigarette number per day. An ex-smoker was defined as stopping smoking at least one year before the survey, and a current smoker was defined as smoking at least one cigarette daily for at least one year. Based on the amount of cigarettes smoked per day and smoking duration, pack-year of smoking was calculated for each former or current smoker. One pack-year was defined as having smoked 20 cigarettes (one commercial pack) per day for one year.

### **Measurement of height, weight and lung function**

The height and weight of each participant wearing underwear only was measured by a 420KL Physician Balance Beam Scale (Health-O-meter, Australia). As the most commonly used indices of mechanical function of lungs in hospital (Levy & Wegman 2000), the FEV<sub>1</sub> and Forced Vital capacity (FVC) of each subject were measured with a computerised spirometry system (Medgraphic, Pulmonary Function System 1070, Series 2, St. Paul, Minnesota, USA) according to American Thoracic Society (ATS) recommendations (ATS 1987). The largest observed value of three efforts for each subject was used. The FEV<sub>1</sub>% and ratio of FEV<sub>1</sub> to FVC (FEV<sub>1</sub>/FVC) were calculated individually.

### **Statistical analysis**

Mean and standard deviation (SD) of variables on personal characteristics and demographic information for unexposed workers and underground coal miners were calculated respectively. Prevalence of respiratory abnormalities, mean and SD of lung function parameters in each exposure group were also calculated.

Student *t*-test (for variables with normal distribution) or Mann-Whitney *U* test (for variables without normal distribution) were used to compare the difference in mean of continuous variables on population characteristics and lung function between unexposed workers and underground coal miners. Chi-square test or Fisher's exact test was performed to compare the categorical factors on population characteristic between two exposure groups, and assess the difference in prevalence of each respiratory abnormality between exposed and unexposed subjects.

The relationship between each respiratory abnormality (dichotomous variable) and exposure was assessed with adjustment for the confounders in multiple logistic regression analysis. Multiple linear regression analysis was used to assess the

association of lung function parameters with exposure after adjustment for the confounding effects. The relationship between lung function and underground work duration in coal miners was also assessed after being controlled for the confounders by applying multiple linear regression analysis.

The statistical package Stata™ release 8.0 was used in all statistical analyses (Ryan 2004). Graphs were made using MS Excel 2003. Statistical significance was assumed for *P* value less than 0.05. 95% confidential intervals (95% CI) of measures of association were calculated.

## **Results**

There were 305 male individuals aged 19-64 years participating in this survey. The response rate was 100%. Nobody had a medical record of TB or renal diseases.

### **Descriptive analysis of personal characteristics for unexposed and exposed workers**

The personal characteristics and demographic information of the study population by exposure are presented in Table 1. The mean ages were  $36.0 \pm 10.2$  years for unexposed workers and  $44.5 \pm 8.37$  years for underground coal miners, the difference being significant. There was a marginally significant difference of mean height ( $p = 0.05$ ) between two exposure groups. Smoking status showed the statistically significant difference between unexposed and exposed workers: non-smokers (32.6% vs 41.9%), ex-smokers (16.9% vs 25.2%) and current smokers (50.5% vs 32.9%). The proportion of current smokers among underground coal miners was smaller than that in the unexposed group. Variables such as proportion of family history of asthma, mean weight, and pack-years of smoking did not reach statistical significance between the unexposed workers and exposed population. Only coal miners worked underground (mean underground work duration  $17.6 \pm 7.7$  years).

**Table 1: Personal characteristics and demographic information of unexposed workers and underground coal miners**

	Unexposed workers	Exposed workers
Number of subjects, n	95	210
Age (year)*, mean $\pm$ SD	36.04 $\pm$ 10.21	44.52 $\pm$ 8.37
Height <sup>†</sup> (cm), mean $\pm$ SD	175.31 $\pm$ 6.84	176.85 $\pm$ 6.35
Weight (kg), mean $\pm$ SD	85.91 $\pm$ 12.74	88.54 $\pm$ 13.15
Smoking status* n (%)		
non-smoker	31 (32.63)	88 (41.90)
ex-smoker	16 (16.84)	53 (25.24)
current smoker	48 (50.53)	69 (32.86)
Pack-years of smoking, mean $\pm$ SD	8.11 $\pm$ 9.56	9.98 $\pm$ 15.54
Family history of asthma, n (%)	16 (16.84)	22 (10.53)
Underground work duration (year), mean $\pm$ SD	N/A	17.63 $\pm$ 7.68

\*  $p < 0.05$ ; <sup>†</sup>  $p = 0.05$ ; N/A, not available.

#### Comparison of respiratory abnormalities between exposure groups

The prevalence of respiratory symptoms and diseases in the two exposure groups is shown in Table 2. Apart from bronchitis, the prevalence of respiratory symptoms in underground coal miners was higher than that of unexposed workers (cough 17.42% vs 9.47%, wheezing 14.61% vs 5.26%, breath shortness 8.99% vs 2.11%). Sore throat was only reported by underground coal miners. However, after adjustment for age and smoking status, exposure was not significantly associated with respiratory abnormalities. There were insufficient cases of sore throat for multivariate analysis.

**Table 2: Prevalence of each abnormality, and comparison in unexposed workers and underground coal miners before and after adjustment**

Symptom	Unexposed with symptom n (%)	Coal miners with symptom n (%)	$p^*$	OR <sup>†</sup> (95% CI)	$p^†$
Cough	9 (9.47)	31 (17.42)	0.08	1.77 (0.76-4.11)	0.18
Wheezing	5 (5.26)	26 (14.61)	0.02	2.86 (0.98-8.40)	0.06
Breath shortness	2 (2.11)	16 (8.99)	0.03	3.72 (0.79-17.42)	0.10
Sore throat	0 (0.00)	12 (5.71)	0.02	N/A	N/A
Bronchitis	17 (17.89)	33 (15.71)	0.63	1.15 (0.56-2.35)	0.71

(1) \* Before adjustment;

(2) <sup>†</sup> Odds ratios (OR) and  $p$  value adjusting for age (continuous variable) and smoking status (categorical variable);

(3) N/A, not available.

#### Comparison of lung function between exposure groups

The unexposed subjects had higher lung function than exposed subjects. Mean FEV<sub>1</sub> of both unexposed and exposed was greater than predicted value (100.77% and 100.64% respectively) (Table 3). Before adjustment, a student  $t$  test revealed a  $p$ -value of FEV<sub>1</sub> with exposure reached of marginal significance ( $p = 0.05$ ). However, after adjustment for age, height, and smoking status, exposure to mixed respiratory hazards at underground coalmines was not associated with parameters of lung function (Table 3). Multiple linear regression analysis also revealed the independent effects of age, height and smoking status on lung function. Age and smoking status were inversely associated with FEV<sub>1</sub> and FVC, but coefficient of height was positive (not shown in the table).

#### Effect of underground work duration on lung function in exposed group

The scatter diagrams between each of FEV<sub>1</sub>, FVC, FEV<sub>1</sub>% and FEV<sub>1</sub>/FVC and underground work duration among coal miners are shown in Figures 1 to 4. There appears to be a linear trend between each parameter and underground work duration. Correlation analysis in coal miners revealed both FEV<sub>1</sub> ( $r = -0.29$ ;  $p < 0.00$ ) and FVC ( $r = -0.29$ ;  $p < 0.00$ ) were correlated with underground work duration. However, after

**Table 3: Mean lung function and exposure with p value before and after adjustment**

	Exposure		P*	P <sup>†</sup>
	Unexposed	Exposed		
FEV <sub>1</sub> (L), mean $\pm$ SD	4.04 $\pm$ 0.77	3.88 $\pm$ 0.61	0.05	0.58
FVC (L), mean $\pm$ SD	4.98 $\pm$ 0.93	4.81 $\pm$ 0.75	0.17	0.40
FEV <sub>1</sub> %, mean $\pm$ SD	100.77 $\pm$ 15.64	100.64 $\pm$ 12.96	0.94	0.56
FEV <sub>1</sub> /FVC (%), mean $\pm$ SD	81.10 $\pm$ 6.60	80.52 $\pm$ 5.72	0.36	0.60

\* Before adjustment; <sup>†</sup> With adjustment for age, height (continuous variable) and smoking status (P-value of FEV<sub>1</sub>% was adjusted only for smoking status).

adjustment for age, height, and smoking status, underground work duration was not a useful predictor of FEV<sub>1</sub> ( $p < 0.721$ ) and FVC ( $p < 0.483$ ) within the exposed group (not shown in the table).

### Discussion

Analysis of the obtained data from the current study did not find, after adjustment for confounders, significant associations between greater prevalence of respiratory abnormalities or diminished lung function and underground coal mining in the investigated mines. Underground work-duration was not found to be a useful predictor of lung function change in the exposed coal miners. Multiple linear regression analysis on FEV<sub>1</sub> and FVC in all participants indicated significantly independent effects of age, height and smoking. FEV<sub>1</sub> and FVC were decreased with the increase in age and decrease in height. Smoking was shown to be a risk factor for FEV<sub>1</sub>, FEV<sub>1</sub>% and FVC.

A number of studies have been conducted to assess the association of occupational exposure to mixed hazards (especially to coal dusts) with respiratory abnormalities and/or loss of lung function, in which the findings supported a significant relation between the exposure and harmful effects. A literature review by Garshick, Schenker and Dosman (1996) demonstrated an association between occupational exposure to a variety of dusts, gases and fumes, with chronic bronchitis and decrements of FEV<sub>1</sub> in coal miners. Ross and Murray (2004) reviewed occupational respiratory diseases in the mining industry and concluded that coal-mining dusts are associated with chronic

bronchitis, chronic airflow limitation and emphysema. Carta et al. (1996) reported even moderate exposures to mixed coal dusts still significantly affected lung function and incidence of symptoms in underground coal miners. Seixas et al. (1993) found a significant association of cumulative exposure to coal mineral dusts with the loss of FEV<sub>1</sub> of approximately 5.9 ml per mg/m<sup>3</sup>-years.

However, the results of this investigation did not provide evidence to confirm that underground coal mining in Newcastle, Australia is related to an increase in the prevalence of respiratory symptoms and reduced lung function. Further, a study conducted during 1995/1996 at three Australian bauxite mines with exposure assessment did not find evidence of a serious adverse effect on respiratory health associated with exposure to bauxite mining (Beach et al. 2001). A five-year prospective study in the United States by Ames, Hall and Reger (1984) also found no significant association between exposure to diesel emissions and chronic respiratory effects among underground coal miners. This is probably due to the improved dust control and health protection programs that have existed for the past 30 years.

The findings in this study should be interpreted cautiously because of certain limitations. First, selection bias could not be avoided in the study design. Those underground coal miners who had withdrawn from the workforce due to poor respiratory health were not included in the study. The healthy worker effect might therefore exist in this investigation. Second, a reporting bias in increased reporting of respiratory symptoms

Figure 1: Scatter plot of FEV<sub>1</sub> against underground work duration with linear regression line of best fit

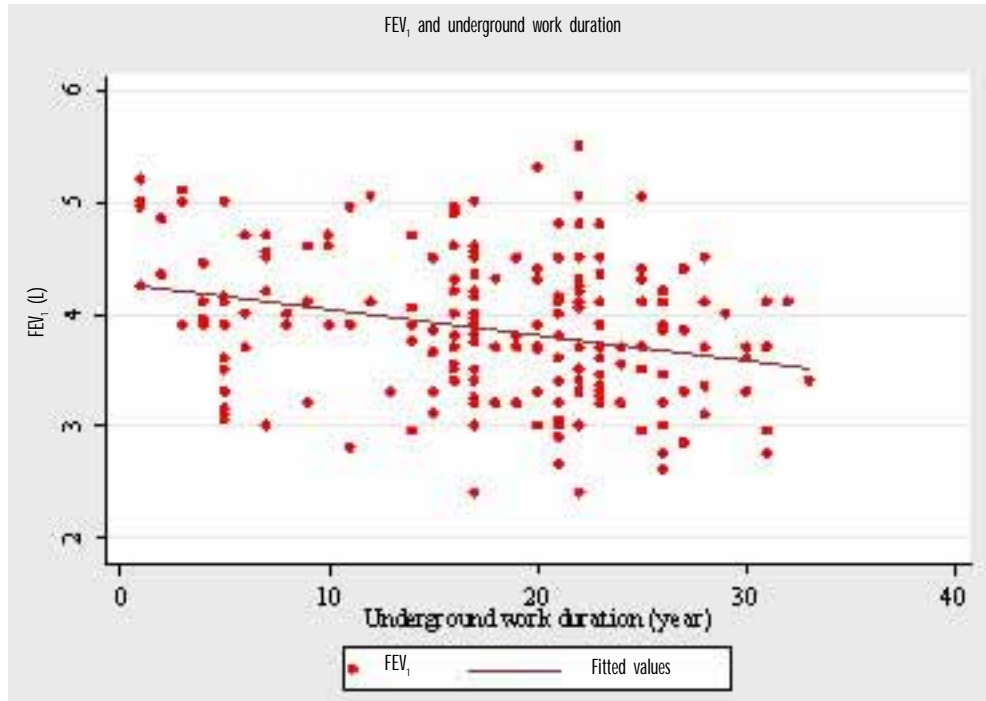
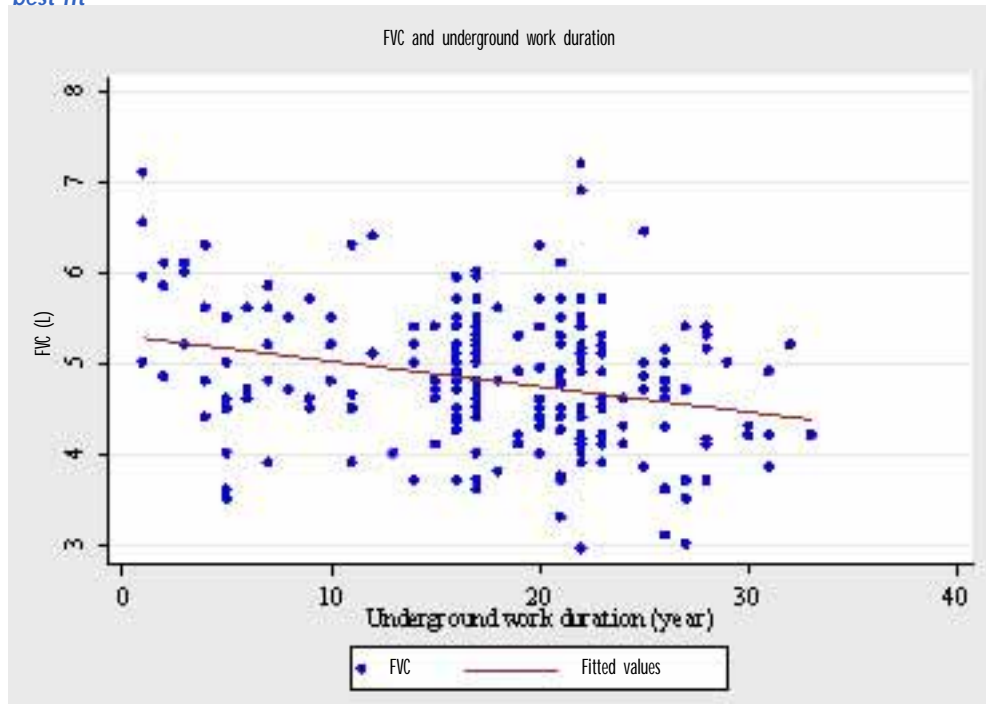
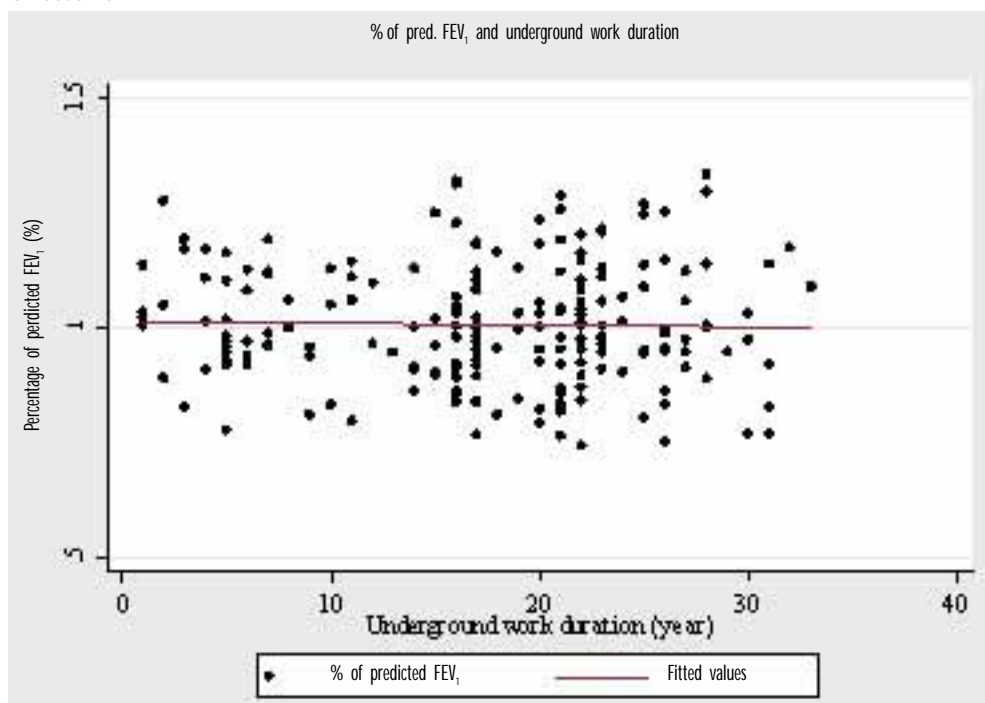


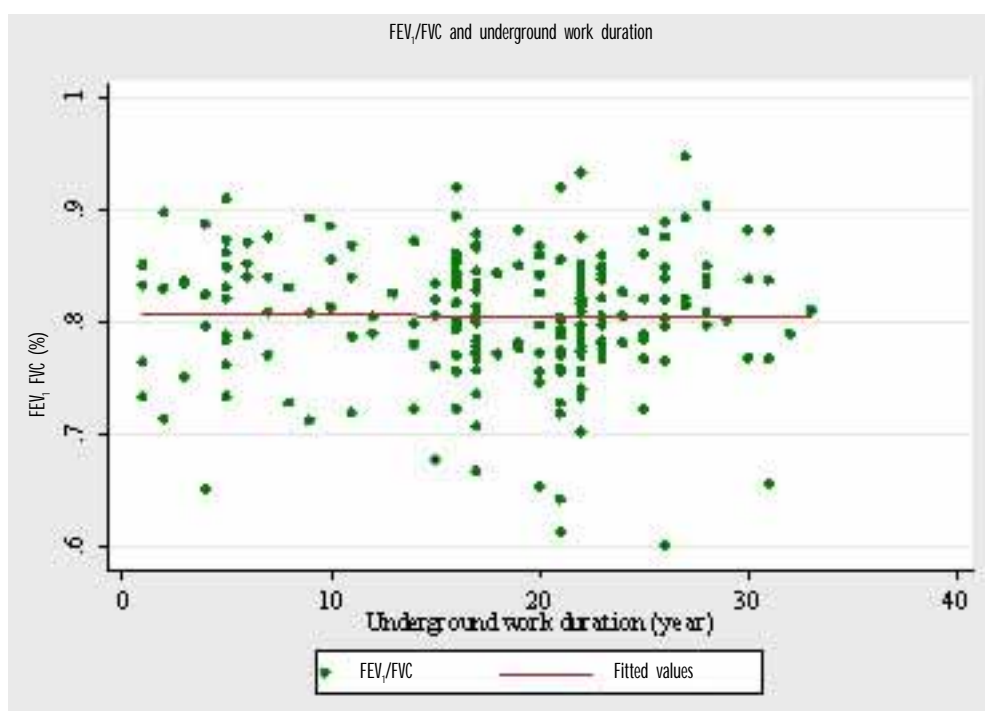
Figure 2: Scatter plot of FVC against underground work duration with linear regression line of best fit



**Figure 3: Scatter plot of  $FEV_1\%$  against underground work duration with linear regression line of best fit**



**Figure 4: Scatter plot of  $FEV_1/FVC$  against underground work duration with linear regression line of best fit**



by the unexposed workers could disguise the results. Unexposed subjects working in the office who usually have higher education might be more sensitive to their uncomfortable feelings.

Another consideration is that a number of possible confounding factors were not investigated in the study design. Some studies performed skin-prick tests to diagnose atopy that might confound the relationship between dust exposure and, respiratory abnormalities and lung function (Beach et al. 2001; Coplu 2005). Alternatively, a study by Burchfiel et al. (1997) implied that lung function is affected by many factors including grip strength, physical activity, subscapular skinfold thickness, presence of an ECG abnormality, heart rate, white blood cell count, and eosinophil percentage. Wang et al. (1999) found that childhood pneumonia, childhood exposure in the home to passive tobacco smoke, and possibly smoke due to wood and coal fuels were also associated with declines in pulmonary function. The potential confounders that were not controlled for might also disguise the true association between respiratory abnormalities and exposure in this investigation. Although it is difficult to collect this amount of detailed

information, the better-designed studies that collect more comprehensive information might help confirm or deny the outcomes of the present study.

### Conclusion

In conclusion, this study did not find a significant association between underground coal mining and lung function change. Similarly, no association between respiratory symptoms and mining work could be established. However, some limitations of the investigation should be mentioned with the interpretations of these results. The effects of the coal mine's mixed airborne exposure on the respiratory system might be further investigated in larger populations by a prospective cohort study with the measurement of exposure level and hazard compositions at working sites. More sensitive methods might be used to identify early subtle changes before detectable lung function reduction. Currently, exhaled nitric oxide in combination with cellular and molecular changes in induced sputum is being examined to establish the possible association between exposure and potentially compromised lung health (Lund et al. 2000; Zeidler & Castranova 2004).

### Acknowledgment

The study was financially supported by a grant from the Dust Disease Board in New South Wales, Australia. There was no conflict of interest in the study.

### References

- American Thoracic Society 1987, 'Standardization of spirometry: 1987 update', *The American Review of Respiratory Disease*, vol. 136, no 5, pp. 1285-98.
- Ames, R.G., Hall D.S. & Reger, R.B. 1984, 'Chronic respiratory effects of exposure to diesel emissions in coal mines', *Archives of Environmental Health*, vol. 39, no. 6, pp. 389-94.
- Attfield, M.D. & Hodous, T.K. 1992, 'Pulmonary function of U.S. coal miners related to dust exposure estimates', *The American Review of Respiratory Disease*, vol. 145, pp. 605-9.
- Beach, J.R., De Klerk, N.H., Fritschi, L., Sim, M.R., Musk, A.W., Benke, G., Abramson M.J. & McNeil J.J. 2001, 'Respiratory symptoms and lung function in bauxite miners,' *International Archives of Occupational and Environmental Health*, vol. 74, pp. 487-94.
- Beeckman, L.F., Wang, M.L., Petsonk, E.L. & Wagner G.R. 2001, 'Rapid declines in FEV1 and subsequent respiratory symptoms, illness, and mortality in coal miners in the United States', *American Journal of Respiratory and Critical Care Medicine*, vol. 163, pp. 633-9.
- British Medical Research Council 1986, *Questionnaire on Respiratory Symptoms*, British Medical Research Council, London.

- Burchfiel, C.M., Enright, P.L., Sharp, D.S., Chyou, P., Rodriguez, B.L. & Curb J.D. 1997, 'Factors associated with variations in pulmonary function among elderly Japanese-American men', *Chest*, vol. 112, no. 1, pp. 87-97.
- Carta, P., Aru, G., Barbieri, M.T., Avataneo, G. & Casula, D. 1996. 'Dust exposure, respiratory symptoms, and longitudinal decline of lung function in young coal miners', *Occupational and Environmental Medicine*, vol. 53, no. 5, pp. 312-9.
- Coplu, L., Demir, A.U., Kalyoncu, A.F., Coplu, N., Selcuk, Z.T., Enunlu, T., Karakoca Y., Sahin A.A., Baris Y.I. 2005. 'Lung health in workers exposed to reed dust', *Respiratory Medicine*, Vol. 99, pp. 421-8.
- Department of Protection of the Human Environment 1999, *Hazard Prevention and Control in the Work Environment: Airborne Dust*, World Health Organization, Geneva.
- Driscoll, T., Steenland, K., Nelson, D.I. & Leigh, J. 2004, *Occupational Airborne Particulates: Assessing the Environmental Burden of Disease at National and Local Levels*, Protection of the Human Environment, World Health Organization, Geneva.
- Fang, J.Q. ed. 2003, *Health Statistics*, 5th edn, People's Medical Publishing House, China.
- Garshick, E., Schenker, M.B. & Dosman, J.A. 1996, 'Occupationally induced airways obstruction', *The Medical Clinics of North America*, vol. 80, no. 4, pp. 851-78.
- Jacobsen, M., Smith, T.A., Hurley, J.F., Robertson, A., Roscrow, R. 1988, 'Respiratory infections in coal miners exposed to nitrogen oxides', *Research Report, Health Effects Institute*, vol. 18, pp. 51-6.
- Levy, B.S. & Wegman, D.H. ed. 2000, *Occupational Health: Recognizing and Preventing Work-related Disease and Injury*, 4th edn, Lippincott Williams & Wilkins, City of Philadelphia PA, USA.
- Lewis, S., Bennett, J., Richards, K. & Britton, J. 1996, 'A cross sectional study of independent effect of occupation on lung function in British coal miners', *Occupational and Environmental Medicine*, vol. 53, no. 8, pp. 575-6.
- Lund, M.B., Oksne, P.I., Hamre, R. & Kongerud, J. 2000, 'Increased nitric oxide in exhaled air: An early marker of asthma in non-smoking aluminium pot room workers', *Occupational and Environmental Medicine*, vol. 57, no. 4, pp. 274-8.
- McKay, B., Lambert, I. & Miyazaki, S. 2001, *The Australian Mining Industry: From Settlement to 2000*, Australian Bureau of Statistics, Canberra.
- Minerals Council of Australia 2001. *Safety and Health Performance Report of the Australian Minerals Industry 2001-2002*, Minerals Council of Australia, Canberra.
- Ross, M.H. & Murray, J. 2004, 'Occupational respiratory disease in mining', *Occupational Medicine*, vol. 54, pp. 304-10.
- Ryan, P. 2004, *Biostatistics Elective: Course Documentation*. Department of Public Health, University of Adelaide.
- Seixas, N.S., Robins, T.G., Attfield, M.D. & Moulton L.H. 1993, 'Longitudinal and cross sectional analyses of exposure to coal mine dust and pulmonary function in new miners', *British Journal of Industrial Medicine*, vol. 50, no. 10, pp. 929-37.
- Wang, M.L., Petsonk, E.L., Beeckman, L.A. & Wagner G.R. 1999, 'Clinically important FEV1 declines among coal miners: an exploration of previously unrecognised determinants', *Occupational and Environmental Medicine*, vol. 56, no. 12, pp. 837-44.
- World Health Organization 1986, *Recommended Health-based Limits in Occupational Exposure to Selected Mineral Dusts [silica, coal]*, World Health Organization, Geneva.
- Zeidler, P.C. & Castranova, V. 2004, 'Role of nitric oxide in pathological responses of the lung to exposure to environmental/occupational agents', *Redox Report*, vol. 9, no. 1, pp. 7-18.

Correspondence to:

He Wang  
Department of Public Health,  
University of Adelaide  
Adelaide, South Australia, 5005  
AUSTRALIA  
Email: he.wang@adelaide.edu.au



# Intervention of Ticlopidine on Silica-Induced Inflammatory Reaction and Apoptosis

<sup>1</sup>He Wang and <sup>2</sup>James Leigh

<sup>1</sup>*Department of Public Health, University of Adelaide, South Australia* & <sup>2</sup>*School of Public Health, University of Sydney*

*Silica is a potent inducer of pulmonary inflammation that involves leucocyte apoptosis. It is hypothesised that regulation of the apoptotic reaction might occur in this condition through platelet activation arising from endothelial injury, by releasing products that modify the inflammatory cell function or life span, thereby influencing the reaction of inflammation. Because neutrophil apoptosis contributes to the resolution of acute inflammation induced by endotoxin, promotion of the apoptosis process might also inhibit the inflammatory reaction induced by silica. In order to test this hypothesis, rats were intratracheally instilled with 22.5 mg silica dust suspension, together with or without a four-week treatment of intraperitoneally injected daily (30mg/kg) doses of ticlopidine; a drug that can inhibit platelet aggregation in the process of activation. The results indicate that ticlopidine administration significantly enhanced the percentage of apoptotic neutrophils and reduced the number of lavagable cells and rate of lung weight increase. This evidence suggests that ticlopidine is an effective agent for producing an enhanced resolution of inflammation by the action of promoting neutrophil apoptosis.*

**Key words:** Silica; Inflammation; Rats; Apoptosis; Neutrophils; Macrophages

Although standards of industrial hygiene have improved markedly over the past more than 30 years, silica exposure remains a significant cause of occupational lung disease in Australia. Globally, it remains the major cause of occupational interstitial lung disease. Silica is frequently encountered in the workplace of many industries. It has been estimated by the National Occupational Health and Safety Commission (1993) that the number of workers occupationally exposed to silica-bearing dust was about 136,000 in Australia. This number is likely to have increased because of rapid development of mining and quarrying industries with a booming international market.

Silica is a well-known agent capable of inducing lung injury as characterised by inflammation and fibrosis. This inflammatory reaction, which is unresolvable and eventually leads to fibrosis, involves apoptosis (programmed cell death) of inflammatory

cells, mainly neutrophils (Leigh et al. 1997). It has been proposed that apoptosis of neutrophils and subsequent engulfment by alveolar macrophages in inflammatory reaction of lung contribute to the resolution of inflammation (Cox et al. 1995). It has also been demonstrated that promotion of neutrophil apoptosis can enhance the resolution of inflammation (Cox 1996).

In silica-induced lung reaction, injury of endothelium might be involved, and this, in turn, can activate platelets. This assumption, yet to be demonstrated directly, has some support from evidence that bleomycin, a drug which can also induce pulmonary inflammation and fibrosis, induces increased platelet trapping in the exposed lung (Piguet & Vesin 1994a). In addition, platelet activation factor (PAF) is released in pulmonary inflammation, which can induce platelet activation in the lung. The activation of platelets is a process that includes the somewhat artificially

subdivided steps of adhesion, aggregation, swelling, and secretion (Majno & Joris 1996). Activated platelets are capable of releasing a number of molecular species that play roles in inflammation (Nachman & Weksler 1980; Weksler 1988; White 1993). There is also some evidence that platelets can interact with neutrophils (Evangelista et al. 1996), and platelet-secreted diadenosine polyphosphates can delay neutrophil apoptosis in inflammatory conditions (Gasmi et al. 1996a, b). Inhibition of platelet activation might prevent platelets from releasing their molecules and thus intervene in the inflammatory reaction.

Ticlopidine is a clinically used drug that prevents thrombosis formation by inhibiting platelet aggregation (Phillips et al. 2005). It is hypothesised that such action can prevent platelet swelling and secretion during the process of platelet activation, which might then modify the inflammatory reaction and neutrophil function or life span. In order to test this hypothesis, an inflammatory reaction was established by intratracheal (i.t.) instillation of silica in rats, and effects studied of the cotreatment with intraperitoneal (i.p.) injection of ticlopidine.

## Method

### Animals

Specific pathogen free Wistar male rats (295-305g purchased from University of New South Wales Animal Breeding Centre in Little Bay) were used in this experiment and were maintained under barrier conditions. Food and water was freely accessible and the light was set on a 12/12 hours on/off cycle.

### Design

Twenty rats were distributed into 4 groups of 5 animals by randomised block design. The 4 groups were: i.t. silica and i.p. saline (QS); i.t. silica and i.p. ticlopidine (QT); i.t. saline and i.p. saline (SS); i.t. saline and i.p. Ticlopidine (ST).

### Treatment

Rats were anaesthetised by a mixture of ketamine (100 mg/kg) and xylazine (3.3 mg/kg) i.p. and i.t. instilled with 22.5 mg silica dissolved in 0.5 ml saline or with saline alone. Injection of ticlopidine and saline i.p. with 30 mg/kg ticlopidine started immediately after recovery of animals from anaesthetisation and maintained daily thereafter. After 4 weeks, the rats were killed by i.p. injection of 100 mg/kg pentobarbital and cutting of the aorta. Details can be seen in previous publication (Leigh et al. 1997).

### Bronchoalveolar lavage (BAL)

To obtain the airway lining fluid and free cells in the alveolar space, the rats were immediately lavaged with two 5 ml aliquots of phosphate buffered saline (PBS). The recovery fluid (BALF) was pooled and the volume recorded. Part of the fluid was used for counting of total cell number, some was used for cytopsin preparation, and the remaining fluid was centrifuged to obtain supernatant for LDH measurement. The lung weights were measured and recorded after removal of the bronchioles and trachea.

### Cytology and differential scoring

To observe cell morphology, a 100 µl volume of the lavage fluid was placed on a slide by cytopsin centrifugation immediately after the lavage. For each rat, at least two slides were prepared and stained with Diff-Quik (Lab Aids Pty Ltd, Sydney Australia). The slides were read under oil immersion (x 1000) and 500 leucocytes were counted to determine the frequency of different types of cells based on their morphology.

### Total cell number and lactate dehydrogenase (LDH) activity

Total cell number was estimated with a haemocytometer and the remaining fluid was centrifuged (1500 x g, 10 minutes). The supernatant was collected to measure LDH activity by a kit method (Trace, Sydney Australia). Total cell number can reflect the

infiltration of inflammatory cells and LDH can reflect the level of cell content release or necrosis.

#### Counting of apoptotic cells

To estimate the apoptotic occurrence of apoptosis in inflammatory cells, a minimum of 1000 leucocytes were scored for the occurrence of cells with apoptotic features to determine the proportion of apoptotic leucocytes, and a minimum of 500 neutrophils were counted to determine the percentage of apoptotic neutrophils.

#### Statistical analysis

The results are expressed as means (standard error (se). Student-Newman-Keul's test was used for multiple comparison of different animal groups of each index with the software package InStat because this test method is suitable for multiple comparison. Statistical significance was preset at  $p < 0.05$ .

### Results

#### Inflammatory reaction

Intratracheal instillation of silica induced an obvious pulmonary inflammatory reaction. Figure 3 shows mean lung weight in the four groups. Lung weight in the QS group was significantly higher than that in the SS group (QS=6.193±0.201; SS=2.392±0.063 grams). The lung weights in SS group and ST group were similar and no significant difference could be detected between the SS and ST groups.

Lung weight in the QS group was significantly higher than that in the QT group (QS=6.193±0.201; QT=5.347±0.279; grams;  $p < 0.01$ ). The lung weights in both the silica-instilled groups were significantly higher than the SS and ST groups. ( $p < 0.001$ ).

The total cell numbers in BALF of the QS, QT, SS and ST groups were 50.73±5.15, 36.48±4.22, 12.00(0.69, and 13.47±0.71 ( $\times 10^4$ )/ml, respectively (Figure 4). The silica-instilled groups had significantly higher cell numbers than the saline-instilled

groups ( $p < 0.001$ ). The QT group had lower cell numbers than the QS group and the difference was statistically significant ( $p < 0.01$ ). There was no significant difference between the SS and ST groups.

The LDH activity in BALF in the QS, QT, SS and ST groups was 220.8±21.1, 197.8±15.7, 37.4±5.8, and 39.2±4.3 U/L, respectively (Figure 5). Silica-instilled groups had significantly higher activity than the saline-instilled groups ( $p < 0.001$ ). No significant difference could be detected between the QS and QT or SS and ST groups.

#### Cell composition in BALF

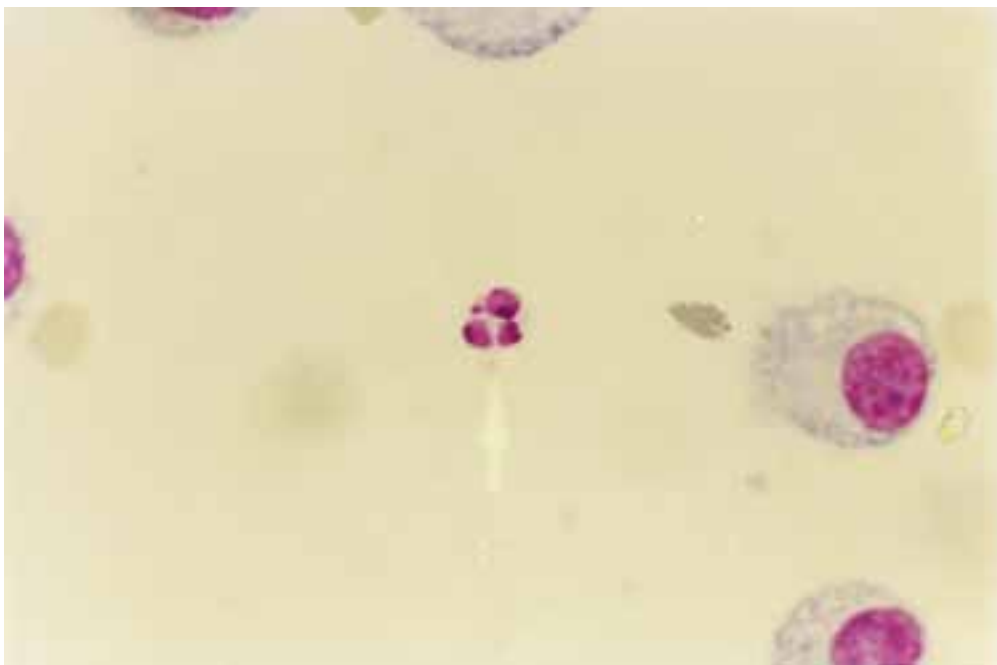
The cell type in the two saline-instilled groups was mainly macrophage. Neutrophils were very few and other cell types such as lymphocytes and monocytes were rarely seen. In the silica-instilled groups, however, the cell type was mainly neutrophil. Macrophages became a minority and other cell types were also few. No statistically significant difference could be detected between QS and QT groups in both percentages of macrophages and neutrophils. The percentages of macrophages and neutrophils in the four groups are shown in Figure 6 and 7, respectively.

#### Apoptotic reaction in BALF cells

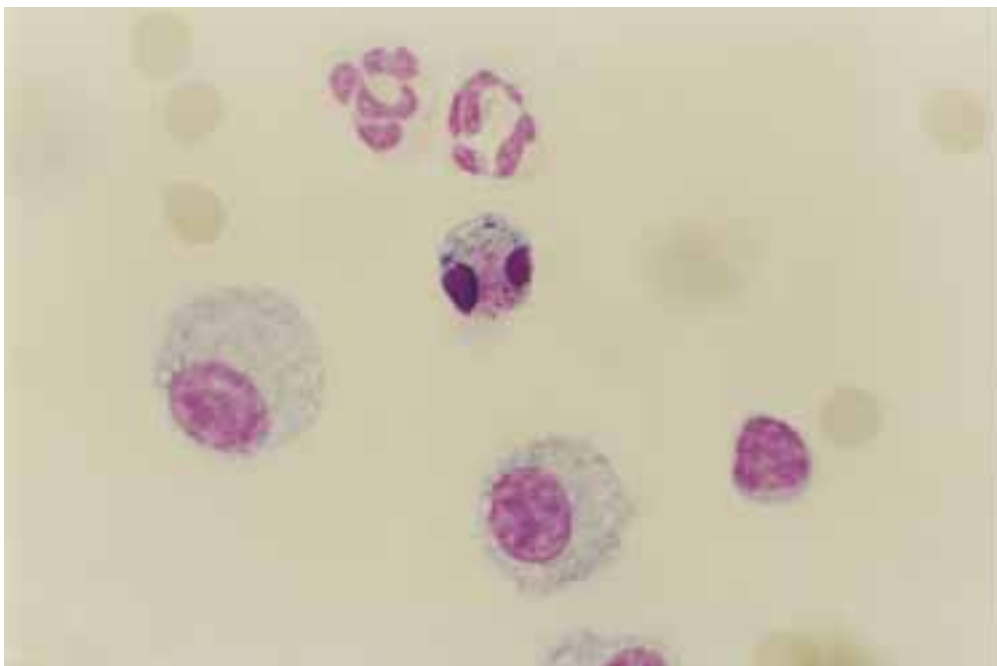
In the Diff-Quik-stained slides, apoptotic cells, mainly neutrophils, were detected. The proportion of apoptotic cells among leucocytes in QS and QT groups was 16.97±0.66 and 44.31±3.43, respectively (per 1000 cells, see Figure 8). The proportion in the QT group was significantly higher than that of QS ( $p < 0.001$ ). The percentages of apoptotic neutrophils among total neutrophils in QS, QT, SS and ST groups were 2.7±0.3, 7.2±0.6, 0 and 0, respectively (Figure 9). The QT group was significantly higher than the QS group ( $p = 0.0002$ ).

The proportions of macrophages with engulfed cells in total macrophages in QS,

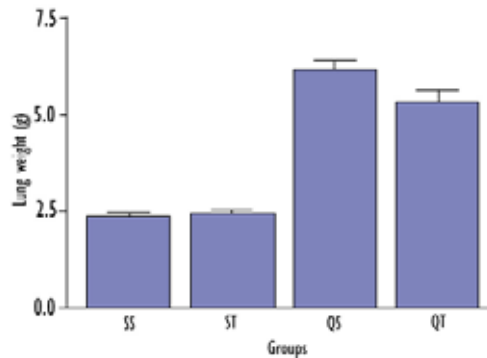
*Figure 1: Apoptotic neutrophil in bronchoalveolar lavage fluid from silica-instilled rats (x1000)*



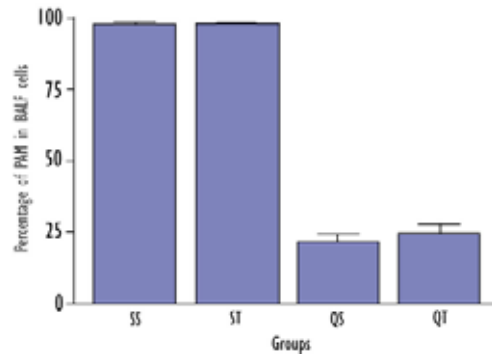
*Figure 2: Apoptotic macrophage (under the two neutrophils with condensed parts of nucleus) in bronchoalveolar lavage fluid from silica-instilled rats (x1000)*



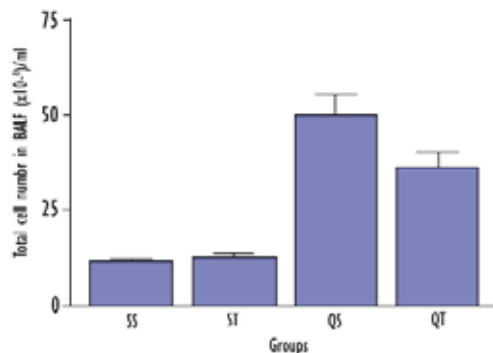
**Figure 3:** Lung weight of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean+se)



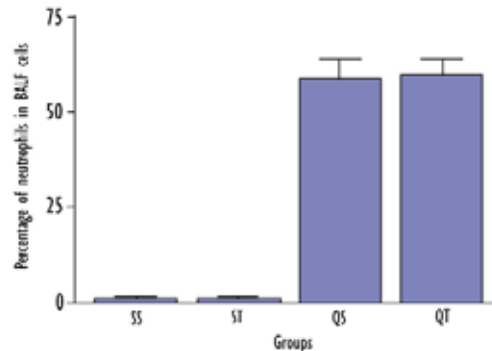
**Figure 6:** Percentage of pulmonary alveolar macrophages (PAM) in BALF cells of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean+se)



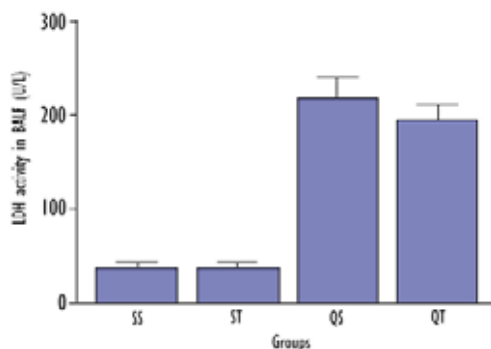
**Figure 4:** Total cell number in bronchoalveolar lavage fluid of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean+se)



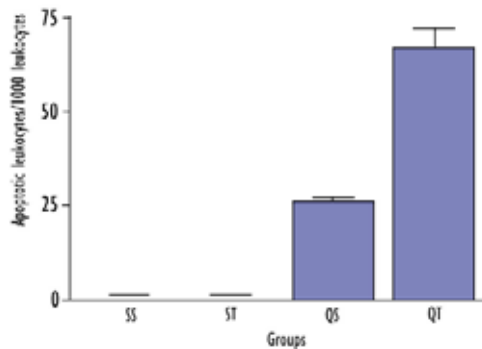
**Figure 7:** Percentage of neutrophils in BALF cells of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean+se)



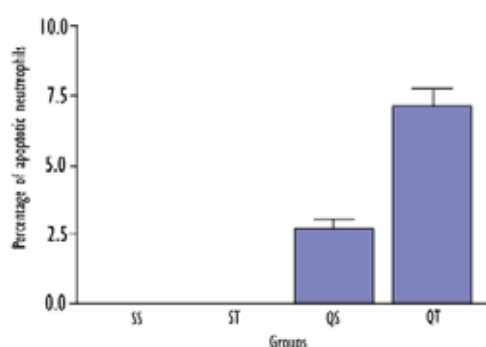
**Figure 5:** Lactate Dehydrogenase (LDH) activity in bronchoalveolar lavage fluid of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean+se)



**Figure 8:** Proportion of apoptotic leukocytes in BALF leukocytes of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean+se)



**Figure 9: Percentage of apoptotic neutrophils in BALF neutrophils of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean±se)**

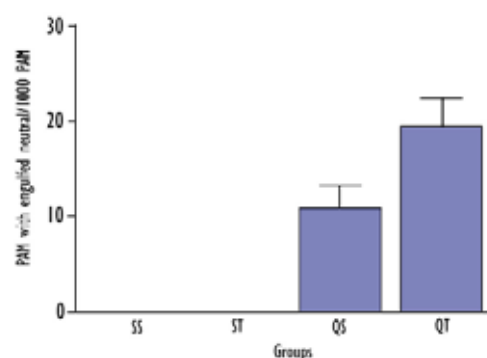


QT, SS and ST groups were  $11.0 \pm 2.4$ ,  $19.6 \pm 2.9$ , 0 and 0, respectively (per 1000 macrophages, see Figure 10). The QT group was higher than the QS group, but the difference was only statistically marginal ( $p=0.0517$ ).

### Discussion

Chronic inhalation exposure of workers to crystalline silica can result in silicosis. The general public can also be exposed to lower levels of crystalline silica from quarries, sand blasting, and entrained fines particles from surface soil (Collins et al. 2005). There is always a lag period between silica exposure and diagnosis of silicosis. The diagnosis of silica-induced diseases is mainly based on clinical findings, lung function and X-Ray examinations. None of these is sensitive to early stage disease or subtle progression. Equally, though the disease is poorly treatable at present, none of these modalities is likely to offer a means to measure the success of novel treatments. These facts indicate that silica-induced disorders are generally persistent, insidious and irreversible. The underlying mechanism on the persistence of silica-induced harmful effects is not yet completely understood. Revealing the

**Figure 10: Proportion of pulmonary alveolar macrophages (PAM) with engulfed apoptotic cells in BALF macrophages of rats after intratracheal instillation of silica or saline and intraperitoneal injection of saline or ticlopidine (mean±se)**



mechanism may help explain the process of silica-induced fibrosis and potential carcinogenic effect.

The experiments in this study describe a rat model of silica-induced inflammation. In our previous studies of this process, it was demonstrated that neutrophil apoptosis occurred in a delayed pattern and the delay could have contributed to the persistence of the inflammation (Leigh et al. 1998). There is some evidence that platelet/neutrophil interaction can occur in inflammatory reactions (Bengtsson & Grenegard 1994; Naum 1994), and that platelets are able to secrete certain molecules that delay neutrophil apoptosis (Gasmi 1996a; Gasmi 1996b). The present study demonstrated that ticlopidine administration can enhance neutrophil apoptosis and concurrently reduce the increase of total cell number in BALF and lung weight increase. This is the first demonstration that a platelet aggregation inhibitor, ticlopidine, can enhance neutrophil apoptosis in silica-induced reaction and reduce the inflammatory reaction.

Regarding the mechanism of neutrophil apoptosis enhancement by ticlopidine, it is possible that this substance exerts its effect

via inhibition of platelet aggregation. This is a well-established property of this drug that is clinically employed to prevent thrombosis. It is a reasonable speculation that silica exposure might cause platelet activation by injury of pulmonary vessels or activation of inflammatory cells. In fact, it has been demonstrated that bleomycin, a drug that causes pulmonary inflammation and fibrosis, increases platelet trapping in the lung of treated mice (Piguet & Vesin 1994a). The inhibition of platelet aggregation may prevent the subsequent steps of platelet activation in an inflammatory condition and hence prevent the release of the contained chemical agents. Platelets can produce and release many agents including cytokines, growth factors and other agents such as diadenosine polyphosphates. The diadenosine polyphosphates  $Ap_3A$ ,  $Ap_4A$ ,  $Ap_5A$  and  $Ap_6A$ , secreted by platelets, can effectively delay neutrophil apoptosis and they can exert their effects in combination with granulocyte-macrophage colony-stimulating factor (Gasmi 1996a,b). Since cytokines such as  $TNF-\alpha$  and  $IL-1$ , which have the potential capability to induce neutrophil apoptosis, are also secreted in silica-induced inflammation (Driscoll et al. 1990), it is possible that platelet-secreted factors delay neutrophil apoptosis as a dual regulation of neutrophil function and life span.

Apoptotic features included formation of condensed chromatin bodies with sharp edges and convolution of the cell surface (Newman et al. 1982; Savill et al. 1989). Apoptotic macrophages and apoptotic neutrophils can be distinguished by morphology (see Figures 1 and 2). The morphological apoptosis is confirmed by agarose gel electrophoresis in our previous study (Leigh et al. 1997). Neutrophil apoptosis and subsequent engulfment by macrophages are a proposed mechanism for the resolution of acute inflammation (Cox et al. 1995). Promotion of neutrophil apoptosis was also demonstrated to enhance resolution of acute pulmonary inflammation (Cox 1996).

Importantly, visualisation of small percentages of in situ stained apoptotic cells yields biological significant data (Hussain et al. 1998). A rate of tissue regression as rapid as 25% per day can result from apoptosis in 2% to 3% of the cells at any one time (Brusch et al. 1990). The ticlopidine reduction of total cell number in BALF and of increased lung weight in silica-instilled rats may be attributed to the enhanced neutrophil apoptosis induced by ticlopidine. Apoptotic neutrophils may lose some of their functions (Lee et al. 1993; Dransfield et al. 1994), fail to release their lytic enzymes into the extracellular environment and become engulfed by alveolar macrophages and ultimately be degraded. This process might help to limit the degree and extent of the silica-induced inflammation and accelerate the removal of superfluous neutrophils.

Silica-induced inflammation predisposes to the development of fibrosis. There is accumulating evidence that cytokines and growth factors released in inflammatory conditions are responsible for the ensuing fibrotic process (Driscoll et al. 1990a; Elias et al. 1990; Krane et al. 1985; Li et al. 1992; Piguet et al. 1989, 1990, 1993; Piguet & Vesin 1994b). It is possible that enhanced resolution of inflammation leads to the inhibition of fibrosis. If silica-induced chronic inflammation is a contributing factor for silica carcinogenesis (Borm & Driscoll 1996), it is possible that enhanced resolution of inflammation can reduce the risk of cancer development.

The underlying mechanism of silica-induced effects is complicated and poorly understood. Pulmonary macrophages are currently considered to play a central role in the development of silicosis. Nevertheless, other cell types including platelets might act on macrophages to modulate the inflammatory and fibrotic process. Activated platelets can release some agents modulating the function of monocytes (Engstad et al. 1995; Tsuji et al. 1994), which are the precursor of alveolar macrophages and differentiate into alveolar

macrophages in large quantity in silica-induced inflammation. This study demonstrated that ticlopidine can reduce silica-induced increase in lung weight and it is hypothesised that this effect is exerted by intervening in not only platelet/neutrophil interaction, but also in platelet/monocyte interaction. In addition, macrophages are also proposed to regulate neutrophil apoptosis (Aliprantis et al. 1996). Further studies on the role of platelets in the modulation of macrophage functions in silica-induced effects are also required.

## Conclusion

In summary, this study demonstrated that ticlopidine can enhance neutrophil apoptosis in silica-induced inflammatory reaction and reduce the severity of the reaction. There seems to be a correlation between enhanced apoptosis and reduced inflammation, which provides a basis for further study of the role of platelets in the development of silica-induced effects. The basis for clinical trial of ticlopidine in the treatment of silicosis is also provided.

## References

- Aliprantis, A.O., Diez-Roux, G., Mulder, L.C.F., Zychlinsky, A. & Lang, R.A. 1996, 'Do macrophages kill through apoptosis?' *Immunology Today*, vol. 17, pp. 573-6.
- Bengtsson, T. & Grenegard, M. 1994, 'Platelets amplify chemotactic peptide-induced changes in F-actin and calcium in human neutrophils', *European Journal of Cell Biology*, vol. 63, pp. 345-9.
- Borm, P.J.A. & Driscoll, K. 1996, 'Particles, inflammation and respiratory tract carcinogenesis', *Toxicology Letters*, vol. 88, pp. 109-13.
- Bursch, W., Paffe, S., Putz, B., Barthel, G. & Schulte-Hermann, R. 1990, 'Determination of the length of the histological stages of apoptosis in normal liver and in altered hepatic foci of rats', *Carcinogenesis*, vol. 11, pp. 847-53.
- Collins, J.F., Salmon, A.G., Brown, J.P., Marty, M.A. & Alexeeff, G.V. 2005, 'Development of a chronic inhalation reference level for respirable crystalline silica', *Regulatory Toxicology and Pharmacology* (in press).
- Cox, G., Crossley, J. & Xing, Z. 1995, 'Macrophage engulfment of apoptotic neutrophils contributes to the resolution of acute pulmonary inflammation in vitro', *American Journal of Respiratory Cell and Molecular Biology*, vol. 12, pp. 232-7.
- Cox, G. 1996, 'IL-10 enhance resolution of pulmonary inflammation in vivo by promoting apoptosis of neutrophils', *American Journal of Physiology*, vol. 271, pp. L566-L571.
- Dransfield, I., Buckle, A-M., Savill, J.S., McDowall, A., Haslett, C. & Hogg N. 1994, 'Neutrophil apoptosis is associated with a reduction in CD16 (FcγRIII) expression', *Journal of Immunology*, vol. 153, pp. 1254-63.
- Driscoll, K.E., Lindenschmidt, R.C., Maurer, J.K., Higgins, J.M. & Ridder, G. 1990, 'Pulmonary response to silica or titanium dioxide: inflammatory cells, alveolar macrophage-derived cytokines, and histopathology', *American Journal of Respiratory Cell and Molecular Biology*, vol. 2, pp. 381-90.
- Elias, J.A., Freundlich, B., Kern, J.A. & Rosenbloom, J. 1990, 'Cytokine networks in the regulation of inflammation and fibrosis in the lung', *Chest*, vol. 97, pp. 1439-45.
- Engstad, C.S., Lia, K., Rekdal, O., Olsen, J.O. & Osterud B. 1995, 'A novel biological effect of platelet factor 4 (PF4): Enhancement of LPS-induced tissue factor activity in monocytes', *Journal of Leukocyte Biology*, vol. 58, pp. 578-81.
- Evangelista, V., Manarini, S., Rotondo, S., Martelli, N., Polischuk, R., McGregor, Gaetano G. de, & Cerletti, C. 1996, 'Platelet/Polymorphonuclear leukocyte interaction in dynamic conditions: Evidence of adhesion cascade and cross talk between P-selectin and the b2 integrin CD11b/CD18', *Blood*, vol. 88, pp. 4183-94.
- Gasmi, L., McLennan, A.G. & Edwards, S.W. 1996a, 'The diadenosine polyphosphates Ap3A and Ap4A and adenosine triphosphate interact with granulocyte-macrophage colony-stimulating factor to delay neutrophil apoptosis: Implications for neutrophil:platelet interactions during inflammation', *Blood*, vol. 87, pp. 3442-9.

- Gasmi, L., McLennan, A.G. & Edwards, S.W. 1996b, 'Neutrophil apoptosis is delayed by the diadenosine polyphosphates, Ap5A and Ap6A: Synergism with granulocyte-macrophage colony-stimulating factor', *British Journal of Haematology*, vol. 95, pp. 637-9.
- Hussain, N., Wu, F., Zhu, L., Thrall, R.S. & Kresch, M.J. 1998, 'Neutrophil apoptosis during the development and resolution of oleic acid-induced acute lung injury in the rat', *American Journal of Respiratory Cell and Molecular Biology*, vol. 19, pp. 867-74.
- Krane, S.M., Dayer, J.M., Simon, L.S. & Byrne, M.S. 1985, 'Mononuclear cell-conditioned medium containing mononuclear cell factor (MCF), homologous with interleukin 1, stimulates collagen and fibronectin synthesis by adherent rheumatoid synovial cells: Effects of prostaglandin E2 and indomethacin', *Collagen Related Research*, vol. 5, pp. 99-117.
- Lee, A., Whyte, M.K.B. & Haslett C. 1993, 'Inhibition of apoptosis and prolongation of neutrophil functional longevity by inflammatory mediators', *Journal of Leukocyte Biology*, vol. 54, pp. 283-8.
- Leigh, J., Wang, H., Bonin, A., Peters, M. & Ruan X. 1997, 'Silica induced apoptosis in alveolar and granulomatous cells in vivo', *Environmental Health Perspectives*, vol. 105, S5, pp.1241-5.
- Leigh, J., Wang, H., Bonin, A. & Peters, M. 1998, 'Persistence of silica-induced inflammation is related to delayed occurrence of apoptosis in alveolar leucocytes', *Excerpta Medica*, vol. No. ?? Suppl. 53, pp. 520-5.
- Li, W., Kumar, R.K., O'Grady, R. & Velan, G.M. 1992, 'Role of lymphocytes in silicosis: Regulation of secretion of macrophage-derived mitogenic activity for fibroblasts', *International Journal of Experimental Pathology*, vol. 73, pp. 793-800.
- Majno, G. & Joris, I. 1996, 'Hemostasis and thrombosis', in *Cells, Tissues, and Disease: Principles of General Pathology*, eds G. Majno & I. Joris, Blackwell Science, Cambridge.
- Naum, C.C., Kaplan, S.S. & Basford, R.E. 1991, 'Platelets and ATP prime neutrophils for enhanced O2---generation at low concentration but inhibit O2--- generation at high concentration', *Journal of Leukocyte Biology*, vol. 49, pp. 83-9.
- Nachman R.L., Weksler B.B. 1980, 'The platelet as an inflammatory cell', in *The Cell Biology of Inflammation*, ed. G. Weissmann, Elsevier/North-Holland Biomedical Press, Amsterdam.
- National Occupational Health and Safety Commission (NOHSC) 1993, National Occupational Health and Safety Commission Report, Sydney.
- Newman, S.L., Henson, J.E. & Henson, P.M. 1982, 'Phagocytosis of senescent neutrophils by human monocyte-derived macrophages and rabbit inflammatory macrophages', *Journal of Experimental Medicine*, vol. 156, pp. 430-42.
- Phillips, D.R., Conley, P.B., Sinha, U. & Andre P. 2005, 'Therapeutic approaches in arterial thrombosis', *Journal of Thrombosis and Haemostasis*, vol. 3, pp. 1577-89.
- Piguet, P.F., Collart, M.A., Grau, G.E., Kapanci, Y. & Vassalli, P. 1989, 'Tumor necrosis factor/cachetin play a role in bleomycin-induced pneumopathy and fibrosis', *Journal of Experimental Medicine*, vol. 170, pp. 655-63.
- Piguet, P.F., Collart, M.A., Grau, G.E., Sappino, A.P. & Vassalli P. 1990, 'Requirement of tumor necrosis factor for development of silica-induced pulmonary fibrosis', *Nature*, vol. 344, pp. 245-7.
- Piguet, P.F., Versin, C., Grau, G.E. & Thompson, R.C. 1993, 'Interleukin 1 receptor antagonist (IL-1ra) prevents or cures pulmonary fibrosis elicited in mice by bleomycin or silica', *Cytokine*, vol. 5, pp. 57-61.
- Piguet, P.F. & Vesin, C. 1994a, 'Pulmonary platelet trapping induced by bleomycin: correlation with fibrosis and involvement of the b2 integrins', *International Journal of Experimental Pathology*, vol. 75, pp. 321-8.
- Piguet, P.F. & Vesin, C. 1994b, 'Treatment by human recombinant soluble TNF receptor of pulmonary fibrosis induced by bleomycin or silica in mice', *European Respiratory Journal*, vol. 7, pp. 515-8.
- Savill, J.S., Wyllie, A.H., Henson, J.E., Walport, M.E., Henson, P.M. & Haslett, C. 1989, 'Macrophage phagocytosis of aging neutrophils in inflammation', *Journal of Clinical Investigation*, vol. 83, pp. 865-875.
- Tsuji, T., Nagata, K., Koike, J., Todoroki, N. & Irimura T. 1994 'Induction of superoxide anion production from monocytes and neutrophils by activated platelets through the P-selectin-sialyl Lewis X interaction', *Journal of Leukocyte Biology*, vol. 56, pp. 583-7.

He Wang and James Leigh

Weksler, B.B. 1988, 'Platelets', in *Inflammation: Basic Principles and Clinical Correlates*, eds, J.I. Gallin, I.M. Goldstein & R. Synderman, Raven Press, New York.

White, J.G. 1993, 'Platelet secretory granules and associated proteins', *Laboratory Investigation*, vol. 68, pp. 497-8.

Correspondence to:

He Wang

Department of Public Health,

University of Adelaide

Adelaide, South Australia, 5005

AUSTRALIA

Email: he.wang@adelaide.edu.au



## Rural Health Systems Change

Peter Harvey

*The University of Adelaide Rural Clinical School, Spencer Gulf  
Rural Health School, Whyalla Norrie, South Australia*

*Much effort has been expended in recent years attempting to reform the Australian health system in order to deliver more efficient and effective systems of care for an ageing and increasingly chronically ill population. Rural health care systems in particular have been a focus of reform programs, and new initiatives such as University Departments of Rural Health, Regional Health Service structures and Commonwealth primary care initiatives have been designed to improve service provision and health status for rural people. However, with these attempts to reform the way rural communities understand and manage their health care, surprisingly little has changed in the day-to-day business of health care in rural and regional areas. Paradoxically, while rural communities have moved to embrace new farming technologies and environmental perspectives along with modern land management practices, revegetation and sustainable production systems, the same enthusiasm for change does not appear to have been kindled in relation to health system reforms. Rural communities, in terms of health care, are still using the equivalent of outmoded farming practices and other environmentally and economically unsustainable approaches to managing their affairs. Why might this be and what can be done to improve the current state of health reform in our rural and regional areas? The paper explores systems change in relation to health reform in rural communities and highlights several strategies for bringing about a functional synthesis of research and health service practice to create a more effective health care system in rural South Australia.*

**Key words:** Rural Health System Reform; Change; Sustainability; Efficiency; Integration; Collaboration

The extent of the health crisis in rural Australia is well documented, indicating that rural people suffer a greater burden of disease, and that their health status is more likely to be affected adversely by a wide range of risk factors, than is the health status of their metropolitan counterparts (Harvey 2003b, 2004; Smith 2004; Wilkinson & Blue 2002). The paper focuses on some of the more recent attempts to deal with emerging health care problems, such as increased rates of chronic disease, rising service provision costs, and recruitment and retention of health professionals. An overview of possible reasons for the failure of change initiatives in rural health systems (Harvey, 1996, 2000, 2002) is offered with a

description of key phenomena surrounding the business of rural health care.

### Recent Change Initiatives

In response to health systems stresses in recent years, rural communities across Australia have been the recipients of significant funding to support a range of new primary health care initiatives. Much of this funding, additional to normal recurrent budgets in the health system, has been allocated to facilitate change and development through demonstration and research projects across South Australia in both mainstream and indigenous health systems:

*Considerable effort and money has been invested in rural and remote health in Australia since it emerged as an identifiable field of activity some fifteen years ago. There has been significant investment in a rural general practice strategy; in rural and remote academic infrastructure through the University Departments of Rural Health and Rural Clinical Schools; some additional funding for regional and Aboriginal health services and financial support for advocacy groups and rural professional associations. Have there been good returns for those investments? What has been achieved? What are the continuing challenges?* (National Rural Health Alliance 2004).

Further, programs such as those outlined below have been developed to enhance rural health care systems:

- Council of Australian Governments (COAG) coordinated care trials
- More Allied Health Services (MAHS)
- Enhanced Primary Care (EPC) funding for GPs and allied health services
- Commonwealth Regional Health Service initiatives (CRHS)
- Quality Use of Medicines (QUM)
- Community packages for aged care services
- Indigenous Chronic Disease Self-Management pilot program (CDSM)
- Chronic Disease Self-Management (CDSM) programs - Sharing Health Care SA
- Chronic Disease Self-Management (CDSM) programs in indigenous communities.

In addition to the initiatives and resources listed above, funding has also been provided by the Commonwealth to establish the

combined University Departments of Rural Health (UDRH) across Australia. This new funding has led to substantial developments in chronic illness management in particular, but in spite of the considerable injection of resources in support of the change and development process in rural health systems, major impediments to change still exist.

The following phenomenological analysis of the change process in rural health attempts to outline some of the key factors involved in leading and managing the emerging rural health environment and offers some suggestions for improving collaboration and cooperation within and between the various components of the system that may contribute to the process of sustaining improved health outcomes for rural people.

### **The Culture of Rural Health Units**

Rural health units form a unique culture, and have achieved an importance and status comparable with that of the local churches, schools, or the police service in small rural communities. They are often holders of major recurrent budgets and are frequently, like schools and other formal institutions, among the largest employing bodies in the community. Because of this, and the importance and status of health professionals in rural communities, health units exercise power in the local culture.

Those who work within these structures are frequently the most qualified and influential professionals in the community. They often sit on other boards of management and on community management structures because of their level of education and expertise in management, finance, and health. The hospital and health units portray an image of dependability, strength, purpose and formality.

### **Leadership in rural health**

Many rural health unit leaders have spent a lifetime in the health system, usually working their way through the various levels of the service and grafting on, in the process,

a certain attitude to management in the health care arena. This 'attitude' might be characterised in terms of a leadership style that focuses predominantly on the management of resources rather than on the management of human capital and other related resources. It is difficult for rural health system leaders, having been nurtured in a certain school of thought, to confront these constraints and to work outside of this way of thinking.

### **The Service Funding Dilemma**

Private providers (general practitioners [GPs] and other practitioners) are paid a 'fee for service', so there are incentives in the system for providers to provide services without necessarily focusing on health outcomes to be achieved through service provision. One solution to this dilemma might be to change the way providers are remunerated for the work they do. An example could be to fund GPs, for instance, to do more preventive, early intervention, and patient management work to keep people out of hospital, and to reduce their reliance on medical and pharmaceutical interventions. The idea of providing linked funding for specific health related outcomes underpins the Commonwealth government's Enhanced Primary Care (EPC) program (Commonwealth Department of Health and Ageing 2002a, 2002b, 2002c; Commonwealth Department of Health and Family Services 1998), which is inexorably moving health service provision in Australia towards the application of structured protocols for the management of health care services for patients with chronic conditions such as diabetes and arthritis, as is the case now in other developed countries (Alessandrini et al. 2001; Dally et al. 2002; Light 1999; Zuckerman et al. 2002).

In the United States and other developed countries, those who manage health systems, health insurance schemes, and other funding bodies providing health services are recognising a new imperative to manage this major social resource in a more

effective and efficient manner in order to improve the return on these resources for the individual and the community generally (Robinson 2004). No longer can health care remain as an uncapped commodity in modern societies. It is far too valuable a resource to allow major inefficiencies to inflate the cost of providing comprehensive health care services. As Bodenheimer et al. (2002, p. 2470) argue in relation to new trends in patient self-management, for example, there is no longer a question about whether we manage things differently, but how we manage to do that.

Hospitals in particular are funded through the 'casemix' formula for admissions in various illness categories. If hospital managers initiate processes in the community to prevent or mitigate admissions and clinical costs, they might lose funding and ultimately find themselves unable to fund even basic clinical care. An example of this is the reducing capacity of small rural hospitals to maintain clinical services like obstetrics and the need for the State to keep many small hospitals open at great cost to operate virtually as aged care and long-stay facilities because other medical work can no longer be carried out in these facilities. Many small rural hospitals are expensive monuments to earlier days when they were staffed to carry out major medical procedures and when the focus of health care was predominantly acute services. Today the focus of health care is changing to preventive, earlier intervention programs, and for reasons of safety and the fear of litigation, the range of acute services provided in rural hospitals is reducing.

There appears, constantly, to be insufficient funds allocated through central authorities for health units to deliver what they need to deliver. New developments around the implementation of the Generational Health Review (GHR) in South Australia, for example (South Australian Department of Human Services 2003), and the advent of 'Population Based

Funding' may lead to more extensive change. However, even the advent of new population funding models based on aggregated population need and adjusted for disadvantage and remoteness will carry the rider that rural communities will need to realise efficiencies in the management of their health services. Sustainability, as is now being realised in the wider rural context, is also an imperative in the management of rural health services.

As local communities begin controlling their own population-based funding they will need to make their own changes to meet funding allocations whereas today they enjoy relative insulation from this harsh economic reality and look to outside agencies such as state and federal governments for direction and support. Population funding might not only give rural communities funding freedom and flexibility at the local level, but also it could provide the responsibility that goes with this freedom (Harvey 2005). Communities themselves, not unknown public servants, will then be responsible for their future business management decisions. While a positive outcome in terms of ownership and self-determination, this development would also bring new difficulties (Harvey 2001, 2003a).

#### *Service duplication and competing interests*

Historically, numerous funding mechanisms for health service resources have emerged. Hospital and allied health services are funded directly by state government while the Commonwealth government funds private service provision through the Medical Benefits Schedule (MBS) and Pharmaceutical Benefits Scheme (PBS). In addition, the Commonwealth also funds a range of new initiatives such as Divisions of General Practice to support GPs, research and development activities, the relatively new Commonwealth Regional Health Services, and Aboriginal Health Services. Many of these systems overlap resulting in competing activities and service

duplication. At best these elements can be coordinated by local groups, but at worst they run unchecked. Divisions of General Practice, for example, have from time to time become de facto service providers, being funded to introduce mental health services, more allied health services (MAHS) and the EPC program. The Divisions of General Practice were originally established as a professional resource for GPs, not as service providers.

Currently the new care planning and Enhanced Primary Care (EPC) initiatives from the Commonwealth, while offering increased resources for communities which can organise well enough to tap them, also present the possibility that privately run primary care teams could emerge to duplicate, compete with, and ultimately perhaps replace struggling, state-based allied health services. This might be part of the declared Commonwealth intention to 'take a greater interest in the direct funding of health services in the states' (The Australian Federal Health Minister, Mr Tony Abbott, speaking at a luncheon forum in Whyalla, South Australia, 16 June 2004).

People who work in rural health units, from managers, to clinicians and maintenance workers either come from or tend to become attached to small communities and many are not able easily to move place of employment. Hence, there is limited opportunity to bring in external staff, as existing permanent employees remain as stable members of the community. The task of revitalising, refreshing and re-training long-term locally based staff, therefore, becomes crucial for rural health units.

Rural communities still need to address, as do all communities, the necessity for lifelong learning and re-skilling. There is no 'qualification for the whole of life' now and although living in rural communities carries its disadvantages (Anderson & Thomson 2002; Simmons & Hsu-Hage 2002; Wakerman & Lenthal 2002), rural people will need to come to grips with the

challenge to up-skill, re-train and re-think about the way they live and work. There is a price to be paid for living in rural communities and social justice frameworks and principles of fair play might not protect rural people in the future to the extent that they might expect or hope (Lockie 2000; Smith 2004)

In addition, the problem of attracting to and retaining new professionals in remote regions is still possibly one of the most difficult challenges facing rural communities. This is exacerbated now that individuals are making decisions about where and how they work based on a new range of personal priorities; priorities that do not always rank income as the most important criterion when selecting where to work (Florida 2003).

Fear of the unknown can be a pronounced problem in rural health systems, as is the belief that external people and ideas should not disproportionately inform local practice. There is a perception among rural people that external agencies and individuals do not live in or understand rural communities and that they should not presume to tell rural people how to arrange their business. Rural communities are fiercely independent and they resent the intrusion into their culture of ideas originating from what they might perceive to be a city culture. This is epitomised in the notion that families may need to live for generations in a rural community before they are classed as 'locals'. Smith reminds us of this phenomenon when she writes:

*There is often distrust and suspicion of newcomers, who can be defined as those who have not been born in the town, or do not have their family name on a plot at the cemetery (Smith 2004).*

Being independent, rural people do not necessarily embrace the notion of teamwork and collaboration, and might be reluctant to share information about how they do or have done things. There might also be a belief among health service managers that research and subsequent policy proposals

might not contribute much of real value to health service management:

*As Donald Horne has noted in The Lucky Country there is an impatient optimism in the Australian temperament which makes for an intolerance of carefully thought out proposals (Conway 1985, p. 36).*

This idea has translated into a kind of 'muddling through' in rural Australia specifically where practical measures are valued above intellectual approaches to problem solving or systems change.

### The Way Forward: Rotation Programs for Health Service Leaders

There are initiatives, such as newly established University Departments of Rural Health (UDRH), that have the potential to make contributions to regional health service development if potential differences between managers of health services and academics are overcome. Health units might ignore the potential for university departments to become effective business partners, and universities need to be able to convince managers of rural health units of their value in training and research to the health system. For a synthesis between these two groups to be realised, a way forward needs to be found.

One way forward might be for the newly established academic and training frameworks to create opportunities for leaders of the two worlds to rotate their roles and experiences and in the process to learn from each other. UDRH structures have been established in the bush because practitioners have been unable to gain access to ongoing professional development and because it has been, and is becoming, more difficult to attract new health professionals to work in the rural sector.

### Mentoring

A GP mentoring program developed in one UDRH has resulted in GPs partnering with researchers to develop research, writing, and grant application skills. Each leader in a

health unit would benefit by being matched to a researcher or educator in the university sector. This would not only support ongoing study and professional development, but also provide a mentoring relationship that would serve to inform both through the process of regular interaction, discussion, problem solving, and publishing.

#### **Coordination and funds pooling**

As discussed above, the competing interests in the public service provision arena can lead to duplication and inefficiency. It makes sense for small health services to pool their resources and to deal directly with the Commonwealth in setting budgets and planning priorities at the local level. This is potentially the case with the new commonwealth Regional Health Service and Multi-purpose Services (MPS) arrangements in some areas. At the very least, such a direct fund pooling approach would see rural communities funded more equitably on the basis of need and the cost of service provision rather than, as is the case currently, on the basis of fees paid for services provided. This is inequitable as there are insufficient rural providers to provide all the services communities require. Some rural communities therefore lose revenue as in the case of GP service provision where GPs simply cannot see enough patients, on a fee for service arrangement, to ensure service parity between rural and metropolitan patients who have similar health service needs.

The potential to pool health related resources, avoid cost shifting and service duplication and fund populations on the basis of need is a much more equitable way to approach rural funding, provided, of course, that communities have the financial and managerial expertise to run such a system properly. The main risks in such a process are that the pooling coefficients used to calculate funding allocations might not be adequate in cash-out arrangements and that considerable testing of the model would be required to finetune it. As discovered during the recent Council of Australian

Governments (COAG) Coordinated Care Trials (Battersby et al. 2005; Centre for Health Care Evaluation (CHCE) 2000; Commonwealth of Australia 1999, 2001; Harvey 2000), this would be expensive, as would the provision and maintenance of the necessary information technology (IT) systems to monitor and report on utilisation costs and resultant health outcomes for individuals and populations. Another important factor to consider would be the ability of organisations to manage for capital development and facility renewal over time. Currently, the cost of maintaining infrastructure is managed at a state level and is often politically driven.

These potential problems notwithstanding, locally managed fund-pooling is a promising option, however, and could be seriously explored by leaders of smaller rural communities as a way of ensuring the provision of more services in a more efficient manner. This would avoid the pitfalls of the current three and four tiered funding devolution model, in which considerable resources are consumed in the process of distributing and managing these resources.

#### **Communication**

In addition to a mentoring program for all senior health service staff, the universities in rural areas could invest in the services of a facilitator for communication between the various agencies and bodies for which the university sector may be of benefit in one way or another. This is not simply a matter of having people coordinating and promoting activities across the country, of publicising conferences and workshops, but of being involved with health unit managers in their work to understand what they need in order for them to do their job more systematically. The burgeoning UDRH sector could then be in a better position to mesh its activities with those of the health services in rural communities and, in the process, bring the functions of both organisations closer together. Such a communication strategy would provide a

connection between the two worlds, enabling a dialogue between them out of which could grow mutual respect, beneficial relationships, research work, teaching and learning; a genuinely collaborative relationship that would build the capacity and skills of both sectors of the health system.

### Conclusion

Is it time to move beyond tentative primary health care programs such as chronic illness management and other short-term initiatives and into rural health system reform? For example, it has now been shown that there is much to be gained, both for patients and for the system, from new initiatives such as improved coordination of primary care services and initiatives such as self-management programs for patients with chronic conditions (Bodenheimer et al. 2002; Fries & McShane 1998; Lorig et al., 1999; Lorig et al. 2001; PricewaterhouseCoopers 2005; Strong et al. 2005). Better management leads to improved patient health outcomes and can reduce demand for unplanned hospital and emergency services. Many admissions to rural hospitals requiring expensive services, in terms of infrastructure and staffing, could

be either prevented, or, if not prevented outright, patients could be managed more effectively in the community as part of a wider primary health care program. These efficiencies, if achieved, might underpin the long-term survival of rural health services as they become more and more financially independent and sustainable into the future.

In addition, the development of a more productive intellectual culture in rural health services, through collaboration and sharing between key agencies, as well as leading the wider community towards lifelong learning and development, might ensure that rural cultures adjust favourably over time to the changing health care system. The survival of the rural health care culture, like the survival of the rural primary production systems, is linked to people in both realms doing business differently, perhaps even less traditionally than has been the case historically. Modern farmers cultivate less so as to use less fuel and conserve and nurture the soil out of which their livelihood grows (Diamond 2005). Such strategies are equally applicable to the way rural communities might approach the task of building and sustaining their health care industries.

### References

- Alessandrini, E.A., Shaw, K.N., Bilker, W.B., Perry, K.A., Baker, M.D. & Schwartz, D. F. 2001, 'Effects of medical managed care on health care use: Infant emergency department and ambulatory services', *Pediatrics*, vol. 108, no. 1, pp. 103-110.
- Anderson, I. & Thomson, N. 2002, 'Health of indigenous Australians: A rural perspective', in *The New Rural Health*, eds D. Wilkinson & I. Blue, Oxford University Press, Melbourne.
- Battersby, M.W., McDonald, P.J., Frith, P.A., Harvey, P.W., Pols, R.G., McGowan, C. H. et al. 2005, 'Health reform through coordinated care: SA HealthPlus', *British Medical Journal*, vol. 330, 19 March, pp. 662-5.
- Bodenheimer, T., Lorig, K., Holman, H. & Grumbach, K. 2002, 'Patient self-management of chronic disease in primary care', *Journal of the American Medical Association*, vol. 288, no. 19, pp. 2469-75.
- Centre for Health Care Evaluation (CHCE) 2000, *Evaluation of SA HealthPlus: Coordinated Care Trial*, Final Report, March, Flinders University, Adelaide, South Australia.
- Commonwealth Department of Health and Ageing 2002a, Care Planning Document, <[www.health.gov.au/hsdd/primcare/enhancpr/newmbs/pdf/healthst.pdf](http://www.health.gov.au/hsdd/primcare/enhancpr/newmbs/pdf/healthst.pdf)>
- Commonwealth Department of Health and Ageing 2002b, Enhanced Primary Care: Medicare Benefits Items, <[www.health.gov.au/epc/index.htm](http://www.health.gov.au/epc/index.htm)>
- Commonwealth Department of Health and Ageing 2002c, Health Assessment Document,

- <[www.health.gov.au/hsdd/primcare/enhancpr/newmbs/healthas.htm](http://www.health.gov.au/hsdd/primcare/enhancpr/newmbs/healthas.htm)>
- Commonwealth Department of Health and Family Services 1998, *Changing the Future through Partnerships*, General Practice Strategy Review Group, Commonwealth Government Publications Unit, Canberra.
- Commonwealth of Australia. 1999, *The Australian Coordinated Care Trials: Background and Trial Descriptions*, Commonwealth Department of Health and Aged Care, Canberra.
- Commonwealth of Australia 2001, *The Australian Coordinated Care Trials: Final Technical National Evaluation Report on the First Round of Trials, July 2001*, Commonwealth Department of Health and Aged Care, Canberra.
- Conway, R. 1985, *The Great Australian Stupor: An Interpretation of the Australian Way of Life*, Sun Books - Macmillan Australia, Melbourne.
- Dally, D.L., Dahar, W., Scott, A., Roblin, D. & Khoury, A.T. 2002, 'The impact of a health education program targeting patients with high visit rates in a Managed Care Organization', *American Journal of Health Promotion*, vol. 17, no. 2, pp. 101-11.
- Diamond, J. 2005, *Collapse: How Societies Choose to Fail or Survive*, Allen Lane - Penguin, London.
- Florida, R. 2003, *The Rise of the Creative Class: And How It's Transforming Work, Leisure, Community and the Everyday Life*, Pluto Press, Melbourne.
- Fries, J. & McShane, D. 1998, 'Reducing need and demand for medical services in high-risk persons: A health education approach', *Western Journal of Medicine*, vol. 169, no. 4, pp. 201-7.
- Harvey, P.W. 1996, *Eyre Regional Health Service Needs Analysis Report*, Port Lincoln, SA, ERHS Board, Eyre Peninsula South Australia.
- Harvey, P.W. 2000, Coordinated Care Trials and Change in Rural Health Systems, *Australian and New Zealand Journal of Public Health*, vol. 24, no. 2, pp. 217-8.
- Harvey, P.W. 2001, Preventive Social Health Programmes: Are They Australia's Answer to Rising Health Care Costs in Rural Communities? *Australian Journal of Rural Health*, vol. 9, no. 6, pp. 293-6.
- Harvey, P.W. 2002, 'Sustainable population health: A pressing priority for community wellbeing', *Environmental Health*, vol. 2, no. 3, pp. 66-74.
- Harvey, P.W. 2003a, 'Managing health care in Australia: Steps on the Health Care Roundabout', *Australian Journal of Primary Health*, vol. 9, nos 2&3, pp. 105-108.
- Harvey, P.W. 2003b, 'The politics of public sector change', *Australian Family Physician*, vol. 32, no. 5, pp. 373-5.
- Harvey, P.W. 2004, 'Tantalus and the tyranny of territory: Pursuing the dream of parity in rural and metropolitan population health outcomes through effective primary health care programmes', *Australian Journal of Primary Health*, vol. 10, no. 3, pp. 83-8.
- Harvey, P.W. 2005, 'Approaches to population health care: The emerging context', *Australian Journal of Primary Health*, vol. 11, no. 2, pp. 45-53.
- Light, D.W. 1999, 'Good managed care needs universal health insurance', *The Annals of Internal Medicine*, vol. 130, no. 8, pp. 686-9.
- Lockie, S. 2000, 'Crisis and conflict: Shifting discourses of rural and regional Australia, in *Land of Discontent: The Dynamics of Change in Rural and Regional Australia*, eds B. Pritchard & P. McManus, University of NSW Press, Sydney.
- Lorig, K., Sobel, D., Stewart, A., Brown, B., Bandura, A., Ritter, P., et al. 1999, 'Evidence suggesting that a chronic disease self-management program can improve health status while reducing hospitalization: A randomized trial', *Medical Care*, vol. 37, no. 1, pp. 5-14.
- Lorig, K., Sobel, D.S., Ritter, P.L. & Hobbs, M. 2001, 'Effect of self-management programs on patients with chronic disease', *Effective Clinical Practices*, vol. 4, no. 6, 256-62.
- National Rural Health Alliance 2004, Evaluating 15 years of investment, Editorial, *Party Line: National Rural Health Alliance Newsletter*, December, pp. 3-4.
- PricewaterhouseCoopers 2005, *Sharing Health Care Initiative*, Final Report of the National

- Evaluation, Commonwealth Department of Health and Ageing, Sydney.
- Robinson, J. (2004). Business opportunities in transforming health care: A conversation with William W. McGuire. *Health Affairs*, 23(6), 114-121.
- Simmons, D. & Hsu-Hage, B. 2002, 'Determinants of health, disease and disability: Differences between city and country, in *The New Rural Health*, eds D. Wilkinson & I. Blue, Oxford University Press, Melbourne.
- Smith, J.D. 2004, *Australia's Rural and Remote Health: A Social Justice Perspective*, Tertiary Press, Melbourne.
- South Australian Department of Human Services 2003, *The Generational Health Review*, Department of Human Services, Adelaide.
- Strong, K., Mathers, C., Leeder, S. & Beaglehole, R. 2005, 'Preventing chronic diseases: How many lives can we save?', *The Lancet*, pp. 1578-1582.
- Wakeman, J. & Lenthal, S. 2002, 'Remote health', in *The New Rural Health*, eds D. Wilkinson & I. Blue, Oxford University Press, Melbourne.
- Wilkinson, D. & Blue, I. eds 2002, *The New Rural Health*, Oxford University Press, Melbourne.
- Zuckerman, S., Brennan, N. & Yemane, A. 2002, 'Has Medicaid managed care affected beneficiary access and use?', *Inquiry: The Journal of Health Care Organization Provision & Financing*, vol. 39, no. 3, pp. 221-42.

Peter Harvey  
The University of Adelaide Rural Clinical School  
Spencer Gulf Rural Health School  
Nicolson Avenue  
Whyalla Norrie, SA, 5608  
AUSTRALIA  
email: peter.harvey@unisa.edu.au



# Environmental Health Impact Assessment (EHIA) in the Western Pacific Region

J. T. Spickett and J. Graham

***WHO Collaborating Centre for Environmental Health Impact Assessment, Western Pacific Region, Division of Health Sciences, Curtin University of Technology, Western Australia***

*Environmental Health Impact Assessment (EHIA) is an evolving concept in the Western Pacific Region. The Region has been strengthening the health impact assessment component of the environmental impact assessment systems over the last few years in the face of emerging global environmental changes. Providing an integrated approach to EHIA using widely adopted social indicators as well as quantitative and toxicological tools will provide a realistic and pragmatic approach to delivering an effective evaluation system to the Region. This approach has been under development in the region.*

**Key words:** Health; Environment; Impact; Assessment

Environmental Health Impact Assessment (EHIA) is a process which combines Environmental Impact Assessment and Health Impact Assessment to evaluate the impact of a development, policy or plan on the environment and on health, and with necessary depth in both aspects to include qualitative and quantitative evaluation processes. Environmental Impact Assessment (EIA) is a well-established process to predict the impact of development projects on the environment. Health Impact Assessment (HIA) is a process that assesses health risks to people either directly or indirectly as a result of environmental conditions or hazards.

The EHIA process has been developed to enable HIA to be included as an integral component of the Environmental Impact Assessment (EIA) process. This process deals with health issues that can be quantified; such as level of pollutant exposure or symptomatic health reactions, as well as providing qualitative information obtained from community and social assessments.

Many countries in the Western Pacific region have EIA processes upheld in their legislation. However, when legislation was established, it was probably intended that the impact on *human health* would be included in these procedures. Although EIA has been in place in some Western Pacific countries for about 25 years, there still remains a lack of proper coverage of health impacts within the assessment. In recent years, there has been an increased expectation that greater consideration should be given to the impacts on health for development projects, and with the introduction of new development policies and plans, and/or changes to them.

As an evaluative process using an evidence-based interdisciplinary approach to determine potential adverse effects on health, EHIA uses a range of determinants of health, such as:

- Biological factors - for example, age, genetics, gender;
- Preconception and inutero exposure - for example, maternal nutrition and health during pregnancy;

- Personal behaviour and lifestyle - for example, diet, smoking, alcohol, exercise, risk taking behaviour;
- Psychosocial environment - for example, family structure, community networks, culture, social exclusion;
- Physical environment - for example, air, water, housing, transport, noise, waste disposal; and
- Socio-economics - for example, employment, education (Lock 2000).

#### *The Development of Environmental Health Impact Assessment*

The development of EHIA has been shaped by a number of international and regional influences in the last 15 years.

##### *International*

The report of the World Commission on Environment and Development, often referred to as the Brundtland Commission (World Commission on Environment and Development 1987), recognised that EIA, including public participation, was an important part of the promotion of sustainable development.

The World Health Organization is concerned with the health impact of economic development projects and policy. Most often, these health impacts are secondary consequences of other environmental effects. The development of EIA procedures has provided a way to include and assess health impacts of new industrial development projects, but, in general, this has not been applied to new policies or other developments. The combination of environmental and health impact assessment procedures as an integrated approach should lead to the identification of alternative policies or projects which will have less adverse effects

on human health and the environment.

In 1987, a Working Group of the WHO (World Health Organization 1987) established four basic principles to assist in the incorporation of issues relating to the protection of human health into the EIA process. In 1989, the WHO First European Conference on Health and the Environment unanimously approved the European Charter on Environment and Health (World Health Organization 1989). The Charter recognises that every individual is entitled to an environment conducive to the highest attainable level of health and wellbeing; it underlines the shared responsibilities of individuals, public authorities and economic sectors of society. The Rio Declaration, introduced in 1992, aims to establish new and equitable global strategies to protect the environmental and developmental system. The role of EIA is covered in Principle 17 of this decree (United Nations 1993).

##### *Regional*

In the Western Pacific region, the National Health and Medical Research Council in Australia (National Health and Medical Research Council 1994) published a report in 1984 on Environmental Impact Assessment and Health Impact Assessment (HIA). The report discusses the political and administrative contexts in which they operate, and the technical and methodological issues that need to be considered. The report emphasises that HIA should not be a parallel process to EIA, but should be integrated into an overall *Environmental and Health Impact Assessment* process. The report identifies sets of principles that reflect the main concerns in the implementation of EHIA. Subsequent to this document, the Australian Government established the National Environmental Health Council (enHealth Council) as a peak environmental health advisory group for Australia, which has the responsibility for the provision of national leadership and implementation of the National

Environmental Health Strategy. The New Zealand government adopted a similar approach in regards to HIA in 1998 (New Zealand Public Health Commission 1998). With support from the WHO, the Philippines' government published a National Framework and Guidelines for EHIA in 1997 to support EHIA processes (Environmental Health Service 1997).

### ***General Issues in the Development and Implementation of EHIA in the Western Pacific Region***

The Western Pacific Region (WPR) comprises a range of countries, which have diverse political and operational issues affecting each country within the region. There is a need to provide an integrated approach that incorporates both social and scientific tools presenting an EHIA strategy that can be easily adapted and applied, and is capable of sustainability in each relevant country.

Countries in the WPR have undergone rapid changes in urbanisation and industrialisation since the 1980s. These changes were brought about by shifts in economic policy, which often encouraged direct foreign investment in their countries. The impact of this development has generally resulted in positive changes in the socio-economic status of the population. But development has also caused negative environmental changes leading to adverse health impacts on the population, especially children, women and the elderly.

Unfortunately, many developing countries in the WPR have limited capacity adequately to assess and manage health impacts resulting from rapid environmental changes, and subsequently provide timely information for policy decision making. They also lack appropriate legislation, institutional capacity and human resources to monitor, mitigate and control the health impacts of environmental hazards.

Since the early 1990s, WHO has collaborated with several countries in the Region to strengthen the health impact

assessment component of the environmental impact assessment systems in place. Over the last few years, WHO has collaborated with several countries in the region, such as Malaysia, Papua New Guinea and Mongolia, to develop HIA processes for environmental developments that pose potential health hazards in these countries, and which have had knock-on effects addressing the health impacts of emerging global environmental changes.

The concept of EHIA and evolving methodologies have since been introduced into countries where HIA was not previously considered part of the EIA process; namely in the Philippines, Malaysia, Papua New Guinea, Solomon Islands, China, Mongolia, Laos, Cambodia, and Vietnam. These countries have different government structures and processes used to introduce and implement the concept. Progress to date has been varied, with only the Philippines, Malaysia, Papua New Guinea, Australia, New Zealand, and Mongolia taking further steps to develop the health impact assessment component as part of the environmental impact assessment procedures. The only Australian state, at present, to have legislated HIAs into their EIA process, is Tasmania.

In general terms, countries in the region are striving to develop their economies, which has resulted in significant environmental changes. Although health status, as measured by increasing life expectancies and live births, is improving as the countries develop, there is still a need for a better understanding of the linkages between environmental factors and health outcomes. For example, there is a significant changing health burden in the region as a result of modern hazards such as pesticide exposure, transport accidents, and non-communicable diseases due to changing lifestyle practices. At the same time, many of the traditional hazards such as unsafe food, contaminated drinking water and disease vectors associated with communicable diseases still persist. A

double burden of disease and injury therefore exists.

Why are EHIA, or even EIA, difficult concepts and processes to conduct and enforce? In many of the countries in the WPR region, the EIA process occurs fairly late in the overall decision making process. For development projects such as a new industrial plant, the EIA is not undertaken until consideration has been given to the technical and financial feasibility of the development. By the time this has occurred, options such as performing a health impact assessment within an EIA have often already been eliminated from further consideration. This means that opportunities to protect the health and social amenities of the community have been removed, and so the focus then shifts to mitigation, rather than elimination of the potential hazards.

#### *Impediments to EHIA*

The four main reasons cited for a lack of inclusion of a health impact assessment in the EIA process, as identified by Steinemann (2000), are:

1. Analytical complexity - Factors that make HIA complex are associated with multiple cause and effects of health outcomes, uncertainty in the cause-effect relationship and variations in individual susceptibilities.
2. Methodology - There is a lack of established methodologies. This is linked to the first reason since a methodology is dependent on an understanding of cause/effect and dose/response relationships.
3. Legal requirements - Often legislation does not make explicit mention of the requirement for the inclusion of HIA. The inclusion of HIA could lead to more complex issues being raised.
4. Environment and health - There is a view that EIA applies to impacts on the environment and not on humans, so EIA has primarily focused on the potential impacts on the environment. This issue is not assisted by the lack of communication between those areas of government responsible for the environment and those responsible for health. Consequently, EIAs tend to focus on the impacts of the project on the environment without considering the potential impact on human health resulting from changes in the environment as a consequence of the development proceeding.

There are other reasons for the lack of consideration of HIA in the EIA process. Environmental Health Impact Assessment, as a concept, is very new to most countries, and as yet, there are no clearly established and accepted processes and procedures for EHIA internationally. Currently, there is an increasing appreciation of the need for health impact assessment to be integrated into the EIA processes in the Western Pacific Region, but before this can happen there is a need for the technical components of appropriate processes and procedures to be more clearly established.

Also on the technical side, there is the issue of intersectoral collaboration. Without this collaboration it is difficult to implement an interdisciplinary approach to health impact assessment within the EIA processes. In considering barriers to this collaboration, it is necessary to consider political and cultural factors affecting different countries.

While the technical and process aspects can present difficulties in the implementation of EHIA, there are also the cultural, political, economic, and social conditions, which have a complex role in EHIA. Although the concept of EHIA is utilised in many Westernised countries, consideration needs to be given to the cultural, political, economic, and social

norms and expectations that prevail in countries where Westernised practices are not likely to be viewed as an appropriate model.

#### *Specific issues on EHIA in the region*

The Western Pacific Region has undergone rapid changes in urbanisation and industrialisation since the 1980s. These changes have been brought about, in part, by strategies in economic policy, which often encourage foreign investment in their respective countries. These economic changes have also caused a number of negative environmental changes leading to adverse health impacts on people, especially children, women and older persons. To date, there has been limited capacity in undertaking adequate assessment and management of health impacts resulting from rapid environmental changes in many countries of the WPR.

Although environmental threats that impact on human health are well known, the health component of an environmental impact assessment to support EHIA is lacking for a number of reasons in this Region. In addition to those limitations listed above, other reasons include:

- financial constraints;
- an insufficient capacity of health professionals familiar with health research methodology and project management to undertake a health impact assessment;
- a deficiency of appropriate equipment for laboratory work;
- the lack of well-defined scientific evidence on the impact of specific environmental hazards on human health; and
- the ability of many government and non-governmental health professionals to work, and collaborate with other professionals.

This last point is crucial to ensuring proper environmental health impact assessment is undertaken to protect the current and future health of populations.

Recognising many of these barriers, WHO has supported several countries in the Region in strengthening the health component of the environmental impact assessment systems in the past ten years. More recently, WHO has collaborated with Vietnam, Fiji and Mongolia in conducting a health impact assessment of priority environmental hazards in these countries, and has begun to address health impacts of emerging global environmental changes.

Health departments in the participating countries have established and strengthened infectious disease surveillance activities that monitor the impacts of environmental hazards on human health. Some countries have established a comprehensive monitoring and surveillance system to monitor environmental health-related illnesses, morbidity and mortality, which are important in establishing the relationships between health and environmental hazards in the countries.

Also, the functions and roles of research institutions, universities, and other private sector institutions have supported the EHIA process by providing services in the capacity of:

- human resources development;
- conducting health impact assessments;
- providing laboratory support;
- participating in monitoring and surveillance activities; and
- providing consultancy in the mitigation and control of environmental hazards.

Recently, a high level meeting on health and the environment in ASEAN and East Asian Countries was held in late 2005

(*Proceedings of the Second High-level Meeting on Health and Environment in ASEAN and East Asian Countries 2005*). This meeting was a regional initiative on environment and health launched by ADB, WHO and UNEP, and provided a forum for review of environmental health issues in the Asia Pacific region. A result of this meeting is a draft Charter for environment and health issues that takes into account capacity building, transferring knowledge from other regions, making use of existing institutional bases and existing inter-governmental processes, and following an inclusive approach involving more countries and agencies. The Charter is a vehicle where countries can develop individual master plans for environment and health policies, developing EH risk assessment tools and other assessments particular to the respective country.

#### Developments in EHIA from Selected Countries

Up until 1992, efforts in the Western Pacific Region were spent strengthening national capacities in implementing EIA. Then from 1992-1999, the focus was towards integrating health components into EIA through EHIA activities. Since then, the focus has been on strengthening the capacity in EHIA and management of the impact of development changes, especially on specific developmental activities (Rozlan 2003).

##### *The Philippines*

There have been several cases in the Philippines where insufficient attention had been paid to the potential impact on human health of development projects. In response to concerns raised from the Department of Health, and with support from WHO, a *National Framework and Guidelines for EHIA* was developed and published in 1997. The document describes the EIA system in the Philippines and provides a general framework for EHIA. The framework

provides guidance on procedures to assess development projects and for the assessment of current situations where environmental hazards are present. In cases where there is no requirement for the preparation of an EHIA, information is provided for the development of a health and safety management plan.

##### *Mongolia*

Unique pressures on the environment and health of this population have resulted from the growing population, urbanisation processes and industrial development occurring in this country.

In conjunction with WHO, providing technical assistance, a recent study was conducted by the Ministry of Health in Ulaanbaatar assessing the health impacts of environmental hazards. The objective of the study was to determine the relationship between environmental pollution (air and water) and the impacts on the morbidity of the population. The study was carried out using retrospective environmental and health data. Air and water quality data were collected and analysed to determine bacteriological and chemical content and contamination levels. Although the main outcome from the study was to improve environmental health conditions of the capital city, the results will provide a useful reference tool and data for future EHIA activities in other areas of Mongolia as it develops.

##### *China*

Historically, the focus on EIA in mainland China, that is, pollution of air, water and soil, was at the expense of the consideration of wider environmental, social and health impacts inherent in the EHIA process (Wanga, Morgan & Cashmore 2003). Environmental protection agencies were funded by many of the development-oriented local government administrations, resulting in a lack of consideration of alternatives in EIA processes and effective public participation. There is now much

greater awareness of the impact of environmental factors on the health of the population.

### Malaysia

There have been several recent developments in Malaysia where required HIA in EIA processes were incorporated into both the project development and the assessment of current situations where environmental hazards are present. The Twin Towers project is most notable, where issues such as traffic flow, noise, safety and health of the workers, food, accommodation, hygiene, and vector borne disease, such as dengue and waste management, were addressed. Another is the Hydroelectric Bakun Dam in Sarawak costing US \$4 billion, and situated on the Balui river affecting 10,000 indigenous people where it occupies over 69,640 hectares of land and virgin rainforest. Also included are hazardous waste incineration plants, the Kampong Bohol incinerator plants (in Kuala Lumpur) and sanitary landfills in Selangor (Rozlan 2003).

### Conclusion

Implementing health impact assessment within the environmental impact assessment process has provided a necessary component to the growing development in the Western Pacific Region. Some of the challenges associated with adopting rigid and narrowly focussed scientifically based procedures to assess risks to health from environmental conditions, is that they do

not take account of the changing societal and environmental conditions many of these countries possess.

For many developing nations within the WPR, the rapid pace of change and the proliferation of new potential risks require the adoption of effective and appropriate means to assess and manage the risks. It is not viable, or appropriate, to transfer an EHIA process with culturally entrenched values from one country to another. A lack of relevance to the reality of the situations in a developing nation will see the assessment mechanism lose the ability to deliver an effective evaluation.

In order to create an environment where a predictive and evaluative tool such as EHIA can be used to help assess all the impacts of development activities, legislation is also necessary. However, it must be remembered that legislation alone, regardless of how well drafted, will not be sufficient to ensure that such measures are used effectively. Some form of enforcement mechanism is invariably necessary to ensure that these measures are implemented to their full capacity and that, consequently, public health goals are achieved.

With rapid economic growth and urbanisation occurring in most of the WPR, targeting an effective collaboration between health and environment issues is a priority. The regional Charter initiative for the Asia Pacific region is a step in the right direction to facilitate development for a regional road map that includes EHIA as part of the larger picture in regional policy and planning initiatives.

### References

- Environmental Health Service 1997, *National Framework and Guidelines for EHIA*, Department of Health, Government of the Philippines, Manila.
- Lock, K. 2000, 'Health impact assessment', *British Medical Journal*, vol. 320, pp.1395-8.
- National Health and Medical Research Council 1994, *National Framework for Environmental and Health Impact Assessment*, Australian Government Publishing Service, Canberra.
- New Zealand Public Health Commission 1998, *A Guide to Health Impact Assessment*, New Zealand Public Health Commission, Wellington.
- Proceedings of the Second High-level Meeting on Health and Environment in ASEAN and East Asian Countries 2005*, 'Health and environment', Bangkok.

- Rozlan, I. 2003, *Health Impact Assessment in Countries of the WHO Western Pacific Region*, Western Pacific Regional Office, Manila.
- Steinemann, A. 2000, 'Rethinking human health impact assessment', *Environmental Impact Assessment Review*, vol. 20, pp. 627-45.
- United Nations 1993, *Agenda 21: Programme for Action for Sustainable Development*, United Nations, New York.
- Wanga, Y., Morgan, R. & Cashmore, M. 2003, 'Environmental impact assessment of projects in the People's Republic of China: New law, old problems', *Environmental Impact Assessment Review*, vol. 23, no.5, pp. 543-79.
- World Commission on Environment and Development 1987, *Our Common Future*, Academic Press, London.
- World Health Organization 1987, *Health and Safety Component of Environmental Impact Assessment*, Environmental Health Series, no. 15, World Health Organization, Copenhagen.
- World Health Organization 1989, *European Charter on Environment and Health*, First European Conference on Environment and Health, World Health Organization, Frankfurt.

Correspondence to:

J. T. Spickett

WHO Collaborating Centre for Environmental Health Impact Assessment, Western Pacific Region

School of Public Health

Curtin University of Technology

GPO Box U1987

Perth, Western Australia, 6845

AUSTRALIA

Email: J.Spickett@curtin.edu.au





## MAKE YOUR OWN ENVIRONMENTAL IMPACT.

An Environmental Health Officer is one of the most evolving and challenging roles in the Army and Air Force. As the person responsible for planning, co-ordinating and implementing the Australian Defence Force's environmental control strategies, it's your chance to have a really positive effect on your surroundings.

Working alongside other motivated professionals, you'll be part of overseas deployments to assist in environmental disaster areas, such as the Australian Defence Force's recent humanitarian operations in Banda Aceh, East Timor and the Middle East. You'll also manage food safety and water quality, perform epidemiological studies, pest and vector control and be a vital part of the nuclear, biological and chemical defence team. Both full-time and part-time Environmental Health Officer positions are available.

Working full-time, you'll receive a starting salary of \$52,200p.a. plus free health and dental care and subsidised accommodation. If you join part-time in the Army Reserve, you'll receive leadership and management training and any money you earn is completely tax-free. So for more information, call 13 19 01 or visit

[www.defencejobs.gov.au/graduate](http://www.defencejobs.gov.au/graduate) to complete an online application.

**GRADUATE OFFICER**  
**HAVE YOU GOT WHAT IT TAKES?**

Call 13 19 01 or visit [www.defencejobs.gov.au](http://www.defencejobs.gov.au)



# Faecal Source Tracking in Surface Waters: A Review of Faecal Indicator Microorganisms and Current Methods

W. Ahmed, R. Neller and M. Katouli

*Institute of Sustainability, Health and Regional Engagement,  
University of the Sunshine Coast, Queensland*

*Faecal indicator microorganisms have long been used by water quality industries to assess the quality of surface, ground and shellfish-harvesting waters. However, none of these indicators are universally accepted as they lack the desired characteristics of a suitable indicator. Further, they do not provide sufficient information regarding their source of origin. Over the past ten years, researchers have developed many phenotypic, genotypic and chemical methods to identify the sources of faecal contamination in waterways. However, there is no 'gold standard' method for faecal source tracking. This paper is a critical evaluation of faecal indicators along with the advantages and limitations of current source tracking methods.*

**Key words:** Faecal Contamination; Microbial Source Tracking; *Escherichia Coli*; *Enterococci*; Faecal Indicator Bacteria

Faecal contamination is believed to be one of the major causes for increased microbiological and nutrient loads in waterways (Lipp, Farrah & Rose 2001), and poor water quality results in the deaths of an estimated 5 million children annually (Watson 1996). Non-point sources (NPS) (Barnes et al. 2004; Griffith, Weisberg & McGee 2003) and/or point sources (PS) (Parveen et al. 1997) are both known to be potential sources of such contamination. Human enteric pathogens such as *Salmonella* spp., *Shigella* spp., hepatitis A and Norwalk viruses have been found in surface waters due to human faecal contamination (Barnes et al. 2004; Dombek et al. 2000). Wastewater from domestic and/or farm animals might further contribute pathogens such as *Escherichia coli* belonging to serotype O157:H7, *Cryptosporidium* spp. and *Giardia* spp. (Dombek et al. 2000; Hill & Sobsey 1999). Identification of major sources of such faecal bacteria, as well as potential pathogens in water, is therefore necessary to minimise the potential public health risks associated with such contamination.

Faecal indicator microorganisms have long been used by the water quality industry to indicate faecal contamination and the presence of potential pathogenic microorganisms. However, these indicators alone do not provide sufficient information regarding their source. Over the past ten years, microbiologists have developed new techniques, collectively known as microbial source tracking (MST) methods. These methods, along with various faecal indicators have been used to predict the sources of faecal contamination. This review will in the first instance provide an overview of current faecal indicators followed by a discussion of various MST methods, with regard to their advantages and limitations in ecological studies so as to predict the sources of faecal contamination in surface waters.

## Faecal Indicator Microorganisms

Faecal indicator microorganisms are biological agents, which have entered the water at the same time with faecal matter and therefore indicate the presence of potential pathogenic microorganisms.

However, to be an ideal candidate, an indicator microorganism should meet certain criteria such as it should be universally native to the intestine of humans and animals in large numbers, should not be pathogenic and the number should be higher than pathogens, should be easily isolated, enumerated and identified, should be resistant to a variety of environmental stresses, should survive long enough in the natural waters to be detected, should not multiply in the environment, and their presence should be associated with the presence of pathogenic microorganisms (Bitton 2005; NHMRC 1993).

The most commonly used indicator microorganisms are discussed below.

### Coliforms

Coliforms have long been used to assess the quality of recreational and shellfish-harvesting waters (Hagedorn et al. 1999; Hartel et al. 2002; Harwood, Whitlock & Withington 2000). These groups of bacteria include *E. coli* and several coli-like (coliform) bacteria, mainly belonging to the family Enterobacteriaceae, and are commonly found in the gastrointestinal (GI) tracts of warm-blooded animals (Harwood, Parrish & Wagner 1999; Meays et al. 2004). Over the past few years coliform dynamics have been examined (Barnes et al. 2004), and their value as an indicator has recently been questioned because these bacteria can also be derived from various other sources such as soil, decaying vegetation and industrial processes (Dombek et al. 2000; Hagedorn et al. 1999). In addition, their ecology and prevalence differ from pathogenic microorganisms (Craun et al. 1997; Desmarais, Solo-Gabriele & Palmer 2002). Finally, the sensitivity of these bacteria to environmental stresses is low compared to viruses and protozoans (Desmarais, Solo-Gabriele & Palmer 2002). These factors collectively limit their use as a standard indicator and, therefore, other bacteria such as *E. coli*, enterococci, *Bifidobacterium* spp.,

have been suggested as alternative indicators (Godfree, Kay & Wyer 1997).

### *E. coli*

*E. coli* has been considered 'the pioneer marker' as these bacteria colonise in the GI tracts of human and other warm-blooded animals in relatively high numbers (Baudisöva 1997; Fujioka 2001). *E. coli* possess several desirable criteria of an ideal indicator as mentioned earlier, such as not normally pathogenic, and easy to culture and detect, the concentration in receiving waters is much higher than those of pathogens (Edberg et al. 2000; Toranzos & McFeters 1997) and they may survive a prolonged period in natural environments under favourable conditions (Baudisöva 1997). However, it has also been reported that *E. coli* can replicate in pristine waters in tropical environments (Desmarais, Solo-Gabriele & Palmer 2002) and certain encapsulated strains of *E. coli* can cause blooms in surface waters even in the absence of faecal sources (Power et al. 2005).

### Enterococci

Enterococci, also known as faecal streptococci, are members of the genus *Enterococcus* (Ludwig et al. 1985). They can be easily distinguished from other faecal streptococci by their ability to grow at 10–45°C, at high pH (i.e. pH=9.6), and in medium with 6.5% NaCl. So far, 19 species have been included in the genus (Topp et al. 2003). The most common species of enterococci include *E. faecalis*, *E. faecium*, *E. durans*, *E. gallinarum* and *E. avium* among which *E. faecalis* and *E. faecium* are commonly found in humans (Scott et al. 2002). Enterococci have the ability to survive in the natural environment for lengthy periods under favourable conditions (Baudisöva 1997; Hagedorn et al. 1999; Kibbey, Hagedorn & McCoy 1978; Sinton, Donnison & Hastie 1993), are less numerous than faecal coliform in human faeces and rapid methods are available for their detection and identification. Several

epidemiological studies have shown a correlation between enterococci concentrations and swimming-associated gastrointestinal diseases in recreational waters (Cabelli et al. 1982, 1993; Chung et al. 1998; Fujioka 2001; Kay et al. 1994; Roll & Fujioka 1997). However, it has to be noted that enterococci might regrow in subtropical and tropical waters once they are released into the environment (Desmarais, Solo-Gabriele & Palmer 2002).

### *Bifidobacteria*

*Bifidobacteria* are anaerobic, Gram-positive enteric bacteria, which are considered as a potential faecal indicator due to their high abundance in human faeces relative to those of faecal coliforms (Evison & James 1974). One important advantage of *bifidobacteria* as an indicator of faecal contamination is that several species (i.e. *B. bifidum*, *B. adolescentis*, *B. infantis*) appear to be primarily associated with humans (Bitton 1999; Mara & Oragui 1983). The presence of *Bifidobacteria* in surface waters indicates that faecal contamination has occurred through human faeces (Beutin 1999; Rhodes & Kator 1999; Scott et al. 2002). Another key advantage of *Bifidobacteria* is that they do not replicate in the environment due to their strict growth requirements (Levin 1997; Mara & Oragui 1983). *Bifidobacteria* have the ability to ferment sorbitol and can be easily detected in sorbitol agar (Rhodes & Kator 1999). However, one disadvantage of these bacteria is that they do not survive in the environment for lengthy periods (Bernherd & Field 2000a; Carrillo, Estrada & Hazen 1985; Rhodes & Kator 1999) to be detected (Gilpin, Gregor & Savill 2002). The use of *Bifidobacteria* is also limited due to the difficulty in isolation and identification from environmental samples using traditional biochemical methods (Matsuki et al. 1999).

### *Clostridium perfringens*

*C. perfringens* are spore forming, sulphite-reducing, rod-shaped anaerobic bacteria

(Holt et al. 1994). These groups of bacteria are commonly found in the GI tract of warm-blooded animals and have been isolated from surface waters (Cato, George & Finegold 1986). Spores of *C. perfringens* are largely of faecal origin and comprise approximately 0.5% of the faecal flora (Sorensen, Eberl & Dicksa 1989). The advantage of using this bacterium is that unlike other indicator bacteria, they do not replicate in natural waters due to their strict growth requirements (Davies et al. 1995). Their spores are resistant to disinfection processes and environmental stresses (Bitton 1999). Due to this resistance, *C. perfringens* has been suggested as an indicator for the inactivation and removal of viruses in drinking water treatment (Payment & Franco 1993). It has also been reported that the number of *C. perfringens* correlates with human enteric viruses in surface waters (Fujioka & Shizumara 1995; Payment & Franco 1993). However, these bacteria may not be suitable for identifying recent pollution events because their persistence allows detection long after any pollution event (NHMRC 2003).

### *Bacteroides*

*Bacteroides* are anaerobic non-spore forming bacteria commonly found in the GI tract of warm-blooded animals including humans. The number of these bacteria is quite high in faeces, representing more than 30% of total human faecal flora (Moore & Holdeman 1974). One advantage of using such bacteria is that they do not replicate in the natural environment and their presence in natural waters indicates that recent contamination has occurred (Meays et al. 2004). Again, the use of these bacteria as an indicator is limited due to difficulties in isolation and identification. Further, they do not survive in the environment for lengthy periods (Sinton et al. 1998).

### *Bacteriophages*

Bacteriophages are viruses that infect *E. coli* found in human intestines. Bacteriophages

are generally found in large number in sewage and wastewater. Three groups of bacteriophages have been proposed as indicators. These include somatic coliphage (Hilton & Stotzky; Kott et al. 1974), male-specific RNA coliphage (Havelaar & Hogeboom 1984) and phages infecting *B. fragilis* (Grabow et al. 1995; Jofre et al. 1986; Tartera & Jofre 1987). It has been reported that coliphage lack host specificity. For instance, F<sup>+</sup> coliphages attack *E. coli* as well as other coliforms (Rhodes & Kator 1999). It has also been reported that F<sup>+</sup> coliphages and somatic coliphages can multiply in the environment and may act as a false indicator (Leclerc et al. 2000). A large volume of water samples needs to be analysed for isolation of this bacterium, which is not practical for routine monitoring (Leclerc et al. 2001).

#### Human enteric viruses

Enteric viruses infect the human GI tract and they are generally transmitted by the faecal-oral route (Bitton 1999). Over 100 different enteric viruses are found in the GI tract of humans. The acellular nature of viruses alters them to acquire resistance to environmental stresses and therefore survive in the natural waters for a lengthy period (Funderburg & Sorber 1985). Because of their high degree of host specificity, they could be regarded as excellent indicators of human faecal contamination (Sinton et al. 1998). However, while numerous types of human pathogenic viruses might be present in environmental samples, many of them are not detectable using conventional cell culture technique (Arraj et al. 2005). Further, cell culture assays are laborious, time-consuming and lack sensitivity for unequivocal detection of viruses (Baggi et al. 2001). It has to be noted that in recent years, molecular methods have been developed which are useful to detect viruses in environmental samples (Fong et al. 2005) and, therefore, the lack of specificity in faecal indicators could be avoided.

#### Microbial Source Tracking Methods

The currently used source tracking methods can be broadly categorised as microbial (Griffith, Weisberg & McGee 2003; Scott et al. 2002) and chemical methods (Seiler et al. 1999). Microbial methods can be further categorised as genotypic and phenotypic methods. Some of these methods have been further categorised as database-dependent methods, based on the hypothesis that phenotypic or genotypic characteristics of specific strains are associated with specific animals (Amor et al. 2000; Hartel, Summer & Segars 2003; Johnson et al. 2004; McLellan 2004). On the basis of this hypothesis, a reference database is assembled of either the genotypic or phenotypic profile of the indicator bacteria from several known host groups and classified to host categories using a variety of statistical models such as discriminant, cluster and principal component analyses (Simpson, Santo Domingo & Reasoner 2003; Whitlock, Jones & Harwood 2002). The database is then used to compare with profiles obtained from the same indicator bacteria found in receiving waters, presumed to be contaminated with the same faecal indicator bacteria. In this manner, the source(s) of unknown environmental isolates can be identified, or at least predicted, based on the similarity to the database. Genotypic database-dependent methods distinguish between sources of faecal contamination by identifying patterns in the genetic makeup of bacterial isolates and matching them with the database from known host groups, while phenotypic database-dependent methods rely on growth patterns produced when bacterial isolates are subjected to a given test system. In contrast, certain methods do not require development of a database and are referred to as 'database-independent methods'. These include host specific polymerase chain reaction (PCR) (Dombek et al. 2004; Parveen et al. 1999), terminal-restriction length fragment polymorphism (t-RFLP) (Bernherd & Field 2000a, b), and toxin gene biomarkers (Olson, Khatib & Chern 2002) which differentiate

between sources by identifying the presence of genetic markers unique to the faecal bacteria of the targeted host groups. Database-independent methods mainly operate at the population level rather than the isolate level. Certain database-independent genotypic methods target viruses that occur in human faeces while not found in animals and include those that detect human enteroviruses and adenoviruses or F<sup>+</sup> coliphage, a virus that infects *E. coli*. Chemical methods such as detection of caffeine and faecal sterols analysis have also been used to detect the source(s) of faecal contamination in surface waters (Leeming et al. 1996).

#### Database-dependent genotypic methods

##### Pulsed field gel electrophoresis

Pulsed field gel electrophoresis (PFGE) is considered as being the most popular method for typing bacterial isolates (Olive & Bean 1999). This method is highly discriminatory and stable for analysis of numerous species of bacteria (Barbier 1996; Grundmann et al. 1995; Murray et al. 1990; Poh, Yeo & Tay 1992; Saulnier et al. 1995; Schlichting, Branger & Fournier 1993). In this method, DNA fingerprints are generated by *in situ* detergent-enzyme lysis and digestion with infrequently cutting restriction endonucleases. The digested bacterial plugs are then subjected to electrophoresis. The pulsed-field allows clear separation of very large molecular length DNA fragments ranging from 10 to 800 kb. The electrophoresis patterns are visualised following staining of the gels with a fluorescent dye. A reference database is then developed according to bacterial types and compared to unknown environmental isolates.

##### Repetitive extragenic palindromic - PCR

Repetitive extragenic palindromic (rep)-PCR targets repetitive extragenic palindromic elements to compare bacterial genome diversity (Carvalho de Moura, Irino

& Vidotto 2001; Dalla-Costa et al. 1998; Johnson et al. 1998; Koeuth, Versalovic & Lupski 1995). This method uses PCR and specific primers such as BOX primer (i.e. 154 base-pair [bp]), rep primer (35-40 bp) or enterobacterial repetitive intergenic consensus (ERIC primer) (124-127 bp) to amplify specific portions of the microbial genome (Versalovic, Koeuth & Lupski 1991) followed by electrophoresis, staining and visualising band pattern for each genomic DNA. This method is based on the hypothesis that isolates having indistinguishable banding patterns can be regarded as genetically identical (i.e. genetically related). The banding patterns are stored in a database and compared with unknown environmental isolates to identify the source.

##### Ribotyping

Ribosomal ribonucleic acids (rRNA) are an integral part of all living cells, and the genes coding rRNA tend to be highly conserved (Farber 1996). In this method, DNA is isolated from bacterial isolates and cut into fragments using one single restriction enzyme such as *HindIII* (Parveen et al. 1999) or a combination of two enzymes such as *EcoRI* and *pvuII* (Samadpour & Checowitz 1995). The resulting fragments are separated based on their molecular weight using gel electrophoresis. Hybridisation with a labelled DNA probe creates a pattern of the fragments, which are specific to each strain. The fingerprints are then analysed by discriminant analysis and compared to a reference database.

#### Database-dependent phenotypic methods

##### Antibiotic resistance analysis

Antibiotic resistance analysis (ARA) has been used extensively for MST studies using *E. coli* and/or enterococci. (Geary & Davies 2003; Graves et al. 2002; Hagedorn et al. 1999; Parveen et al. 1997; Wiggins et al. 2003). Antibiotics are used to prevent and

treat infections in humans and domestic animals as well as to promote growth in animals. Microorganisms develop resistance to antibiotics to which they are regularly exposed. The ARA method is based on the hypothesis that bacteria present in the intestine of different animals, subjected to different types and concentrations of antibiotics, would result in host-specific resistance profiles (US EPA 2005). ARA fingerprints of unknown environmental isolates can be compared to a host specific reference database of several known host groups.

#### *Carbon source utilisation*

Carbon source utilisation (CSU) has been recently used for MST (Hagedorn et al. 2003) and compares differences in the utilisation of several carbon and nitrogen substances by bacterial isolates. This method has been developed for species identification and can be used with the Biolog database to identify more than 2000 species of microorganisms. This method has also been extensively used for characterisation and identification of microorganisms in medical microbiology, soil and aquatic microbiology (Holmes et al. 1994; Noble & Gow 1998). This method is rapid and simple, requiring only a micro-plate reader to determine CSU pattern. For each bacterial isolate, it yields a fingerprint pattern, which is saved to a database and compared with the pattern of unknown environmental isolates.

#### *Biochemical fingerprinting*

Biochemical fingerprinting uses quantitative measurements of the kinetics of several biochemical reactions of bacteria in micro-titer plates with dehydrated substrates (Kühn et al. 1995; Möllby, Kühn and Katouli 1993). It is based on the hypothesis that bacterial isolates, belonging to the same clone, share identical metabolic properties, whereas isolates with different genotypes have differences in one or more of the measured metabolic processes, and thus will

show different activities in the reactions involved. For each bacterial isolate, it yields a biochemical fingerprint made of several quantitative data, which are used to calculate the level of similarity between the tested isolates. A reference database is developed from known host groups to be compared with unknown environmental samples (Ahmed, Neller & Katouli 2005b).

#### *Database-independent methods*

Database-independent methods are generally PCR-based and offer several advantages over database-dependent methods. For instance, these methods circumvent the need for the cultivation of bacterial isolates and the development of a reference database. PCR amplification of 16S rDNA from bacteroides has been used for MST studies (Bernherd & Field 2000a, b; Field, Bernhard & Brode 2003; Bernherd et al. 2003). Among others, reverse transcription PCR (RT-PCR) and quantitative PCR (qPCR) have also been used to detect viruses such as adenoviruses and enteroviruses in surface waters (Noble et al. 2003) which derive from humans and animals respectively (Jiang 2002; Ley, Higgins & Fayer 2002; Pina et al. 1998). Biomarkers, based on enterotoxin genes in *E. coli* have also been proposed as an alternative method (Olson, Khatib & Chern 2002). The advantage of such a method is that it targets clinically significant strains of *E. coli* rather than commensal strains found in the intestine.

### **Chemical Methods**

#### *Optical brighteners*

It has been reported that the laundry detergent compounds such as optical brighteners and ethylenediamine tetra acetic acid (EDTA) have been found in groundwater (Alhajjar, Harkin & Chesters 1990; Barber et al. 1998; Sargeant 1999). Optical brighteners can be used as a potential indicator of grey water discharge (Sargeant 1999). However, this method is

only suitable for PS identification. In addition, this method does not indicate public health risks that may be associated with failed on-site wastewater treatment systems.

#### **Caffeine and pharmaceuticals**

Caffeine and human pharmaceuticals have also been used as potential indicators of contamination of surface and ground waters by on-site wastewater treatment systems (Seiler et al. 1999). Caffeine is of anthropogenic origin and is found in beverages and many pharmaceutical products. It has been suggested that the presence of caffeine in the environment could indicate the presence of human sewage (Burkhardt et al. 1999). However, application of these compounds as indicators of contamination is limited because high concentration of these chemicals must be present in receiving waters to be detected. It has been reported that only 3% of ingested caffeine is excreted in the urine (Tang-Liu, Williams & Riegelman 1993). A dilution of more than 1:200 would make it difficult to detect (Sargeant 1999). Pharmaceutical substances such as pentobarbital, meprobamate, and phenisuximide are used to cure diseases (Seiler et al. 1999). These substances are also potential wastewater indicators and have also been detected in groundwater (Eckel, Ross & Isensee 1993).

#### **Faecal sterols**

Human and animal faeces contain sterols and stanols (a by product of sterols). The sterol profiles of human and animal faeces vary from each other due to different feeding habitat, gut flora and types of metabolism. Sterols such as 5 $\beta$ -stanols and coprostanol are dominant in human faeces and have not been naturally found in surface waters unless contaminated by human faeces (Venkatesan 1995). Similarly, animals such as cattle, sheep and horse faeces are dominated by 24-ethylcoprostanol, which is different from human sterol and can be used as biomarkers

for faecal contamination from these host groups (Leeming et al. 1996). Leeming et al. (1996) profiled a range of sterols and stanols in human and animal faeces and concluded that sterol/stanols ratios are distinctive enough to differentiate between human and animal host groups. This method is considered a viable alternative to microbiological indicators of faecal contamination (Murtaugh & Bunch 1976) and has been used to identify faecal contamination in surface waters (Leeming et al. 1997; Suprihatin et al. 2003).

Tables 1, 2 and 3 summarise the preceding section and provide a useful 'first glance' comparison of the advantages and disadvantages of these methods.

### **Ecological Application of MST Methods**

#### **Phenotypic database-dependent methods**

ARA has been widely used in MST studies (Graves et al. 2002; Hagedorn et al. 1999; Harwood, Whitlock & Withington 2000; Parveen et al. 1997; Webster et al. 2004; Whitlock, Jones & Harwood 2002; Harwood et al. 2003; Wiggins et al. 1999). For example, a large enterococci database (i.e. 7058 isolates) was developed from human, livestock and wildlife, and used successfully to identify cattle as the predominant sources of contamination in a watershed in Virginia (Hagedorn et al. 1999). Graves et al. (2002) developed an ARA database comprising 1174 enterococci isolates from 7 host groups. In all, 2012 isolates were tested from another watershed in Virginia, of which 50% were identified as livestock followed by wildlife (40%) and human (10%).

Geary & Davies (2003) used ARA to identify the sources of faecal contamination in a shellfish growing area in NSW, Australia. In all, 166 enterococci isolates were tested from 4 host groups. Application of this database in an ecological study failed to identify any dominant source. Hagedorn

et al. (2003) developed a CSU database of 365 enterococci isolates from human (i.e. 105 isolates) and non-human (i.e. 260 isolates) sources. Ninety unknown enterococci isolates were collected from three sampling sites with pre-suspected sources. In this case the database was able to identify the suspected sources correctly. However, this method has not been frequently used in MST and requires further evaluation. Biochemical fingerprinting has also recently been used to provide the evidence of septic system failure by comparing enterococci and *E. coli* from defective septic systems with those found in creek water samples (Ahmed, Neller & Katouli 2005a). In addition, this system has also been used to develop a large database of enterococci (i.e. 4057) and *E. coli* (i.e. 3728) isolates from 10 host groups. The database was successful in identifying more than 65% of faecal indicator bacteria in Eudlo Creek, Southeast, Queensland, Australia (Ahmed, Neller & Katouli 2005b).

#### **Genotypic database-dependent methods**

Scott et al. (2004) used the ribotyping method and tested 515 *E. coli* isolates from a watershed in Southern California. Of these, 88% isolates were identified as animal sources and the remaining were identified as human. Simmons et al. (2000) used PFGE method and tested a collection (i.e. 439) of *E. coli* isolates and identified wild animals and dogs as predominant sources. However, this method has not been extensively used in MST studies and requires further evaluation. Dombek et al. (2000) reported that rep-PCR fingerprinting of *E. coli* strains can be used to differentiate between human and animals host groups and reported that 100% of the chicken and cow isolates and 83% of the human isolates were assigned to the correct host groups. Yet another study used ARA in combination with rep-PCR to identify the sources of *E. faecalis* in Pensacola Beach, Florida and identified seagulls as the main contributor (Genther et al. 2005).

#### **Methods Comparison Study**

The performance of a combination of ARA, AFLP and 16S rRNA sequences has been evaluated to differentiate 319 *E. coli* isolates from human and animals. Among all the methods tested, AFLP performed better than others did (Guan et al. 2002). Moyda et al. (2003) evaluated PFGE, rep-PCR and ribotyping to identify the sources of contamination in water samples mixed with faeces from known sources. All methods were able to identify the dominant sources. However, the methods also yielded high false positive rates (i.e. 57%). In another study, Stoeckel et al. (2004) compared seven protocols including ARA, CSU, ribotyping using the restriction enzyme *HindIII* and *EcoRI*, PFGE, rep-PCR and BOX-PCR. A low accuracy was obtained for all methods tested.

#### **Key assumptions of microbial source tracking methods**

As mentioned before, in MST methods, the clonal population structure of indicator bacteria is used to categorise microorganisms on the basis of their genotypic or phenotypic fingerprint. However, the successful outcome of MST methods depends on the following assumptions.

##### **Host specificity**

The indicator bacterium should be specific to the host that contributes faecal contamination to waterways. However, certain indicator bacteria appear not to be quite host-specific as they are found in multiple host groups. These groups of indicator bacteria are referred as cosmopolitan (Whitlock, Jones & Harwood 2002). It has been argued that the lack of host specificity could be due to insufficient sampling of indicator bacteria. It has to be noted though that highly discriminatory methods such as PFGE also identify cosmopolitan isolates. Cosmopolitan host distribution is well documented in *E. coli* (Hartel et al. 2002; McLellan 2004) and F+ specific coliphages (Cole, Long & Sobsey 2003).

**Table 1: Advantages and disadvantages of database-dependent genotypic and phenotypic methods**

Methods	Target indicator	Advantages	Disadvantages
Genotypic methods	<i>E. coli</i> Enterococci	1. Highly discriminatory 2. Highly reproducible 3. Quantitative 4. Discriminate multiple host groups	1. Requires development of a large reference database 2. Bacterial culture required 3. Too sensitive to broadly discriminate source 4. Database temporally and geographically specific 5. Labour-intensive 6. Requires special training
Rep-PCR	<i>E. coli</i>	1. Rapid 2. Requires modest resources 3. Requires less technical expertise 4. Quantitative 5. Discriminate isolates from multiple host groups	1. Requires development of a reference database 2. Bacterial culture required 3. Database temporally and geographically specific 4. Results may vary in different laboratories due to different protocols
Ribotyping	<i>E. coli</i> Enterococci	1. Highly stable 2. Discriminate isolates from multiple host groups 3. Quantitative 4. Can be automated	1. Requires development of a large reference database 2. Bacterial culture required 3. Complex fingerprinting procedure 4. Labour intensive 5. Database temporally and geographical specific 6. Lack of discriminatory power 7. Requires special training
Phenotypic methods	<i>E. coli</i> Enterococci	1. Rapid 2. Require limited training 3. Quantitative 4. Discriminate isolates from multiple host groups 5. Inexpensive 6. Quantitative	1. Requires development of a reference database 2. Bacterial culture required 3. Antibiotic resistance carried on plasmids which can be lost or gained during cultivation and storage 4. Database temporally and geographically specific 5. Isolates only resistant to antibiotics can be typed 6. Can yield false-positive.
CSU	<i>E. coli</i> Enterococci	1. Rapid 2. Require limited training 3. High stability 4. Quantitative 5. Discriminate isolates from multiple host groups	1. Requires development of a reference database. 2. Bacterial culture required 3. Database temporally and geographically specific 4. Methods variation
Biochemical fingerprinting	<i>E. coli</i> Enterococci	1. Rapid 2. Semi-automated 3. Require limited training 4. Quantitative 5. Discriminate isolates from multiple host groups	1. Requires development of a reference database 2. Target indicator cultivation required 3. Database temporally and geographically specific 4. Only metabolically active bacteria can be typed

### Temporal stability

The indicator bacterium should be stable within individual host groups over time. *E. coli* populations which occur only once at a single sampling occasion are referred to as transient populations, whilst others occurring multiple times are referred to as resident populations (Caugant, Levin & Selander 1981). These resident populations

within host groups should be stable over time, and if not, then the database needs to be updated regularly if being utilised in ecological studies.

### Geographical stability

The indicator bacterium should exhibit geographical stability and therefore a database developed from one geographical

**Table 2: Advantages and disadvantages of database-independent methods**

Methods	Target indicator	Advantages	Disadvantages
Host-specific PCR	Bacteroides Bifidobacteria Enterococci Rhodococcus F <sup>+</sup> coliphage Adenovirus Enterovirus	1. Rapid 2. Development of a reference database not required 3. Bacterial culture not required	1. Non-quantitative 2. May not survive long in natural waters. 3. Primers currently not available for all relevant hosts.
Virus-specific PCR	Adenovirus Enterovirus	1. Rapid 2. Development of a reference database not required 3. Target indicator cultivation not required 4. Host specific 5. High sensitivity	1. Non-quantitative 2. Can identify only human sources 3. Low in number, require s large sample size 4. Can be absent when human contamination evident 5. Concentration and purification of viral nucleic acid from environmental samples can be difficult.
Gene specific PCR	E.coli toxin gene	1. Development of a reference database not required 2. Bacterial culture not required 3. Provide direct evidence that potential harmful bacteria present 4. Rapid	1. Non-quantitative 2. Cannot discriminate among multiple host groups 3. Identify only human 4. Primers currently not available for all relevant hosts. 5. Requires special training
F <sup>+</sup> RNA coliphage	F <sup>+</sup> coliphage	1. Development of a reference database not required 2. Discriminate isolates between human and animals 3. High stability	1. Non-quantitative 2. Can identify only human 3. Lack of host specificity 4. Concentrations can be low in environmental samples 5. Coliphages cultivation required

area is valid for another geographical area. Geographical variation can limit the universal application of a database. Little is known on the geography of faecal indicator bacteria. Miller & Hartl (1986) tested *E. coli* strains from farm animals and humans and reported that strains are clonal in nature and not geographically specific. Another recent study tested 568 *E. coli* from Idaho and at three locations in Georgia for four host groups, reporting that geographical variation exist among these host groups (Hartel et al. 2002).

### Representativeness

Database representativeness is one of the most important factors in database dependent MST studies. This factor (i.e. how many isolates required to develop a representative database) has not been addressed in any study. However, it has to be noted that, cost and time can limit this factor. Development of a large

genotypic reference database could be quite costly depending on the typing method used with regards to phenotypic database. Under-sampling of faecal bacteria can compromise representativeness of a database leading to its inability to capture the temporal or geographical variability, as well as high diversity of faecal indicator bacteria (Whitlock, Jones & Harwood 2002; Wiggins et al. 2003).

### Primary versus secondary habitat

GI tracts of host groups are considered as the primary habitat for faecal indicator bacteria while environments are the secondary habitat. One of the hypotheses in MST is that the clonal composition of the isolates from water (i.e. secondary habitat) represents the clonal composition of the isolates in the host groups (i.e. primary habitat) responsible for faecal inputs to the environment. However, several studies have shown that distinct differences exist among the primary

**Table 3: Advantages and disadvantages of chemical methods**

Chemical methods	Advantages	Disadvantages
Optical brighteners	<ol style="list-style-type: none"> <li>1. Indicate human contamination</li> <li>2. Inexpensive</li> <li>3. simple</li> </ol>	<ol style="list-style-type: none"> <li>1. May not indicate recent contamination</li> <li>2. Cannot identify non-point sources</li> <li>3. Does not provide information regarding public</li> <li>4. health risks</li> </ol>
Caffeine/pharmaceuticals	<ol style="list-style-type: none"> <li>1. Indicate human contamination</li> </ol>	<ol style="list-style-type: none"> <li>1. Analysis expensive</li> <li>2. Easily degraded by soil microbes</li> <li>3. Sensitivity issues</li> <li>4. Dilution makes it difficult to detect in receiving waters</li> </ol>
Faecal sterols analysis	<ol style="list-style-type: none"> <li>1. High sensitivity</li> <li>2. Can distinguish between human and animal contamination.</li> </ol>	<ol style="list-style-type: none"> <li>1. Expensive</li> <li>2. certain sterols Can be found in plants</li> <li>3. Easily degraded by soil microbe</li> <li>4. May not indicate recent contamination</li> <li>5. Not relevant to human health.</li> </ol>

versus secondary habitats of *E. coli*. Whittam (1989) tested 113 *E. coli* electrophoretic types (by multilocus enzyme electrophoresis or MLEE) from bird faeces (primary habitat) and the litter (secondary habitat) on which they had defecated. Only 10% of the isolates were found in both the primary and secondary habitat. Another study using multi locus electrophoretic enzyme of *E. coli* from two septic tanks and their associated residents showed that *E. coli* strains from only one septic tank was similar to those of the residents. In contrast strains recovered from the septic tank of the second household were genetically distinct from strains recovered from its associated residents. Based on the differences between the growth rate and temperature response of these strains they concluded that changes in the primary and secondary habitat of the strains could limit efforts to identify the sources of faecal pollution in the environment (Gordon, Bauer & Johnson 2002).

### Conclusion

Certain indicator bacteria such as enterococci and *E. coli* have been used more frequently than others, but none of these indicators are regarded as universal or possess all the required criteria of an ideal faecal

indicator bacterium. Similarly, while some MST methods have been used frequently, no single method is clearly superior to others and the advantages and disadvantages of these methods have been discussed in this paper among others (McLellan 2004; Meays 2004; Scott et al. 2002; Stewart et al. 2003). Genotypic methods although highly discriminatory, can be laborious and/or expensive for ecological studies where a large number of isolates need to be tested (Hartel et al. 2002; Kühn et al. 1995). In contrast, phenotypic methods can be used to test a large number of isolates within a short time and are relatively inexpensive. However, some of them are not highly discriminatory and lack stability. Therefore, a combination of different methods, where applicable, should be used in ecological studies to obtain confirmatory results. The performance of the majority of database-dependent methods reviewed were either limited by their size, representativeness, stability and discriminatory power, or that their suitability as a MST method has not been completely evaluated in ecological studies and requires further evaluation. A recent study has shown that an ARA database is stable up to 1 year (Wiggins et al. 2003). Another study reported that a large

representative metabolic fingerprint database could be used to identify sources in different catchments of the same nature within the same geographical area. The challenge remains to identify suitable host specific microorganisms and to develop standard methods. Other indicators such as *Citrobacter freundii* or *Hafnia alvei* have also been suggested as alternatives to current indicators (Gordon 2001). Database independent methods offer many advantages over database-dependent methods, as the development of a reference database is not required, and have shown higher accuracy in method comparison studies (Griffith et al. 2003). These methods are currently under development and could

be promising for source tracking. However, these methods have limitations in discriminating among multiple host groups such as among animals. Further, they are not capable of quantifying the level of contribution from various sources. In this scenario, database dependent methods are highly beneficial if the database is developed by addressing the issues discussed earlier. The development of new techniques such as the qPCR or genetic array that target pathogens in a contaminated water sample will greatly enhance the microbial source tracking in future as they also provide the direct evidence of the presence of pathogenic microorganisms in a watershed.

## References

- Ahmed, W., Neller, R. & Katouli, M. 2005a, 'Evidence of septic systems failure determined by a bacterial biochemical fingerprinting method', *Journal of Applied Microbiology*, vol. 98, pp. 910-20.
- Ahmed, W., Neller, R. & Katouli, M. 2005b, 'Host species-specific metabolic fingerprint database for enterococci and *Escherichia coli* and its application to identify sources of fecal contamination in surface waters', *Applied and Environmental Microbiology*, vol. 71, pp. 4461-8.
- Alhajjar, B.J., Harkin, J.M. & Chesters, G. 1990, 'Indicators of chemical pollution from septic systems', *Groundwater*, vol. 28, pp. 559-68.
- Amor, K., Heinrichs, D.E., Frirdich, E., Ziebell, K., Johnson, R.P. & Whitfield, C. 2000, 'Distribution of core oligosaccharide types in lipopolysaccharides from *Escherichia coli*', *Infection and Immunity*, vol. 68, pp. 1116-24.
- Arraj, A., Bohatier, J., Laveran, H. & Traore, O. 2005, 'Comparison of bacteriophage and enteric virus removal in pilot scale activated sludge plants', *Journal of Applied Microbiology*, vol. 98, pp. 516-24.
- Baggi, F., Demarta, A. & Peduzzi, R. 2001, 'Persistence of viral pathogens and bacteriophages during sewage treatment: lack of correlation with indicator bacteria', *Research in Microbiology*, vol. 152, pp. 743-51.
- Barber, L.B. II, Thurman, E.M., Schroeder, M.P. & LeBlanc, D.R. 1988, 'Long-term fate of organic micro pollutants in sewage contaminated groundwater', *Environmental Science and Technology*, vol. 12, pp. 369-76.
- Barbier, N.P., Saulnier, E., Chachaty, S., Dumontier, S. & Andermont, A. 1996, 'Random amplified polymorphic DNA typing versus pulsed-field gel electrophoresis for epidemiological typing of vancomycin-resistant enterococci', *Journal of Clinical Microbiology*, vol. 34, pp. 106-9.
- Barnes, D. & Gordon, D.M. 2004, 'Coliform dynamics and the implications for source tracking', *Environmental Microbiology*, vol. 6, pp. 501-9.
- Baudisöva, D. 1997, 'Evaluation of *Escherichia coli* as the main indicator of faecal pollution', *Water Science and Technology*, vol. 35, pp. 333-56.
- Bernhard, A.E. & Field, K.G. 2000a, 'A PCR assay to discriminate human and animal feces on the basis of the host difference in *Bacteroides-Prevotella* genes encoding 16S rRNA', *Applied and Environmental Microbiology*, vol. 66, pp. 4571-4.
- Bernhard, A.E. & Field, K.G. 2000b, 'Identification of non-point sources of fecal pollution in coastal waters by using host-specific 16S ribosomal DNA genetic markers from fecal anaerobes', *Applied and Environmental Microbiology*, vol. 66, pp. 1587-94.
- Beutin, L. 1999, '*Escherichia coli* as a pathogen in dogs and cats', *Veterinary Research*, vol. 30, pp. 285-98.

- Bitton, G. 1999, *Wastewater Microbiology*, Wiley-Liss, New York.
- Bitton, G. 2005, Microbial indicators of fecal contamination: Application to microbial source tracking. Report submitted to the Florida Stormwater Association.
- Burkhardt, M.R., Soliven, P.R., Werner, S.L. & Vaught, D.G. 1999, 'Determination of sub-microgram per litre concentrations of caffeine in surface and groundwater samples by extraction and liquid chromatography', *Journal of AOAC International*, vol. 82, pp. 161-6.
- Cabelli, V.J., Dufour, A.P., McCabe, L.J. & Levin, M.A. 1982, 'Swimming-associated gastroenteritis and water quality', *American Journal of Epidemiology*, vol. 115, pp. 606-16.
- Cabelli, V.J., Dufour, A.P., McCabe, L.J., & Levin, M.A. 1993, 'A marine recreational water quality criterion consistent with indicator concepts and risk analysis'. *Journal of Water Pollution Control Federation*, vol. 55, pp. 1306-14.
- Carrillo, M., Estrada, E. & Hazen, T.C. 1985, 'Survival and enumeration of the fecal indicators *Bifidobacterium adolescentis* and *Escherichia coli* in a tropical rain forest watershed', *Applied and Environmental Microbiology*, vol. 50, pp. 468-76.
- Carvalho de Moura, Irino A.C.K. & Vidotto, M.C. 2001, 'Genetic variability of avian *Escherichia coli* strains evaluated by enterobacterial repetitive intragenic consensus and repetitive extragenic palindromic polymerase chain reaction', *Avian Disease*, vol. 45, pp. 173-81.
- Cato, E.P., George, W.L. & Finegold, S.M. 1986, 'Clostridium', in *Bergey's Manual of Systematic Bacteriology*, vol. 2, eds P.H.A. Sneath, N.S. Mair, M.E. Sharpe & J.G. Holt, The Williams and Wilkins Co., Baltimore, MD.
- Caugant, D.A., Levin, B.R. & Selander R.K. 1981, 'Genetic diversity and temporal variation in the *Escherichia coli* population of a human host', *Genetics*, vol. 98, pp. 467-90.
- Chung, H., Jaykus, L.A., Lovelace, G. & Sobsey, M.D. 1998, 'Bacteriophages and bacteria as indicators of enteric viruses in oysters and their harvest waters', *Water Science and Technology*, vol. 38, pp. 37-44.
- Cole, D., Long, S. C. & Sobsey, M. D. 2003, 'Evaluation of F+ and DNA coliphages as source-specific indicators of fecal contamination in surface waters', *Applied and Environmental Microbiology*, vol. 69, pp. 6507-14.
- Craun, G.F., Berger, P.S. & Calderon, R.L. 1997, 'Coliform bacteria and waterborne disease outbreaks', *Journal of American Water Works Association*, vol. 89, pp. 96-106.
- Dalla-Costa, L.M., Irino, K., Rodriguez, J., Rivera, I.N. & Trabulsi, L.R. 1998, 'Characterisation of diarrhoeagenic *Escherichia coli* clones by ribotyping and ERIC-PCR', *Journal of Medical Microbiology*, vol. 47, pp. 227-34.
- Davies, C.M., Long, J.A., Donald, M. & Ashbolt, N. J. 1995, 'Survival of fecal microorganisms in aquatic sediments of Sydney, Australia', *Applied and Environmental Microbiology*, vol. 61, pp. 1888-96.
- Desmarais, T.R., Solo-Gabriele, H.M. & Palmer, C.J. 2002, 'Influence of soil on fecal indicator organisms in a tidally influenced subtropical environment', *Applied and Environmental Microbiology*, vol. 68, pp. 1165-72.
- Dombek, P.E, Johnson, L.K, Brown, M.B. & Sadowsky, M.J. 2000, 'Use of repetitive DNA sequences and the PCR to differentiate *Escherichia coli* isolates from human and animal sources', *Applied and Environmental Microbiology*, vol. 66, pp. 2572-7.
- Eckel, W.P., Ross, B. & Isensee, R.K. 1993, 'Pentobarbital found in groundwater', *Groundwater*, vol. 31, pp. 801-4.
- Edberg, S.C., Rice, E.W., Karlin, R.J. & Allen, M.J. 2000, '*Escherichia coli*: the best biological drinking water indicator for public health protection', *Journal of Applied Microbiology*, Symposium Supplement, vol. 88, pp. 106S-16S.
- Evison, L.M. & James, A. 1974, *Bifidobacterium* as an indicator of faecal pollution in water, pp. 107-116, *Proceedings of the 7th International Conference on Water Pollution Research*, Pergamon Press, Oxford.
- Farber, J.M. 1996, 'An introduction to the hows and whys of molecular typing', *Journal of Food Protection*, vol. 59, pp. 1091-101.
- Field, K.G., Bernhard, A.E. & Brode, T.J. 2003, 'Molecular approaches to microbial monitoring: Fecal source detection', *Environmental Monitoring and Assessment*, vol. 81, pp. 313-26.

- Field, K.G., Chern, E.C., Dick, L.K., Fuhrman, J., Griffith, J., Holden, P.A., LaMontagne, M.G., Olson, B., Simonich, M.T. 2003, 'A comparative study of culture-independent, library-independent genotypic methods of fecal source tracking', *Journal of Water and Health*, vol. 1, pp. 181-94.
- Fong, T.T., Griffin, D.W. & Lipp, E.K. 2005, 'Molecular assays for targeting human and bovine enteric viruses in coastal waters and their application for library-independent source tracking', *Applied and Environmental Microbiology*, vol. 71, pp. 2070-8.
- Fujioka, R.S. & Shizumara, L.K. 1985, '*Clostridium perfringens*: A reliable indicator of stream water quality', *Journal of Water Pollution Control Federation*, vol. 57, pp. 986-92.
- Fujioka, R.S. 2001, 'Monitoring coastal marine waters for spore-forming bacteria of faecal and soil origin to determine point from non-point source pollution', *Water Science and Technology*, vol. 44, pp. 181-8.
- Funderburg, S.W. & Sorber, C.A. 1985, 'Coliphages as indicators of enteric viruses in activated sludge', *Water Research*, vol. 19, pp. 547-55.
- Geary P.M. & Davies, C.M. 2003, 'Bacterial source tracking and shellfish contamination in a coastal catchment', *Water Science and Technology*, vol. 47, pp. 95-100.
- Genthner, F.J., James, J.B., Yates, D.F. & Friedman, S.D. 2005, 'Use of composite data sets for source tracking enterococci in the water column and shoreline intestinal waters on Pensacola Beach, Florida', *Marine Pollution Bulletin*, vol. 50, pp. 724-32.
- Gilpin, B.J., Gregor, J.E. & Savill, M.G. 2002, 'Identification of the source of faecal pollution on contaminated rivers', *Water Science and Technology*, vol. 46, pp.9-15.
- Godfree, A.F., Kay, D. & Wyer, M.D. 1997, Faecal streptococci as indicators of faecal contamination in water, *Journal of Applied Microbiology*, Symposium Supplement, vol. 83, pp. 110S-19S.
- Gordon, D.M., Bauer, S. & Johnson, J.R. 2002, 'The genetic structure of *Escherichia coli* populations in primary and secondary habitat', *Microbiology*, vol. 148, pp. 1513-22.
- Grabow, W.O.K., Neubrech, T.E, Holtzhausen, C.S. & Jofre, J. 1995, '*Bacteroides fragilis* and *Escherichia coli* bacteriophages: excretion by humans and animals', *Water Science and Technology*, vol. 5-6, pp. 223-30.
- Graves, A.K., Hagedorn, C., Teetor, A. Mahal, M., Booth, A.M. & Reneau, Jr. R.B. 2002, 'Antibiotic resistance profiles to determine sources of fecal contamination in a rural Virginia watershed', *Journal of Environmental Quality*, vol. 31, pp. 1300-8.
- Griffith, J.F., Weisberg, S.B. & McGee, C.D. 2003, 'Evaluation of microbial source tracking methods using mixed fecal sources in aqueous test samples', *Journal of Water and Health*, vol. 1, pp. 141-51.
- Grundmann, H.C., Schneider, D., Hartung, D., Daschner, F.D. & Pitt, T.L. 1995, 'Discriminatory power of the three DNA-based typing techniques for *Pseudomonas aeruginosa*', *Journal of Clinical Microbiology*, vol. 33, pp. 528-34.
- Guan, S., Xu, R., Chen S., Odumeru, J. & Gyles, C. 2002, 'Development of a procedure for discriminating among *Escherichia coli* isolates from animal and human sources', *Applied and Environmental Microbiology*, vol. 68, pp. 2690-8.
- Hagedorn, C., Robinson, S.L., Filtz, J.R., Grubbs, S.M., Angier, T.A. & Reneau, R.B. Jr. 1999, 'Determining sources of fecal pollution in a rural Virginia watershed with antibiotic resistance patterns in fecal streptococci', *Applied and Environmental Microbiology*, vol. 65, pp. 5522-31.
- Hagedorn, C., Crozier, J.B., Mentz, K.A. Booth, A.M., Graves, A.K., Nelson, N.J. & Reneau R.B. Jr. 2003, 'Carbon source utilization profiles as a method to identify sources of faecal pollution in water', *Journal of Applied Microbiology*, vol. 94, pp.792-9.
- Hartel, P.G., Summer, J.D., Hill, J.L., Collins, J.C., Entry, J.A. & Segers, W.I. 2002, 'Geographic variability of *Escherichia coli* isolates from animals in Idaho and Georgia', *Journal of Environmental Quality*, vol. 31, pp.1273-8.
- Hartel, P.G., Summer, J.D. & Segars, W.I. 2003, 'Deer diet affects ribotype diversity of *Escherichia coli* for bacterial source tracking', *Water Research*, vol. 37, pp.3263-8.
- Harwood, V.J., Parrish, B.D. & Wagner, V. 1999, 'Isolation of fecal coliform bacteria from the diamondback terrapin (*Malaclemys terrapain centrata*)', *Applied and Environmental Microbiology*, vol. 65, pp. 865-7.
- Harwood, V.J., Whitlock, J. & Withington, V. 2000, 'Classification of antibiotic resistance patterns of indicator bacteria by discriminant analysis: use in predicting the source of fecal contamination

- in tropical waters', *Applied and Environmental Microbiology*, vol. 66, pp. 3698-704.
- Harwood, V.J., Wiggins, B., Hagedorn, C., Ellender, R.D., Gooch, J., Kern, J., Samadpour, M., Chapman, A.C.H., Robinson, B.J. & Thomson, B.J. 2003, 'Phenotypic library-based microbial source tracking methods: efficacy in the California collaborative study', *Journal of Water and Health*, vol. 1, pp. 209-24.
- Havelaar, A.H. & Hogeboom, W.M. 1984, 'A method for the enumeration of male-specific bacteriophages in sewage', *Journal of Applied Microbiology*, vol. 56, pp. 439-7.
- Hill, V.R. & Sobsey, M.D. 1999, 'Microbial indicator reductions in alternative treatment systems for swine wastewater', *Water Science and Technology*, vol. 38, pp.119-22.
- Hilton, M.C. & Stotzky, G. 1973, 'Use of coliphages as indicators of water pollution', *Canadian Journal of Microbiology*, vol. 19, pp. 741-51.
- Holmes, B., Costa, M.M., Garner, S.L.W. & Stevens, O.M. 1994, 'Evaluation of Biolog system for identification of some gram-negative bacteria of clinical importance', *Journal of Clinical Microbiology*, vol. 32, pp. 1970-5.
- Jiang, S.C. 2002, 'Adenovirus as an index of human viral contamination: Microbial source tracking workshop', *Proceedings of US EPA Workshop on Microbial Source Tracking*, February, Irvine, CA.
- Jofre, J., Bosch, A., Lucena, F., Girones, R. & Tartera, C. 1986, 'Evaluation of *Bacteroides fragilis* bacteriophages as indicators of the virological quality of water', *Water Science and Technology*, vol. 18, pp.167-77.
- Johnson, J.R., Brown, J.J., Carlino, U.B. & Russo, T.A. 1998, 'Colonization with and acquisition of uropathogenic *Escherichia coli* as revealed by polymerase chain reaction-based detection', *Journal of Infectious Disease*, vol. 177, pp.1120-4.
- Johnson, L.K., Brown, M.B., Carruthers, E.A., Ferguson, J.A., Dombek, P.E. & Sadowsky, M.J. 2004, 'Sample size, library composition, and genotypic diversity among natural populations in *Escherichia coli* from different animals influence accuracy of determining sources of fecal pollution', *Applied and Environmental Microbiology*, vol. 70, pp. 4478-85.
- Kay, D., Fleisher, J.M., Salmon, R.L., Jones, F., Wyer, M.D., Godfree, A.F., et al. 1994, 'Predicting likelihood of gastroenteritis from sea bathing: Results from randomised exposure', *Lancet*, vol. 344, pp. 905-9.
- Kibbey, H.J., Hagedorn, C. & McCoy, E.L. 1978, 'Use of fecal streptococci as indicators of pollution in soil', *Applied and Environmental Microbiology*, vol. 35, pp.711-7.
- Kott, Y., Roze, N., Sperber, S. & Betzer, N. 1974, 'Bacteriophages as viral pollution indicators', *Water Research*, vol. 8, pp. 165-71.
- Koeuth, T.J. Versalovic, J. & Lupski, J.R. 1995, 'Differential subsequence conservation of interspersed repetitive *Streptococcus pneumoniae* BOX elements in diverse bacteria', *Genome Research*, vol. 5, pp. 408-18.
- Kühn, I., Katouli, M., Wallagren, P., Söderlind, O. & Möllby, R. 1995, 'Biochemical fingerprinting as a tool to study the diversity and stability of intestinal microflora', *Microecology and Therapy*, vol. 23, pp. 140-8.
- Leclerc, H., Edberg, S., Pierzo, V. & Delattre, J.M. 2000, 'Bacteriophages as indicators of enteric viruses and public health risk in groundwaters', *Journal of Applied Microbiology*, vol. 88, pp. 5-21.
- Leclerc, H., Mossel, D., Edberg, S.C. & Strujk, C.J. 2001, 'Advances in the bacteriology of the coliform group: Their suitability as markers of microbial water safety', *Annual Review of Microbiology*, vol. 55, pp. 201-34.
- Leeming R., Ball, A., Ashbolt, N.J. & Nicholas, P.D. 1996, 'Using faecal sterols from humans and animals to distinguish faecal pollution in receiving waters', *Water Research*, vol. 30, pp. 2893-900.
- Leeming, R., Latham, V., Rayner, M. & Nicholas, P. 1997, 'Detecting and distinguishing sources of sewage pollution in Australia inland and coastal water sediments', *ACS Symposium Series*, vol. 671, pp.306-19.
- Ley, V., Higgins, J. & Fayer, R. 2002, 'Bovine enteroviruses as indicators of fecal contamination', *Applied and Environmental Microbiology*, vol. 68, pp. 3455-61.
- Lipp, E.K., Farrah, S.A. & Rose, J.B. 2001, 'Assessment and impact of microbial fecal pollution and human enteric pathogens in a coastal community', *Marine Pollution Bulletin*, vol. 42, pp. 286-93.

- Levin, M.A. 1997, Bifidobacterium as water quality indicators, in *Bacterial Indicators: Health Hazards Associated with Water*, ed. A.W. Hadley & B.J. Dutka, ASTM Publications, Philadelphia, Pa.
- Ludwig, W., Seewald, E., Kilpper-Bälz, R., Schleifer, K. H., Magrum, L., Woese, C.R., Fox, G.E. & Stackebrandt, E. 1985, 'The phylogenetic position of *Streptococcus* and *Enterococcus*', *Journal of General Microbiology*, vol. 131, pp. 543-51.
- Matsuki, T., Watanabe, K., Tanaka, R., Fukuda, M. & Oyaizu, H. 1999, 'Distribution of bifidobacterial species in human intestinal microflora examined with 16S rRNA-gene-targeted species-specific primers', *Applied and Environmental Microbiology*, vol. 65, pp. 4506-12.
- McLellan, S.L. 2004, 'Genetic diversity of *Escherichia coli* isolated from urban rivers and beach water', *Applied and Environmental Microbiology*, vol. 70, pp. 4658-65.
- Meays, C.L., Broersma, K., Nordin, R. & Majumder, A. 2004, 'Source tracking fecal bacteria in water: a critical review of current methods', *Journal of Environmental Management*, vol. 73, pp. 71-9.
- Miller, R.D. & Hartl, D.L. 1986, 'Biotyping confirms a nearly clonal population structure in *Escherichia coli*', *Evolution*, vol. 40, pp. 1-12.
- Möllby, R., Kühn, I. & Katouli, M. 1993, 'Computerized biochemical fingerprinting: A new tool for typing of bacteria', *Review of Medical Microbiology*, vol. 14, pp. 231-41.
- Moore, W.E.C. & Holdeman, L.V. 1974, 'Human faecal flora: The normal flora of 20 Japanese Hawaiians', *Applied Microbiology*, vol. 27, pp. 961-79.
- Moyda, S.P., Carson, C.A., Fuhrmann, J.J., Hahm, B.K., Hartel, P.G., Kuntz, R.L., Nakatsu, C.H., Sadowsky, M.J., Samadpour, M. & Yampara-Iquise, H. 2003, 'Comparing genotypic bacterial source tracking methods that require a host origin database', *Journal of Water and Health*, vol. 1, pp. 167-80.
- Murtaugh, J. & Bunch, R. L. 1976, 'Sterols as a measure of faecal pollution', *Journal of Water Pollution Control Federation*, vol. 39, pp. 404-9.
- Murray, B.E., Singh, K.V., Health, J.D., Sharma, B.R. & Weinstock, G.M. 1990, 'Comparison of genomic DNAs of different enterococcus isolates using restriction endonucleases with infrequent recognition sites', *Journal of Clinical Microbiology*, vol. 28, pp. 2059-63.
- National Health and Medical Research Council (NHMRC) 1993, *Review of Coliforms as Microbial Indicators of Drinking Water Quality*, National Health and Medical Research Council, Canberra.
- Noble, L. & Gow, J. 1998, 'The effect of suspending solution supplemented with marine cations on the oxidation of Biolog GN Microplate™ substrates by Vibrionaceae bacteria', *Canadian Journal of Microbiology*, vol. 44, pp. 251-8.
- Noble, R.T., Allen, S.M., Blackwood, A.D., Chu, W., Jiang, S.C., Lovelace, G.L., Sobsey, M.D., Stewart, J.R. & Wait, D.A. 2003, 'Use of viral pathogens and indicators to differentiate between human and non-human fecal contamination in a microbial source tracking comparison study', *Journal of Water and Health*, vol. 1, pp. 195-207.
- Olive, D.M. & Bean, P. 1999, 'Principles and applications of methods for DNA-based typing of microbial organisms', *Journal of Clinical Microbiology*, vol.37, pp.1661-9.
- Olson, B.H., Khatib, L.A., & Chern, E.C. 2002, 'Source tracking of fecal waste material in environmental waters using a biomarker based on enterotoxin genes in *E. coli*', *Proceedings of US EPA Workshop on Microbial Source Tracking*, Irvine, CA, February.
- Parveen, S., Murphree, R.L., Edmiston L., Kaspar, C.W., Portier, K.M. & Tamplin, M.L. 1997, 'Association of multiple-antibiotic-resistance profiles with point and non-point sources of *Escherichia coli* in Apalachicola Bay', *Applied and Environmental Microbiology*, vol. 63, pp. 2607-12.
- Parveen, S., Portier, K.M., Robinson, K., Edmiston, L. & Tamplin, M. 1999, 'Discrimination analysis of ribotype profiles of *Escherichia coli* for differentiating human and non-human sources of fecal pollution', *Applied and Environmental Microbiology*, vol. 65, pp. 3142-7.
- Payment, P. & Franco, E. 1993, '*Clostridium perfringens* and somatic coliphages as indicators of the efficiency of drinking water treatment for viruses and protozoan cysts', *Applied and Environmental Microbiology*, vol. 59, pp. 2418-24.

- Pina, S., Puig, M., Lucena, F., Jofre, J. & Girones, R. 1998, 'Viral pollution in the environment and in shellfish: Human adenovirus detection by PCR as an index of human viruses', *Applied and Environmental Microbiology*, vol. 64, pp. 3376-82.
- Poh, C.L., Yeo, C.C. & Tay, L. 1992, 'Genome fingerprinting by pulsed-field gel electrophoresis and ribotyping to differentiate *Pseudomonas aeruginosa* serotype O11 strains', *European Journal of Clinical Microbiology and Infectious Diseases*, vol. 11, pp. 817-22.
- Power, M.L., Littlefield-Wyer, J., Gordon, D.M., Veal, D.A. & Slade, M.B. 2005, 'Phenotypic and genotypic characterisation of encapsulated *Escherichia coli* isolated from blooms in two Australian Lakes', *Environmental Microbiology*, vol. 7, pp. 631-40.
- Rhodes, M.W. & Kator, H. 1999, 'Sorbitol-fermenting bifidobacteria as indicators of diffuse human faecal pollution in estuarine watersheds', *Journal of Applied Microbiology*, vol. 87, pp. 528-35.
- Roll, B.B. & Fujioka, R.S. 1997, 'Sources of faecal indicator bacteria in a brackish tropical stream and their impact on recreational water quality', *Water Science and Technology*, vol. 35, pp. 179-86.
- Samadpour, M. & Checowitz, N. 1995, *Little Soos Creek Microbial Source Tracking: A Survey*, University of Washington Department of Environmental Health, Seattle.
- Sargeant, D. 1999, Faecal contamination source identification method in surface water, Washington State Department of Ecology, Ecology Report No. 99-345.
- Saulnier, P., Bourneix, C., Prevost, G. & Andremont, A. 1993, 'Random amplified polymorphic DNA assay is less discriminant than pulsed-field gel electrophoresis for typing strains of methicillin-resistant *Staphylococcus aureus*', *Journal of Clinical Microbiology*, vol. 31, pp. 982-5.
- Schlichting, C., Branger, C. & Fournier, J.M. 1993, 'Typing of *Staphylococcus aureus* by pulsed-field gel electrophoresis, zymotyping, capsular typing and phage typing: Resolution of clonal relationships', *Journal of Clinical Microbiology*, vol. 31, pp. 227-32.
- Scott, T.M., Rose, J.B., Jenkins, T.M., Farrah, S.R. & J. Lukasik, J. 2002, 'Microbial source tracking: current methodology and future directions', *Applied and Environmental Microbiology*, vol. 68, pp. 5796-803.
- Scott, T.M., Caren, J., Nelson, G.R., Jenkins, T.M. & Lukasik, J. 2004, 'Tracking sources of fecal pollution in a South Carolina Watershed by ribotyping', *Escherichia coli: A case study*, *Environmental Forensics*, vol. 5, pp. 15-9.
- Seiler, R.L., Zaugg, S.D., Thomas, J.M. & Howcroft, D.L. 1999, 'Caffeine and pharmaceuticals as indicators of wastewater contamination in wells', *Groundwater*, vol. 37, pp. 405-10.
- Simpson, J.M., Santo Domingo, J.W. & Reasoner, D.J. 2003, 'Microbial source tracking: state of the science', *Environmental Science and Technology*, vol. 36, pp. 5280-8.
- Simmons, G.M., Waye, D.F., Herbain, S., Meyers, S. & Walker, E. 2000, 'Estimating non-point fecal coliform sources in Northern Virginia's Four Mile Run watershed', in *Proceedings of the Virginia Water Research Symposium 2000*, eds T. Younos & J. Poff, VWRRC Special Report SR-19-2000, Blacksburg.
- Sinton, L.W., Donnison, A.M. & Hastie, C.M. 1993, 'Faecal streptococci as faecal pollution indicators: a review. Part II. Sanitary significance, survival and use', *New Zealand Journal of Marine and Freshwater Research*, vol. 27, pp. 117-37.
- Sinton, L.W., Finlay, R.K. & Hannah, D.J. 1998, 'Distinguishing human from animal contamination: a review', *New Zealand Journal of Marine and Freshwater Research*, vol. 32, pp. 323-48.
- Sorensen, D.L., Eberl, S.G. & Dicksa, R.A. 1989, '*Clostridium perfringens* as a point source indicator in non-point polluted streams', *Water Research*, vol. 23, pp. 191-7.
- Stewart, J.R., Ellender, R.D., Gooch, J.A., Jiang, S., Moyda, S.P., & Weisberg, S.B. 2003, 'Recommendations for microbial source tracking methods comparison study', *Journal of Water and Health*, vol. 1, pp. 225-31.
- Stoeckel, D.M., Mathes, M.V., Hyer, K.E., Hagedorn, C., Kator, H., Lukasik, J., O'Brien, L., Fenger, T.W., Samadpour, M., Strickler, K.M., Wiggins, B.A., Kephert, C.M., Harwood, V.J., Anderson, M.A. & Dontchev, M. 2004, 'Comparison of seven protocols to identify fecal contamination sources using *Escherichia coli*', *Environmental Science and Technology*, vol. 38, pp. 6109-17.
- Suprihatin, I.E., Fallowfield, H.J., Bentham, R.H. & Cromer, M.J. 2003, 'Determination of faecal pollutants in Torrens and Patawolong catchment waters in South Australia using faecal sterols', *Water Science and Technology*, vol. 47, pp. 283-9.

- Tang-Liu, D.D., Williams, R.L. & Riegelman, S. 1993, 'Disposition of Caffeine and its metabolism in man', *Journal of Pharmacology and Experimental Therapeutics*, vol. 24, pp. 180-5.
- Tartera, C. & Jofre, J. 1987, 'Bacteriophages active against *Bacteroides fragilis* in sewage polluted waters', *Applied and Environmental Microbiology*, vol. 53, pp. 1632-7.
- Topp, E., Welsh, M., Tien, Y-C., Dang, A., Lazarovitz, G., Conn, K. & Zhu, H. 2003, 'Strain dependent variability in growth and survival of *Escherichia coli* in agricultural soil', *FEMS Microbiology Ecology*, vol. 44, pp. 303-8.
- Toranzos, G.A. & McFeters, G.A. 1997, 'Detection of indicator micro-organisms in environmental fresh waters and drinking waters', in *Manual of Environmental Microbiology*, eds C.J. Hurst, G.R. Knudsen, M.J. McInerney, L.D. Stetzenbach & M.V. Walter, American Society for Microbiology, Washington, D.C.
- US Environmental Protection Agency 2005, Microbial source tracking guide document. EPA/600-R-05-064, Office of Research and Development, Cincinnati, OH.
- Venkatesan, M.I. 1995, 'Coprostanol and other sterols as innovative indicators for human pathogen', *Environmental Science and Technology*, vol. 24, pp. 208-14.
- Versalovic, J., Koeuth, T. & Lupski, J.R. 1991, 'Distribution of repetitive DNA sequences in eubacteria and application to fingerprinting of bacterial genomes', *Nucleic Acid Research*, vol. 19, pp. 6823-31.
- Webster, L.F., Thomson, B.C., Fulton, M.H., Chestnut, D.E., Van Dolah, R.F., Leight, A.K. & Scott, G.I. 2004, 'Identification of sources of *Escherichia coli* in South Carolina estuaries using antibiotic resistance analysis', *Journal of Experimental Marine Biology and Ecology*, vol. 298, pp. 175-95.
- Whitlock, J.R., Jones, D.T. & Harwood, V.J. 2002, 'Identification of the sources of fecal coliforms in an urban watershed using antibiotic resistance analysis', *Water Research*, vol. 36, pp. 4273-82.
- Whittam, T.S. 1989, 'Clonal dynamics of *Escherichia coli* in its natural habitat', *Antonie Van Leeuwenhoek*, vol. 55, pp. 23-32.
- Wiggins, B.A., Andrews, R.W., Conway, R.A., Corr, C.L., Dobratz, E.J., Dougherty, D.P., Eppard, J.R., Knupp, S.R., Limjoco, M.C., Mettenburg, J.M., Rinehardt, J.M., Sonsino, J., Torrijos, R.L. & Zimmerman, M.E. 1999, 'Use of antibiotic resistance analysis to identify non-point sources of fecal pollution', *Applied and Environmental Microbiology*, vol. 65, pp. 3483-6.
- Wiggins, B.A., Cash, P.W., Creamer, W.S., Dart, S.E., Garcia, P.P., Gerecke, T.M., Han, J., Henry, B.L., Hoover, K.V., Johnson, E.L., Jones, K.C., McCarthy, J.G., McDonough, J.A., Mercer, S.A., Noto, M.J., Park, H., Phillips, M.S., Purner, S.M., Smith, B.M., Stevens, E.N. & Varner, A.K. 2003, 'Use of antibiotic resistance analysis for representativeness testing of multi-watershed libraries', *Applied and Environmental Microbiology*, vol. 69, pp. 3399-405.

Correspondence to:

W. Ahmed

Faculty of Science

Institute of Sustainability, Health and Regional Engagement

University of the Sunshine Coast

Maroochydore DC 4558, Queensland

AUSTRALIA

Email: shuhat@yahoo.com



## **Occupational Health and Safety: International Influences and New Epidemics**

**Chris L. Peterson and Claire Mayhew (Eds)**

*Baywood Publishing Company, Inc. Amityville, New York, 2005, 243pp. ISBN 0-89503-303-8*

For many years the risks to people at work were mostly related to exposures to chemical, physical and biological agents and much has been done to reduce these risks in many countries, but there is still much more to be achieved especially in the 'developing' countries. Much is known about these risks and how to control them and so it is largely a lack of action not a lack of knowledge and resources that obstructs more preventive activity. This text covers a range of newer occupational health and safety risks that are emerging in the industrialised countries across the world as the labour market and the organisation of work change in modern society. The book identifies and discusses a range of 'new' epidemics, which were not known a few decades ago and that are still not generally appreciated. The book makes an important point in identifying the lack of formal recognition of many cases of injury and disease which remain unrecorded in official databases. The chapters have been written in such a way as to discuss the risk factors surrounding each of the 'new' epidemics and from the information in the chapters the editors conclude that a new approach is needed to deal with these epidemics.

The introductory chapter provides an excellent overview of the inter-relationship between globalisation and internationalisation and occupational health and safety, in the context of the recent rapid growth of international trade. Chapter 2 further explores international issues, such as deregulation of working conditions and the diminution of the influence of unions, that

have resulted in the complex situation at work providing the grounds for the development of the new epidemics. In chapter 3, the important emerging issue of occupational violence is covered where the author discusses the different scenarios that constitute occupational violence. These include overt aggression and also the more devious and covert forms of behaviour that constitute violence or bullying in the workplace.

Precarious employment or contingent employment is the subject of chapter 4. The increase in less secure or more flexible types of employment over recent years has been well documented. This chapter discusses the ability of preventive occupational health and safety strategies and workers' compensation/rehabilitation processes to be effective in these circumstances. Chapter 5 brings attention to the diseases that have a long latency period and gives a socio-historical approach as to why these epidemics are continuing. Of particular concern is the continued exposure in developing countries to hazards that are reasonably well controlled in many industrialised countries. Four case studies of epidemics are provided to illustrate the issues. Adolescent and child labour is the subject of chapter 6. Evidence is provided to indicate that young people working in family or small businesses are at higher risk and that often the records for illness and injury in this type of working environment are poorly recorded. Chapter 7 deals with the pervasive issue of stress as part of occupational health and safety

considerations. The author mentions the move away from the biomedical and psychological approaches to the more workplace management/organisational approach. There is an interesting section on electronic monitoring as a form of stress. Some 'best-practice' approaches to stress management are provided.

Pain and prolonged standing are the subject of chapter 8. The authors of this chapter present a case for the silent pain from prolonged standing being partly responsible for musculoskeletal injuries, as well as potentially linked to a future epidemic of cardiovascular problems. Some of the problems posed by standing for long periods could be alleviated by applying the same principles used in the ergonomic design of workstations. Chapter 9 focuses on the potential social injury resulting from return-to-work programs where workers might be subjected to abuse. This is a form of injury resulting from occupational health and safety policy and practice concerned with managing the return-to work phase after a period away from work recovering from an injury or disease. The next chapter is concerned with the threat to the viability of rehabilitation programs because of the increasing costs and times taken for workers to return to work. The author discusses the situation in Australia where the compensation and rehabilitation process has shifted from employers to external professionals where there is a tendency for costs to escalate. The steps to

be taken before the epidemic strikes are discussed in chapter 11. Scenarios are analysed where baseline empirical studies could be useful in preventing new epidemics emerging. Some guidance is provided on how to go about conducting the necessary research when first cases emerge. The final chapter is a summary of the approaches discussed in the book to identify the new epidemics. Three different approaches are needed to appreciate the background to the identification of an epidemic and its subsequent acceptance.

Overall, the book is a very useful and interesting contribution to the understanding of the recent issues in occupational health and safety, particularly those associated with increasing internationalisation and the emergence of an increasing global economy. It is clear that more effort needs to be devoted to understanding the identification of the new epidemics, their evaluation, and the potential strategies for their prevention. The book is recommended for those with an interest in, or responsibilities for, managing current and emerging occupational health and safety issues associated with the organisation of work and the labour process.

Jeffery T. Spickett  
Curtin University of Technology  
Perth, Western Australia  
Email: j.spickett@curtin.edu.au

## Environmental Health: From Global to Local

Howard Frumkin (Ed.)

Jossey-Bass, San Francisco, 2005, 1108 pp. ISBN 0-7879-7383-1, \$115.95

Over the last few years a number of general, introductory environmental health textbooks have been released. Some of these have been reviewed in this Journal (cf vol. 2, no. 1, pp. 86-7; vol. 3, no. 2, p. 104; and vol. 4, no. 1, p. 97). *Environmental Health: From Global to Local* is the latest addition to this collection. With such a range of textbooks now available, it does make it difficult to select one text over another. However, if size is anything to go by, at 1108 pages, this latest text is more than twice the length of the previous texts reviewed.

As indicated by the length of the text, *Environmental Health: From Global to Local* provides an extremely comprehensive overview on the broad field of environmental health. To help present such a large range of information in a cohesive manner, the text uses the concept of spatial scales as the framework on which to base the structure of the book. After introducing the methods and paradigms of environmental health in Part 1 (chapters 1 to 9), specific issues are then addressed in the remaining Parts, starting with global issues (chapters 10 to 13), then regional issues (chapters 14 to 18), and then local issues (chapters 19 to 28). The final Part of the text describes the practice of environmental health (chapters 29 to 36).

In regard to content, the text provides a comprehensive coverage of the standard environmental health issues, for example, air pollution, energy, water, waste, and food safety. Not only standard environmental health methods and tools, for example, toxicology, epidemiology, exposure assessment, risk assessment, and risk communication, are covered, but also a

number of novel and interesting chapters on topics, such as environmental psychology, genetics and environmental health, environmental health ethics, environmental justice, religious approaches to environmental health, population pressure, and war.

Two of the sections that are particularly interesting are the introduction, which provides an excellent summary of what environmental health is, a history of environmental health, emerging issues, and the forces that drive environmental health, and the chapter on prevention. Initially, I was a little puzzled about why this chapter would be included, but I found that it nicely consolidated a range of fundamental concepts and provided an excellent discussion on how we should manage environmental health issues by using the precautionary principle as the basis on which we should act.

Often there is inconsistency within texts like this that cover a broad range of topics and include contributions by a number of different authors (57 contributors were used for this text). However, this text is surprisingly consistent throughout and this reflects a very high editorial standard. If I was to find fault, it would be that the page layout and design can sometimes make it difficult to follow the main text due to the way in which the explanatory boxes are placed, and often flow on to the following page/s. The text is also somewhat focused on American issues, legislation and management. However, when considering the total package that the text represents, these are relatively minor quibbles.

Overall, *Environmental Health: From Global to Local* is an exceptional text that provides a comprehensive and up-to-date analysis of the global to local environmental health issues we face, and it provides practitioners with best-practice advice on how to manage these issues. With all the chapters written by the leading authorities in their fields and the

topics elegantly woven together, this text is sure to be a pre-eminent reference text for many years to come. I would therefore recommend it to all environmental health practitioners and students.

*Thomas Tenkate*

*School of Public Health*

*Queensland University of Technology*

*Email: t.tenkate@qut.edu.au*

[Back to  
TOC](#)

# Environmental Health

The Journal of the Australian Institute of Environmental Health

## Environmental Health Subscription Form

**Annual Subscription Rates:** four electronic issues per year (These rates are subject to change)

### Within Australia (includes GST)

Individual rate	AUD \$180.00	<input type="checkbox"/>
Student rate	AUD \$100.00	<input type="checkbox"/>
Institutional rate	AUD \$300.00	<input type="checkbox"/>
IP Access rate*	AUD \$500.00	<input type="checkbox"/>

IP Address (IP Access rate only): \_\_\_\_\_

### Overseas (GST does not apply)

Individual rate	AUD \$ 160.00	<input type="checkbox"/>
Institutional rate	AUD \$ 270.00	<input type="checkbox"/>
IP Access rate*	AUD \$ 480.00	<input type="checkbox"/>

\*Please include your IP Address below

## Subscriber Contact Details

Name: \_\_\_\_\_  
Institution: \_\_\_\_\_  
Address: \_\_\_\_\_  
Postcode: \_\_\_\_\_  
Telephone: \_\_\_\_\_ Email: \_\_\_\_\_

## Payment Details

☐ Please find a cheque enclosed made payable to AIEH Or

☐ Please charge my Credit Card: ☐ Bankcard ☐ Mastercard ☐ Visa

Card Number:

Expiry Date:   /

Cardholder's Name: \_\_\_\_\_ Signature: \_\_\_\_\_

## Journal Contact Details

Please send completed forms/payment to:

Bernadet Ferraro (National Finance Officer)  
Australian Institute of Environmental Health  
PO Box 378, Diamond Creek  
Victoria 3089 AUSTRALIA  
P: (03) 9438 5960  
F: (03) 9438 5955

For all other enquiries:

Jim Smith (Editor)  
Jaclyn Huntley (Editorial Assistant)  
PO Box 225, Kew  
Victoria 3101 AUSTRALIA  
P: (03) 9855 2444  
F: (03) 9855 2442

**Back to  
TOC**

## Environmental Health

---

The Journal of the Australian Institute of Environmental Health

### Guidelines for Contributors

#### **Manuscripts**

Manuscripts should be submitted to Jim Smith, Editor, Environmental Health, PO Box 225, Kew, Victoria, 3101, Australia.

Material will be considered for publication on the understanding that it is original and unpublished work and has not been submitted for publication elsewhere. Authors are responsible for all statements made in the material. Papers accepted for publication become the copyright of the Journal but release for publication elsewhere can be applied for on the understanding that acknowledgment is made to the Journal.

#### Preparation of Manuscripts

Manuscripts should in general conform to the style outlined in the Australian Government Publishing Service 1994 *Style Manual for Authors, Editors and Printers*, 5th edn, AGPS, Canberra. Spelling should conform to the Macquarie Dictionary.

#### Submission of Manuscripts

Articles should not normally exceed 5000 words. Reflections on practice, reports, views and discussion, policy analysis and other material should not normally exceed 3000 words. Authors should forward the manuscript electronically to the editor. A covering letter should identify the author to receive correspondence, including mail and email addresses, telephone and facsimile numbers. Upon acceptance of the manuscript, authors will be requested to submit the document. Manuscripts should generally conform to the following sequence: title page; abstract; text; acknowledgments; endnotes; references; tables and figures, contact details including affiliations and full postal addresses for ALL authors, and telephone, facsimile and email address for contact author.

#### Title Page

The title page should include the manuscript title, names, institutional affiliations, and academic qualifications of authors (please give complete details including addresses).

#### Abstract

All articles should include an abstract. The abstract should summarise the paper in 200 words or less. Abstracts can be reprinted in other publications and data bases so that it is important to include the main purpose, content, and conclusions of the article. Up to six key words should be included.

#### Text

Articles should not normally exceed 5000 words. As the Journal is multidisciplinary, the presentation of material should conform to the standard format according to the particular discipline. Other entries in the Journal, reviews, case reports, editorials, discussion, should not normally exceed 3000 words and are likely to require a different format. Please consult with the editor for guidance.

#### Tables and Figures

Submit three hard copies of tables and figures as black and white prints preferably 80 x 80 mm but no larger than 180 X 250 mm. *Environmental Health* will be happy to produce tables and figures if data and type of table or figure required (i.e., bar chart, line graphs) are supplied. If tables or figures are to be reproduced please supply full details of source. Titles and captions of tables and figures should be placed on the actual table or figure. Figures may be from original artwork, photographs, graphs or charts.

Examine all figures carefully to ensure that the data are presented with the greatest possible clarity to help the reader to understand the text. Similarly, determine if a figure would communicate the information more effectively than narrative. Photographs, which disclose their identity, must be accompanied by signed permission.

Each table and figure must be produced on a separate page, double spaced, numbered consecutively, and given a title. Each table and figure must be cited in the text and its position indicated.

#### Acknowledgments

Acknowledgments should be typed on a separate page, following the text. Where appropriate give credit to grantors, sponsors, technical assistants, and professional colleagues.

#### Endnotes

Notes which are in addition to references should be used sparingly. They can be numbered in superscript in the text and then listed as Endnotes before the Reference List at the end.

#### References

References should conform to the Australian Government Publishing Service 1994, *Style Manual for Authors, Editors and Printers*, 5th edn, AGPS, Canberra. Examples of referencing can be obtained from the Editor.



