

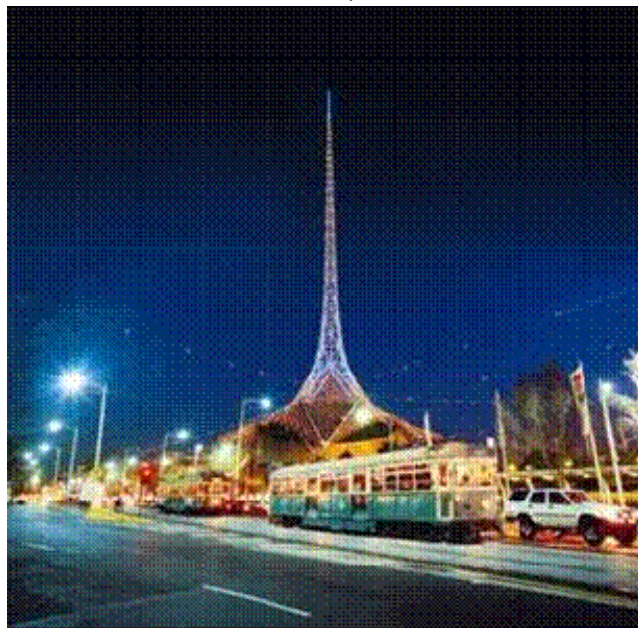
CONFERENCE PROCEEDINGS

STRIVING FOR EXCELLENCE

25th Annual Conference of the Australian Institute of
Occupational Hygienists

1st to 5th December 2007

Grand Hyatt
Melbourne, Victoria



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**25th Annual Conference of the
Australian Institute of Occupational Hygienists
& Annual General Meeting**

Conference Proceedings 2007

STRIVING FOR EXCELLENCE

Editors: Dr Geza Benke and Dave Collins

AIOH 25th Annual Conference
1st to 5th December 2007
Grand Hyatt Hotel, Melbourne, Victoria
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Minimal alterations have been made to original papers in order to conform to uniform style.

In compiling the Scientific Programme the Conference Committee drew upon a wide range of disciplines and would like to thank our international speakers, CES and session presenters for their contributions:

Dr Geza Benke & Dave Collins
Editors

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INVITATION TO THE 25th ANNUAL CONFERENCE OF THE AUSTRALIAN INSTITUTE OF OCCUPATIONAL HYGIENISTS

The first AIOH Conference was in Melbourne in 1982 and it has returned for the 25th conference to our fabulous cosmopolitan Australian city. It is a pleasure to welcome you to the AIOH Conference 2007 at the Grand Hyatt Hotel, Melbourne

The profession of occupational hygiene is constantly striving for excellence both in science and in the protection of people and the environment. It is on this theme that the AIOH's 2007 Conference is based - "Striving for Excellence".

This year's conference will revolve around the changes that are constantly occurring within the occupational hygiene profession and the assessment and management of occupational health risks within the workplace. "Striving for excellence" is reflected in the plenary and concurrent sessions throughout the conference.

The Continuing Education Sessions (CES) on the preceding weekend cover a wide range of subjects. These allow delegates to maintain knowledge and develop skills in a variety of disciplines and fields of interest as they "strive for excellence".

Of particular interest this year will be the role of risk assessment and management within occupational hygiene and its allied fields. The Safety Equipment Australia Keynote Speaker will be Dr Dennis Paustenbach CEO of ChemRisk, USA who is a world renowned expert on chemical risk assessment.

This year will once again see the presentation of the ever popular "Basics Principles of Occupational Hygiene" course. Designed for newer members or members of allied professions, to introduce and stimulate interest in new and old fields of study without the higher level technical content.

Following from last year's successful interactive panel session, we have again included a similar session to address an area often met in the public arena – Risk Communication. This session will be facilitated by Katherine The-White a leading communicator. It is guaranteed to be an entertaining and informative presentation.

- This year we are introducing some new innovations:
- AIOH Conference Ambassadors – to provide an identifiable warm, friendly 'experienced' face for the first-timers at the AIOH Conference to assist them achieve the maximum benefit from the exhibition, networking and conference
- The proceedings will be contained on your conference gift of an USB. This will allow the minimum printing of hard-copies of the proceedings and conserve our planet's resources.
- Enviro-friendly re-usable conference bags
- High visibility vests for clear identification of conference committee members

The Institute and the Conference Organising Committee would like to thank the Institute's generous sponsors, loyal exhibitors, all speakers and session chairpersons who will make this conference a very successful one.

I am sure you will find the 25th annual AIOH conference here in Melbourne a conference to remember. I encourage you to not only absorb the knowledge and information presented but also enjoy the stimulating and sometimes challenging company of your peers and surroundings our city for the next few days.

Stephen Dean
Conference Chairperson

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THE DEVELOPMENT OF AN AUSTRALIAN DATABASE OF WOOD DUST EXPOSURES: ISSUES AND FUTURE DIRECTIONS

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ABSTRACT

The development of national exposure and control databases for hazard surveillance has been the subject of increasing interest in Australia. In late 2006, the University of Adelaide was tasked with the construction of a database framework, as well as a profile of wood dust exposures in the Australian wood industries. This paper provides a perspective on the feasibility of collecting existing data from various sources, and provides a comparison with the UK approach.

Information on the exposed workforce and wood usage was gathered from a combination of ABS data and a pseudo random telephone survey of 201 relevant businesses listed in the Yellow Pages. Exposure and control data were gathered from regulatory agencies, industry and research groups and hygiene consultants.

About two thirds of Yellow Pages-listed companies in South Australia had less than 5 employees, and, depending on the size of the company, about 75% of employees are exposed to wood dust, mostly in the form of reconstituted timber and softwood.

Exposure to inhalable wood dust, as represented by 521 discrete TWA personal measurements across all wood industries yielded a median value of 2.1, an arithmetic mean of 5.8 and range of 0.06 – 210 mg/m³. This distribution is similar to a 1999/2000 UK-wide targeted (purposive) survey where the corresponding figures were 2.6, 6.6 and 0.05 – 157 respectively. A feature of the exposure dataset was multi-tasking (23% of the personal measurements) and mixed wood exposure (37%).

The availability of exposure data from government and consultant sources was variable. It appears likely that such sampling is undertaken infrequently but it is also possible that scientific reports and data are not readily available for reasons of commercial confidentiality or were judged by providers not to be in a form that can be included in a systematic exposure database. It is apparent that simply requesting information may result in a potential bias, particularly as many small businesses would not have conducted monitoring.

In view of these limitations, it is concluded that targeted survey work, as has been conducted in the UK, is a more reliable strategy for exposure profile development and trend assessment.

Key words: Australia, database, exposure, wood dust

INTRODUCTION

Australian wood industries including furniture manufacturing, cabinet making and joinery, saw milling, particle board manufacturing and paper manufacturing are important with respect to economic development and employment in Australia. In Australia, a variety of woods are used, and they can be classified as solid timber including hardwoods (i.e. native species and imported species) and softwoods, reconstituted softwoods (particleboard and medium density fibreboard; MDF) and engineered wood materials.

Exposure to wood dust can cause respiratory disease, dermatitis (Pisaniello *et al.*, 1991; Mandryk *et al.*, 1999), cancer (IARC, 1995; HSE, 2005; ACGIH, 2007; Bornholdt *et al.*, 2007), idiopathic pulmonary fibrosis (IPF) (Gustafson *et al.*, 2007) and nasal symptoms (Pisaniello *et al.*, 1991, 1995; Rabone and Saraswati, 1999; Mandryk *et al.*, 1999, 2000). The Australian Safety and Compensation

Council (NOHSC, 1995) notes that wood dust is a sensitiser causing specific immune response in some people.

The personal time weighted average (TWA) inhalable dust concentration is currently the best metric for wood dust exposure. The Australian Occupational Exposure Standard (NOHSC, 1995) for wood dust (8 hr TWA of 1 mg/m³ for hardwoods (such as beech, oak) and 5 mg/m³ for softwoods) is based on the inhalable dust fraction captured by specified sampling devices. Ideally, measurements should be carried out in accordance with Australian Standard 3640 – 2004 (or equivalent) (Standards Australia, 2004), although there is some debate about the specific details of sampling devices (Harper *et al.*, 2004).

In order to protect workers in wood industries, the profile and extent of occupational exposure to wood dust need to be understood. Recently, the Health and Safety Executive in the UK (Black *et al.*, 2007) and 25 member states of the European Union (Kauppinen *et al.*, 2006) released survey results for the estimation of occupational exposure to inhalable wood dust in the wood industry. In the USA, a number of large studies have been conducted, including reports pertaining to small woodworking shops (Brosseau *et al.*, 2001). In Australia, there have been several studies on wood dust exposure including those in the furniture manufacturing industry and sawmilling industries (Pisaniello *et al.*, 1991; Mandryk *et al.*, 1999, 2000).

The development of Australian exposure and control databases for hazard surveillance has been the subject of increasing interest. In 2006, the Australian Safety and Compensation Council (ASCC) selected wood dust as a test case, and the University of Adelaide was subsequently tasked with the construction of a database framework, as well as a profile of wood dust exposures in the Australian wood industries. This paper, therefore, provides a perspective on the feasibility of collecting existing data from various sources to assess occupational exposure to wood dust in wood industries, and provides a comparison with the UK approach.

METHODOLOGY

Data collection

Wood dust exposure and control data were compiled from journal articles, conference proceedings, research reports, consulting reports, government sources and industry reports throughout Australia. Construction, educational and domestic/hobby activities involving wood dust were excluded. Classifications of industry and tasks were adapted from the Australian and New Zealand Standard Industrial Classification (Australian Bureau of Statistics, 2006) and Jones and Smith (1986).

All data were classified using a variety of factors including industry type (i.e. primary; sawmills & board manufacture, furniture industry and cabinet making/joinery), wood types (i.e. solid timber, reconstituted and mixture of solid/reconstituted), business size (i.e. small; <20, medium; 20-<100 and large; ≥100), tasks, controls (i.e. local exhaust ventilation; LEV) and cleaning (i.e. sweeping, compressed air and vacuuming) and personal protective equipment (i.e. respirator).

Only data derived from inhalable dust measurements were entered into the database. Discrete measurements, rather than aggregate data, reported in the data sources, were utilized for calculations and comparisons.

Telephone survey

In order to gather information on the exposed workforce, relevant businesses (i.e. kitchen renovators, cabinet makers, wooden window frame manufacturers, wood flooring contractors, carpenters and joiners and veneer users) where workers were likely to be exposed to wood dust were selected from categories in the on-line Yellow Pages[®] for SA. Businesses contacted were

briefly asked about total numbers of employees in the organization, the number of employees substantially exposed to wood dust and the types of wood used in that business. Business names were kept confidential.

Statistical analysis

Wood dust exposures tend to follow a lognormal distribution (Pisaniello *et al.*, 1991), rather than a normal distribution. Results were expressed as arithmetic means (AM), geometric means (GM) and medians with ranges. Data were entered into Excel spreadsheets, which were also used to generate descriptive statistics.

RESULTS

General information

Accurate estimates of the workforce size of the wood industry are not available. However, ABS data for 2004/5 in ANZSIC codes 23 and 29 (i.e. relevant wood industries) indicate approximately 100,000 persons of which 10,000 reside in SA, roughly reflecting the population proportion (7.5% for SA in 2004; 1.5 million versus 20.1 million for Australia) (ABS, 2006). It is of interest to note that about 60% of companies in the wood and paper product manufacturing category have less than 100 employees.

Out of our contacts, less than 11% (i.e. 3 out of 67 occupational hygiene consultants, 5 out of 8 government OHS agencies) provided relevant exposure data.

From the telephone survey, 420 businesses in South Australia were contacted, and 48% responded. Sixty-seven percents of the businesses had less than 5 employees, and 27% had employees in between 5 and 20. About 75% of employees were exposed to wood dust, mostly in the form of reconstituted timber and softwood.

Exposure to inhalable wood dust, as represented by 521 discrete time-weighted average personal measurements across all wood industries yielded a median value of 2.1, an arithmetic mean of 5.8 and range of 0.06 – 210 mg/m³.

The ranges of personal wood dust exposures in industry categories were;

1. primary wood industries including sawmills & board manufacture (0.06-113 mg/m³, median: 1.5 mg/m³, geometric mean: 1.7 mg/m³, arithmetic mean: 5.6 mg/m³),
2. furniture industry (0.1-210 mg/m³, median: 2.3 mg/m³, GM: 2.4 mg/m³, AM: 5.2 mg/m³) and
3. cabinet making/joinery (<0.3-47 mg/m³, median: 3.3 mg/m³, GM: 3.0 mg/m³, AM: 6.8 mg/m³).

Percentages of types of woods used by the wood industry were 40% for solid timber, 23% for reconstituted and 37% for mixtures of these woods. Around 23% workers were engaged in multi tasks.

Distribution of wood dust exposures in between 1989 and 2005

From the data collected from 1989 to 2005, three periods of time were classified for the trend of wood dust exposures (Table 1). There appears to be a small reduction in wood dust exposures.

Figure 1 represents cumulative percentages with total wood dust exposures (mg/m³). Around 25% of wood dust exposures exceeded 5 mg/m³, and 27% were less than 1 mg/m³.

Table 1. Wood Dust Exposures in Periods between 1989 and 2005

Periods	No. of Samples	Wood Dust Exposure (mg/m ³)			
		Median	GM	AM	Range
1989-1994	200	3.0	3.2	6.4	0.1 - 210
1995-1999	165	1.4	1.7	4.2	0.06 - 67
2000-2005	156	1.7	2.0	6.8	0.1 - 113

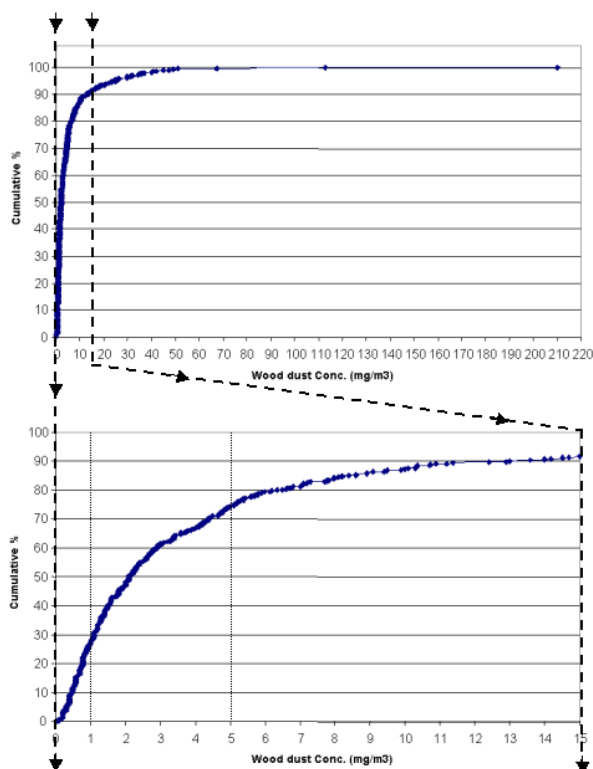


Figure 1. Cumulative Percentage of Wood Dust Exposures

Effects of wood type on exposure

Personal exposures to wood dust by wood type are given in the table below (Table 2). The use of MDF showed the highest statistical result of AM (8.0 mg/m³), although there was a limited number (n=27) of measurements.

Table 2. Wood Dust Exposure with Types of Woods Used

Types of Woods		Wood Dust Exposure (mg/m ³)				
		No. of Samples	Median	GM	AM	Range
Solid Timber	Hard Wood	140	2.2	2.4	7.0	0.1 - 210
	Soft Wood	69	1.3	1.7	4.5	<0.2 - 37
Reconstituted	MDF	27	1.0	1.4	8.0	<0.2 - 113
	Plywood	4	1.4	1.2	1.9	0.2 - 4.7
	Chipboard/ Particleboard	88	2.9	3.1	6.3	<0.2 - 42
Mixture		193	2.2	2.3	4.9	0.06 - 49

* AM: arithmetic mean, GM: geometric mean

* MDF: medium density fiberboard

Effects of tasks on exposure

Types of wood were compared with a variety of tasks for wood dust exposures. Most high levels of wood dust exposures occurred from sanding (GM: 4.0 mg/m³). Cleaning, sawing and moulding also likely to emit high levels of wood dust.

Effects of control and cleaning strategies on exposure

The use of local exhaust ventilation (LEV) would be expected to reduce dust exposures, and this type of information is often reported in occupational hygiene assessments.

The geometric mean for personal exposures to wood dust by wood type and ventilation are given in Table 3. It can be seen that exposures are uniformly lower when local exhaust ventilation is documented to be present and operating.

Table 3. Wood Dust Exposure with and without LEV Control

Types of Woods	Wood Dust Exposure (mg/m ³)					
	Controls (LEV)			Natural Ventilation or Not Specified		
	n	GM	Range	n	GM	Range
Solid	88	1.6	0.1 – 67	121	2.7	<0.3 – 210
Reconstituted	28	0.6	<0.2 – 7.1	91	3.7	<0.3 – 113
Mixture	69	2.1	0.06 – 49	124	2.4	<0.1 – 41

*n: Number of samples

From our database, it can be seen that exposures are uniformly higher when the use of compressed air is documented (Table 4).

Table 4. Wood Dust Exposure with and without Cleaning Processes

Types of Woods	Wood Dust Exposure (mg/m ³)								
	Compressed Air			Vacuum			Not Used or Not Specified		
	n	GM	Range	n	GM	Range	n	GM	Range
Solid	72	3.5	0.1 – 210	7	0.6	0.2 – 1.3	130	1.8	<0.2 – 67
Reconstituted	56	4.0	0.4 – 42	-	-	-	63	1.6	<0.2 – 113
Mixture	53	4.1	0.4 – 41	-	-	-	140	1.8	0.06 – 49

*n: Number of samples

Use of personal protective equipment

Respiratory protection was relatively uncommon. Approximately 3% of records had information about the use of respirators, and where mentioned, half face respirators were the respirators worn.

DISCUSSION AND CONCLUSIONS

Our study found that solid hardwoods were associated with higher dust concentrations than solid softwoods (GM=2.4 vs 1.7). It has been suggested that the use of MDF leads to greater levels of inhalable dust, because of intrinsically smaller wood particles in the matrix (Chung *et al.*, 2000). Our data (n=27) show moderate levels but with high variability (GM=1.4, AM =8). High levels of airborne wood dust were related to the absence of local exhaust ventilation (LEV), certain tasks (especially sanding), cleaning methods and use of compressed air. However, Black *et al.* (2007) pointed out that LEV itself cannot be relied on to provide adequate control, due to issues associated with poor connection, holes in filter bags, long distances between the source and capturing hood and unpredictable air flows. We found that only a small number of workers (3%) wore respirators as also reported in previous studies (Alwis *et al.*, 1999; Mandryk *et al.*, 1999, 2000; Black *et al.*, 2007).

In the UK (Creely *et al.*, 2006), the Health and Safety Executive (HSE) investigated long-term changes in inhalational exposure to wood dust over the last 10 years in a number of industries and the main causes of changes. For this investigation, the HSE reviewed the National Exposure DataBase (NEDB), collected wood dust samples and interviewed employers and staff. The trend of wood dust exposure in the UK between 1985 and 2003 was downward (around 8.0 % per annum, on average), due to hazards awareness, technology improvement and compliance with legislation. Our data also show a trend to lower exposures. The HSE report also emphasized the importance of continuing exposure data collection for future trends.

The Australian data are similar to that for the UK (Black *et al.*, 2007), where 27% of measurements (particularly sanding and circular sawing) exceeded the UK limit of 5 mg/m³. A feature of our exposure dataset was multi-tasking (23% of the personal measurements) and mixed wood exposure (37%). When compared with corresponding classifications in the UK (49% and 64%), it is apparent that wood dust exposure limits that differentiate hardwoods and softwoods pose assessment problems, because there is no analytical method which will allow the hard and soft wood dust to be separated. Thus, the Australian exposure standards for wood dust should be reviewed in order to

1. better reflect the current situation of multi-tasking and mixed wood exposures and
2. take account of differing health end points - the U.S. ACGIH have amended their exposure criteria (TLVs) to take into account factors such as sensitization (TWA: Western red cedar - 0.5 mg/m³, SEN).

To develop a national wood dust exposure database for Australian wood industries, the University of Adelaide contacted regulatory agencies, research groups, industries and occupational hygiene consulting companies/national association throughout Australia. Further information was obtained by conducting a telephone survey based on the Yellow Pages. However, there were low response rates from our contacts, likely due to commercial confidentiality or limited data. It is apparent that simply requesting information may result in a potential bias, particularly as many small businesses would not have conducted monitoring. In any case, the available data were often the results of research projects, carried out from 1989 to 1999.

It is suggested that information on workforce characteristics, exposures, controls and work practices in the Australian wood industry, should be gathered by targeted (purposive) sampling.

REFERENCES

ACGIH, 2007, *TLVs and BEIs; Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices*, American Conference of Governmental Industrial Hygienists.

- Alwis U., Mandryk J., Hocking A.D., Lee J., Mayhew T. and Baker W., 1999, Dust Exposure in the Wood Processing Industry, *Am. Ind. Hyg. Assoc. J.*, 60, 641-646.
- ABS, 2006, 1292.0 - Australian and New Zealand Standard Industrial Classification 9ANZSIC) 1993, Australian Bureau of Statistics Updated in June 2006.
- Black N., Dilworth M. and Summers N., 2007, Occupational Exposure to Wood Dust in the British Woodworking Industry in 1999/2000, *Ann. Occup. Hyg.*, 51(3), 249-260.
- Bornholdt J., Saber A.T., Sharma A.K., Savolainen K., Vogel U. and Wallin H., 2007, Inflammatory response and Genpototoxicity of Seven Wood Dusts in the Human Epithelial Cell Line A549, *Mutation Research*, 632, 78-88.
- Brosseau L.M., Parker D., Lazovich D., Dugan S., Milton T. Pan W., 2001, Inhalable Dust Exposures, Tasks, and Use of Ventilation in Small Woodworking Shops: A Pilot Study, *Am. Ind. Hyg. Assoc. J.*, 62, 322-329.
- Chung K.Y., Cuthbert R.J., Revell G.S., Wassel S.G. and Summers N., 2000, A Study on Dust Emission, Particle Size Distribution and Formaldehyde Concentration during Machining of Medium Density Fibreboard, *Ann. Occup. Hyg.*, 44(6), 455-466.
- Creely K.S., Tongeren M.V., While D., Soutar A.J., Tickner J., Agostini M., de Vocht F., Kromhout H., Graham M., Bolton A., Cowie H. and Cherrie J.W., 2006, *Trends in Inhalation Exposure: Mid 1980s till present*, Research Report 460, Institute of Occupational Medicine, Health and Safety Executive (HSE).
- Gustafson T., Dahlman-Höglund A., Nilsson K., Ström K., Tornling G. and Torén K., 2007, Occupational Exposure and Severe Pulmonary Fibrosis, *Respir. Med.*, doi:10.1016/j.rmed.2007.02.027.
- Harper M., Akbar M.Z. and Andrew M.E., 2004, Comparison of wood-dust aerosol size-distributions collected by air samplers, *J. Env Mon.*, 6, 18-22.
- HSE, 2005, *Asthma and Other Respiratory Diseases*, Health and Safety Executive Statistics. URL: <http://www.hse.gov.uk/statistics/causdis/asthmaindex.htm>.
- IARC, 1995, *Monographs on the Evaluation of Carcinogenic Risks to Humans*, Vol. 62, Wood Dust and Formaldehyde, Lyon, International Agency for Research on Cancer.
- Jones P.A. and Smith L.C., 1986, Personal Exposure to Wood Dust of Woodworkers in the Furniture Industry in the High Wycombe Area: A Statistical Comparison of 1983 and 1976.77 Survey Results, *Ann. Occup. Hyg.*, 30, 171-184.
- Kauppinen T., Vincent R., Liukkonen T. et al, 2006, Occupational Exposure to Inhalable Wood Dust in the Member States of the European Union, *Ann. Occup. Hyg.*, 50(6), 549-561.
- Mandryk J., Alwis K.U. and Hocking A.D., 1999, Work-Related Symptoms and Dose-Response Relationships for Personal Exposures and Pulmonary Function Among Woodworkers, *Am. J. Ind. Med.*, 35, 481-490.
- Mandryk J., Alwis K.U. and Hocking A.D., 2000, Effects of Personal exposures on Pulmonary Function and Work-Related Symptoms Among Sawmill Workers, *Ann. Occup. Hyg.*, 44(4), 281-289.

NOHSC, 1995, *Exposure Standards for Atmospheric Contaminants in the Occupational Environment*, National Occupational Health and Safety Commission, WorkSafe, Canberra.

Pisaniello D.L., Connell K.E. and Muriale L., 1991, Wood Dust Exposure during Furniture Manufacture-Results from an Australian Survey and Considerations for Threshold Limit Value Development, *Am. Ind. Hyg. Assoc. J.*, 52(11), 485-492.

Pisaniello D.L., Gun R.T., Tkaczuk M.N., Schultz M.R. and Stevens M.W., 1995, Nasal Cytology in Australian Furniture Woodworker, *Aust. J. Otolaryng*, 2(2), 137-141.

Rabone S.J. and Saraswati S.B., 1999, Acceptance and Effects of Nasal Lavag in Volunteer Woodworkers, *Occup. Med.*, 49 (6), 365-369.

Standard Australia, 2004, *Workplace Atmospheres - Method for the Sampling and Gravimetric Determination of Inhalable Dust*, AS 3640 – 2004, Sydney.

SILICA ESSENTIALS, APPLYING THE CONTROL BANDING APPROACH TO QUEENSLAND QUARRIES – THE WAY FORWARD!

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1. ABSTRACT

In Queensland, quarry operators are potentially exposed to freshly cut quartzite. Alpha quartz is a form of crystalline silica, which has been shown to cause silicosis. In the United Kingdom COSHH Essentials has been broadened to provide information for operations where crystalline silica can potentially become airborne. These operations include quarries. [Direct guidance](#) is available from the HSE website including [silica essentials](#) which may be applicable to the Australian industry and regulatory bodies.

This paper discusses and analyses whether COSHH guidance material has a benefit to the Queensland Quarrying Industry under the Queensland Mining and Quarrying Safety and Health Regulation (2001).

2. INTRODUCTION

In the United Kingdom, a review undertaken in 2002 by the Health and Safety Executive (HSE) revealed unacceptable risks for workers exposed to respirable crystalline silica at the workplace exposure limit (WEL) which, at that time, was 0.3 mg m^{-3} . In fact, the HSE cited a study that indicated a 20% risk for developing silicosis at this limit.

3. DISCUSSION

In 2002 the European Scientific Committee on Occupational Exposure Limits (SCOEL), made a recommendation to the European Commission. SCOEL noted that to reduce the incidence of silicosis, the Occupational Exposure Limit (OEL), would have to be set below 0.05 mg.m^{-3} (HSE, 2005).

This recommendation challenged the adequacy of the UK WEL and the HSE therefore considered it prudent that they develop a more stringent regulatory position on respirable crystalline silica.

A regulatory impact assessment was carried out, in 2005, looking at a cost benefit analysis of four potential WEL values ranging from 0.3 mg.m^{-3} (the UK Maximum Exposure Limit in 2005), 0.1 mg.m^{-3} , and 0.05 mg m^{-3} to 0.01 mg m^{-3} . This analysis resulted in the revised UK WEL being set, in 2006, for RCS as 0.1 mg m^{-3} (ACTS 2006). This is twice the level recommended by SCOEL.

The HSE (2005), notes that the sampling and analytical methods for quartz MDHS 101, (which has now replaced MDHS 14/3, 37, 51/2), for a 4 hour air sample, gives satisfactory level of precision at 0.3 mg m⁻³. At 0.1 mg m⁻³, the precision is poor but still adequate for compliance monitoring and enforcement. At 0.05 mg m⁻³, the results may fall short of the standard required for legal proof because of the unacceptable level of precision, that is, for a 4-hour sampling period. **An 8-hour sampling period will, however, provide an acceptable level of precision at 0.05 mg m⁻³.**

Measurement at 0.01 mg m⁻³ is just practicable for laboratory test filters but not for field samples. WELs of 0.05 or 0.01 mg m⁻³ would not be enforceable given the limitations of sampling and analytical methods. In their assessment the HSE noted that reducing the concentration to 0.1 mg m⁻³, would result in radiological silicosis being prevented for 547 cases and for fatal silicosis, 187 premature fatalities would be prevented (HSE, 2005). The study reported by the HSE (2002) identified a 2.5% risk of developing silicosis 15 years post-exposure as indicated by an ILO score 2/1+ (HSE, 2002). As a result the UK Control of Substances Hazardous to Health (COSHH) Regulation was significantly updated in April 2005.

The principles of good control have been incorporated in schedule 2A and aligned with Clause 7 (7) of The UK Control of Substances Hazardous to Health Regulations 2002.

The Queensland Mining and Quarrying Safety and Health Regulation (2001) lists in schedule 5 “general exposure limits” for airborne hazards. These “general exposure limits” are based on the Australian Safety and Compensation Council (ASCC) “Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment (NOHSC:1003)”. Schedule 5 lists the “general exposure limit” for crystalline silica at 0.1 mg m⁻³.

Section 135 clause (1) of Queensland Mining and Quarrying Safety and Health Regulation (2001) states that:

“Limiting workers exposure:

The site senior executive must ensure a worker’s exposure to a hazard at the mine—

- a. does not exceed the exposure limit applying to the worker for the hazard; and*
- b. is as low as reasonably achievable”.*

Occupational Exposure Limits (OEL) represents airborne concentrations to individual chemical substances which, according to current knowledge, should neither impair the health of, nor, cause undue discomfort to, nearly all workers. Occupational Exposure Limits are not fine dividing between safe and unsafe exposures.

The definition of **as low as reasonably achievable** (ALARA) is a basic radiation protection concept or philosophy. It is an application of the "Linear No Threshold Hypothesis," which assumes that there is no "safe" dose of radiation. Applying this same concept to respirable crystalline silica, the probability for lung disease increases with increased respirable crystalline silica exposure, no matter how small. Therefore, it is important to keep exposure to respirable

crystalline silica, as low as is reasonably achievable, using proven control technologies. Air monitoring and health surveillance must be undertaken to evaluate the effectiveness of control.

Some examples of controls in Queensland Quarries include:

Remote monitoring of crusher from camera within control room.		
Enclosed crushing and screening plants.		
Dust extraction systems.	Water sprays.	Road watering.
Curtains.	Fogging sprays.	Wind barriers.
Conveyor covers.	Air-conditioned control rooms.	Air-conditioned vehicle cabins.
Stockpile discharge socks.	Stockpile sprinklers.	Screen deck covers.
Wetting agent.	Muck pile watering.	
Worker rotation between dusty and non-dusty jobs.		
Respiratory protection.		

According to the COSHH Regulations, control of exposure is only adequate if:

- The principles of good practice are applied, and
- The Occupational Exposure Limit is not exceeded.

The Queensland Mining and Quarrying Safety and Health Regulation (2001) requires that a risk assessment be undertaken and where the level of risk is unacceptable, the exposure shall be controlled. Quarrying operations potentially are dusty.

Crystalline silica is ubiquitous, unless principles of good control practices are implemented, quarry operators may be at risk of exposure to respirable crystalline silica at concentrations that may be hazardous to health.

Good control practices according to COSHH include:

1. *Design and operate processes and activities to minimize emission, release and spread of substances hazardous to health.*
2. *Take into account all relevant routes of exposure- inhalation, skin absorption and ingestion- when developing control measures.*
3. *Control exposure by measures that are proportionate to the health risk*
4. *Choose the most effective and reliable control options which minimize the escape and spread of substances hazardous to health.*
5. *Where adequate control of exposure cannot be achieved by other means, provide, in combination with other control measures, suitable personal protective equipment.*
6. *Check and review regularly all elements of control measures for their continuing effectiveness.*

7. *Inform and train all employees on the hazards and risks from the substances with which they work and the use of control measures developed to minimize the risks.*
8. *Ensure that the introduction of control measures does not increase the overall risk to health and safety.*

(The Control of Substances Hazardous to Health (Amendment) Regulations 2004 Schedule 2A).

More specific guidance on control is available from the HSE website called Essentials Guidance Publications available online at <http://www.hse.gov.uk/pubns/guidance/index.htm>. The information provides comprehensive guides to assist quarries manage their risk, and includes:

<i>COSHH Essentials in quarries: Silica</i>	<i>COSHH Essentials relating to health surveillance.</i>	<i>COSHH Essentials relating to engineering control.</i>
<i>QY0 : Advice for managers. QY1 : Rock drilling. QY2 : Excavating and haulage. QY3 : Crushing. QY4 : Drying and cooling. QY5 : Dry screening. QY6 : Dry grinding. QY7 : Jumbo bag filling. QY8 : Silica flour: Small bag (15-50 kg) filling and transfer. QY9 : Cleaning up silica dusts. QY10: Control cabins and vehicle cabs.</i>	<i>G401 : Health monitoring for chronic obstructive pulmonary disease. G402 : Health surveillance for those exposed to respirable crystalline silica.</i>	<i>G406 : New and existing engineering control systems.</i>

4. CONCLUSION

COSHH Essentials is a good guide for managers to use in reducing the risks from exposure to respirable crystalline silica in quarries. The guide provides practical solutions that can be used by the industry and regulators to limit workers exposure to respirable crystalline silica to a level that is as low as reasonably achievable. Collecting 8-hr samples will allow for measuring airborne concentrations of RCS at or below 0.05 mg m⁻³.

5. REFERENCES

Advisory Committee on Toxic Substances (ACTS) 2006, proposed workplace exposure limit for respirable crystalline silica: results of public consultation and recommendations to HSC.

Retrieved 2 November from:

<http://www.hse.gov.uk/aboutus/hsc/iacs/acts/030506/acts032006.pdf>

Australian Safety and Compensation Council (ASCC) Hazardous Substances Information System.

Retrieved 30 October 2007 from <http://www.ascc.gov.au/>

HSE (2002). Guidance note, environmental hygiene 75/4. (GNEH75/4 Respirable crystalline silica – phase 1 hazard assessment.

HSE (2005). A Regulatory Impact Assessment (RIA) on proposals to reduce the UK Occupational Exposure Limit for Respirable Crystalline Silica (RCS) - draft for consultation

Queensland Mining and Quarrying Safety and Health Regulation (2001). Retrieved 30 October 2007 from

(<http://www.legislation.qld.gov.au/LEGISLTN/CURRENT/M/MiningQuaSHR01.pdf>)

The Control of Substances Hazardous to Health (Amendment) Regulations, 2004. Retrieved 30 October 2007 from <http://www.opsi.gov.uk/si/si2004/20043386.htm>

UK Health and Safety Executive (HSE) Control of Substances Hazardous to Health (COSHH) Essentials guidance publications. Retrieved 30 October 2007 from <http://www.hse.gov.uk/pubns/guidance/index.htm>

AIRBORNE CRYSTALLINE SILICA (RCS) IN QUEENSLAND QUARRYING PROCESSES, PARTICLE SIZE AND POTENCY.

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1. ABSTRACT

In Queensland, quarry operators are potentially exposed to freshly cut quartzite. Alpha quartz is a form of crystalline silica, which can lead to silicosis. There is also a growing body of evidence to suggest that respirable crystalline silica (RCS) is one of the causative agents that leads to chronic obstructive pulmonary disease (COPD). This paper presents findings from an ongoing literature survey, to explore if the current method of sampling for RCS in Australia for gravimetric determination of respirable dust compares to international practices. This paper also provides an update on particle size, crystal properties and potency. Information presented, can be used to raise awareness about the hazardous nature of RCS in industry.

2. INTRODUCTION

Quarries are the primary source for “extractive materials” or “aggregates” used for building roads, ports, airports, bridges, railways, factories, hospitals and homes. A variety of rock types are extracted in Queensland. Crushed aggregates are generally derived from deposits of intrusive and extruded igneous and volcanic rocks, including granite, trachyte and trachyandsite, rhyolite, basalt and a variety of metamorphic rocks including greywacke, greenstone, hornfels and quartzite. Crushing operations in quarries are potentially dusty operations and the crusher operator has potential to be exposed to respirable dust including respirable crystalline silica (DME 2006).

Silicosis is considered to be a slowly developing and progressive disease, not always diagnosed during a working life. There are a number of studies that demonstrate the relationship between loss of lung function and cumulative exposure to respirable dust and respirable crystalline silica (Ulvestad et al 2001, Meijer et al 2001). Some studies demonstrate a loss of lung function below the current Australian Safety and Compensation Council (ASCC) exposure standard – time weighted average of 0.1 mg m^{-3} (Kim et al 2002). Exposure to respirable crystalline silica, a common contaminant of mining and quarry operations, results in the lung damaging diseases known as silicosis and chronic obstructive pulmonary disease (NIOSH 2002). Long-term exposure to respirable crystalline silica can lead to an increased risk of lung cancer. The International Agency for Research on Cancer (IARC 1997) published a monograph which reported that respirable crystalline silica was a cause of lung cancer in humans (Group 1). According to the National Occupational Health and Safety Commission Exposure Standards for Atmospheric Contaminants in the Occupational Environment (NOHSC 3008, NOHSC 1003: 1995), exposure standards, “according to current knowledge, should neither impair the health of nor cause undue discomfort to nearly all workers” (NOHSC, 1995 p4). The current Australian Safety Compensation Commission exposure standard for the most common form of crystalline silica is 0.1 mg m^{-3} (measured as respirable crystalline silica). The Health and Safety Executive 2002 acknowledged that exposure to crystalline silica at concentrations below 0.1 mg m^{-3} over a long period could lead to silicosis.

From the above, it is important that industry understand that Occupational Exposure Limits (OEL) are not fine dividing lines between safe and unsafe exposures.

The current sampling methodology has limitations, in quantifying similar group exposure concentrations with statistical confidence below 0.1 mg m^{-3} . In addition, the literature is suggesting that a sub-fraction of respirable crystalline silica is more likely a pre-cursor to silicosis.

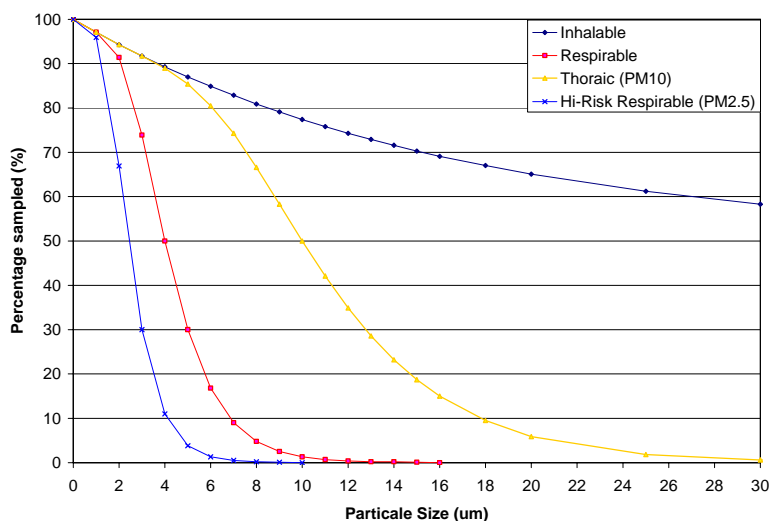
Further research is required towards improve sampling methodologies and understanding the crystalline silica properties, such as particle size and shape that may exert detrimental effects on the lung.

3. DISCUSSION

3.1 Sampling and particle size.

Currently the method used to sample airborne dust containing respirable crystalline silica is AS 2986 (2004) which follows ISO 7708:1995, Air quality – Particle size fraction definitions for health related sampling. According to AS 2986 (2004), respirable dust, is the proportion of airborne particulate matter which, when inhaled, penetrates to the un-ciliated airways.

Figure 1 Particle size distributions and collection efficiency curves according to ISO 7708
(Adapted from ISO 7708:1995, p7)

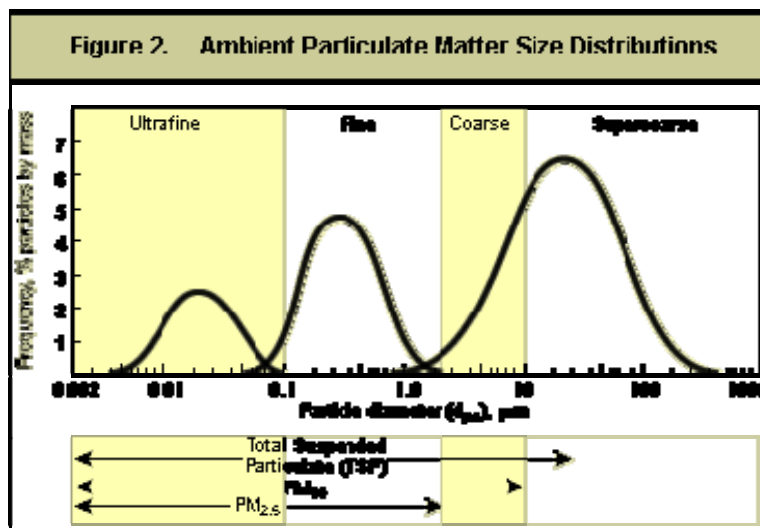


PM_{2.5} is the size fraction that has a high probability of deposition in the smaller airways and alveoli.

Figure 2 Different particle size ranges and naming conventions.

(Source: US Environment Protection Agency

<http://www.epa.gov/eogapt1/module3/category/category.htm#pm2.5>)



According to Vincent (2001) “for assessment of the true health-related dose received by a worker leading (possibly) to ill-health, occupational aerosol measurement ideally requires full characterization of the aerosol as a function of particle size distribution and chemical species”. (p.1)

Vincent (2001) also states that “some occupational health experts have expressed concerns about fine particles and ultra-fine particles as they might relate to workplace aerosols”. (p.1)

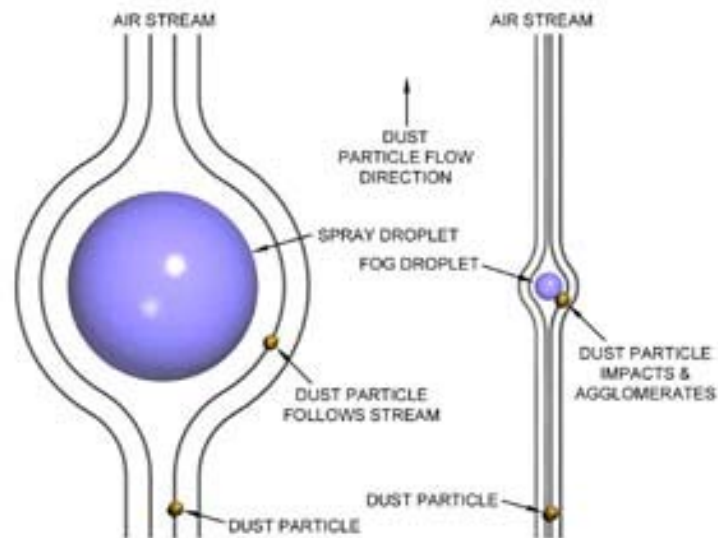
This is an important point as it is generally assumed that silicosis is caused by the fraction of silica that reaches the alveoli (OEHHA, 2005; King et al., 1953). Weissner et al. (1989) on the other hand, observed that relatively larger particles $\geq 5\mu\text{m}$ were more fibrogenic than $1\mu\text{m}$ particles. The more recent literature suggests that total surface area is more important than mass. Surface area is related to particle size; smaller particles possess a larger surface area per unit mass compared to larger particles (HSE, 2002). Fine particles also have much longer residence times than coarse particles when airborne. Exposure to excessive numbers of fine particles may also overwhelm the alveolar macrophages ability to engulf and remove foreign particle.

The literature also suggests that the toxicity of silica may depend on a combination of particle size, particle morphology and surface chemical reactivity.

Removing respirable particulate matter from the air using fogging systems is one option that dusty operations can use to reduce fine airborne dust.

Figure 3 Mechanism by which water droplets in fog removes respirable dust.

(Source: ADSTM Fog based dust control technology. Online at http://www.raringcorp.com/ADS_Tech.htm)

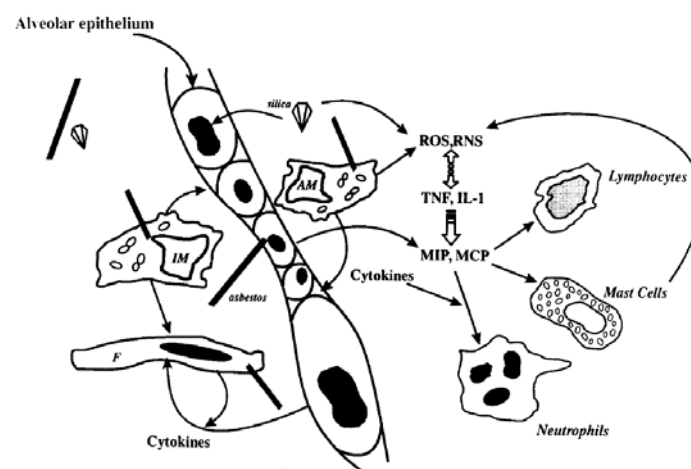


3.2 Respirable crystalline silica and surface chemical reactivity.

There is general agreement that freshly fractured crystalline silica particles are more toxic than aged surfaces as demonstrated by in vitro tests conducted in animal studies (HSE, 2002). The crushing crystalline silica can result in breakage of the Si-Si and Si-OH bonds at the surface. This results in the formation of reactive radicals at the particle surface. These species are highly reactive and through cellular activation resulting in superoxide, hydrogen peroxide and nitric oxide. The formation of reactive oxidative species (ROS) are damaging to cells DNA. Superoxide can also react rapidly with nitric acid to form peroxyxynitrite, an agent that oxidizes and nitrates macromolecules. Studies also indicate that reactive nitrogen species (RNS) are released from affected alveolar macrophages (Brooke et al., 1998). The mechanism in the pathogenesis of silicosis is complex. One model of this pathway is shown in Figure 4..

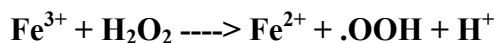
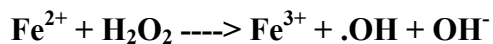
Figure 4 A conceptual model of events occurring in the lung following exposure to pathogenic mineral dusts.

(Source: Brooke et al., 1998, p.1674).



The HSE notes that the activity of free radicals decays with time (ageing) and occurs slowly in air, but rapidly in water. Wet-processes that quench freshly cut quartz will help reduce this reactivity. Metal contaminants may either exasperate the potential for silicosis or provide a protective mechanism.

Driscoll (1995) notes that iron contamination can potentially increase the toxicity of silica by catalyzing the production of reactive oxygen species. This reaction is termed Fenton's reaction which can be described by the following reactions:



Aluminium on the other hand has been suggested as providing a protective layer. Studies in rats and sheep have demonstrated that pulmonary inflammation is reduced by aluminium. The presence of aluminium in coal mines for instance has been used as justification that crystalline silica is less toxic in this environment. The research has challenged this notion by saying that the protective effect is transient and over time the protective effect is lost as aluminium is removed from the silica surface (HSE, 2002).

3.3 Respirable crystalline silica morphology and action of macrophages.

It has been suggested by Champion (2006) that the shape of a particle in the lung, plays a dominant role the macrophages capacity to engulf and remove the particle.

Figure 5 Time-elased video showing macrophages interacting with synthetic particles

(Source: Champion et al.,2005 p. 4931)

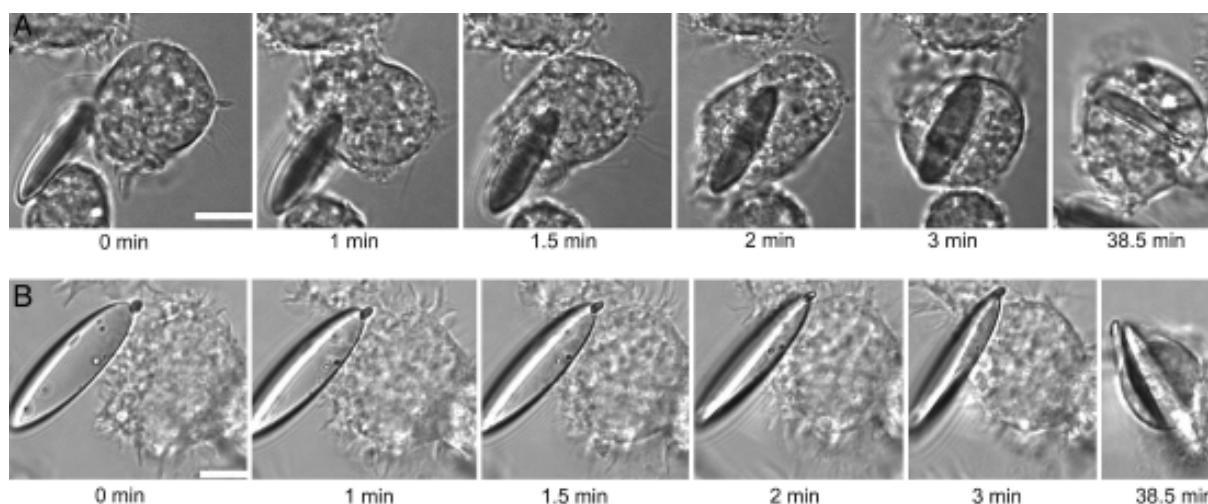
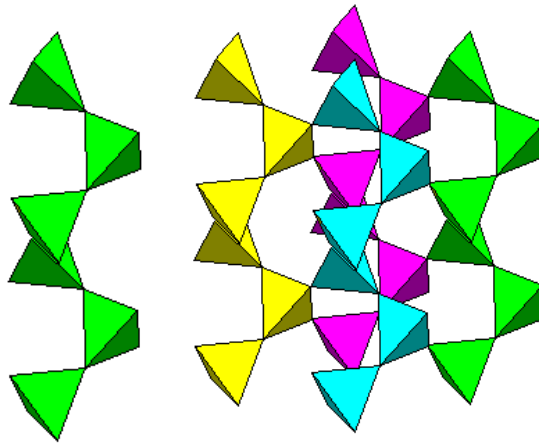


Figure 5 demonstrates that the particle shape and angle at which the macrophage approaches the particle is one determining step in phagocytosis. The crystal system of alpha quartz is trigonal.

The basic structure of quartz consists of spiral chains (helices) of tetrahedra around a three-fold and six-fold screw axes. Figure 6 shows the align of the screw axes with the three-fold helices illustrated on the left and on the right diagram shows the helices connected into a framework.

Figure 6 Models of the basic structure of quartz

(Source: Dutch, S, 2002)



Further research is required to assess crystalline silica morphology and the effect that the shape has on macrophages and phagocytosis.

3.4 Respirable crystalline silica and chronic obstructive pulmonary disease (COPD).

Chronic obstructive pulmonary (lung) disease is a general term used for several lung diseases. The most common types are chronic bronchitis and emphysema. Most patients with COPD have a combination of both of these diseases. COPD worsens gradually causing limited airflow in and out of the lungs. Smoking causes COPD and it is now known that dust exposure also causes COPD.

Figure 7 Mechanism of COPD

(Source: Health Information Home; Understanding COPD. <http://www.cchs.net/health/health-info/docs/2400/2416.asp?index=8709>)

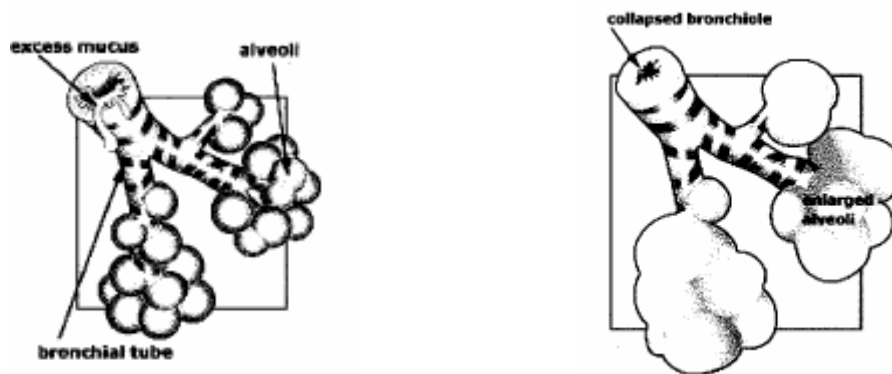


Figure 7 shows how the alveoli and bronchial tubes are affected in chronic bronchitis and emphysema. Emphysema is the destruction, or breakdown, of the walls of the alveoli. Chronic bronchitis is irritation and inflammation of the lining in the bronchial tubes.

Driscoll (2005) cites a study carried out by Korn et al. (1987) who found that in developed countries the relative risk for COPD is 1.0, 1.4 and 1.8 (controlled for smoking) which correlates with non-dust exposed, low exposure and high exposure respectively. Exposure concentrations aren't provided, however mining in developed countries is in the high dust exposed group.

The mechanism of dust exposure and COPD is uncertain. Examination of lungs of workers with occupational dust exposure shows airway fibrosis with thickening of the airway wall and narrowing and distortion of airways. It has been suggested that the membranous bronchioles and respiratory

bronchioles are points of high dust deposition. The respiratory bronchioles are the bridge between bronchioles and alveolar spaces and serve both air-conducting and gas exchange functions.

Table 1 Dimension of human airway model.
(Source: Weibel 1963 cited in Lindstrom 2004 p.10)

Anatomical structure		Generation	Number per generation	Mean diameter (cm)	Mean length (cm)	Cross sectional area (cm ²)
Trachea Main bronchi Lobar bronchi Segmental bronchi Sub-segmental bronchi	LARGE AIRWAYS	0	1	1.80	12	2.54
		1	2	1.22	4.8	2.33
		2	4	0.83	1.9	2.13
		3	8	0.56	0.76	2.00
		4	16	0.45	1.27	2.48
		5	32	0.35	1.07	3.11
		6	64	0.28	0.90	3.94
		7	128	0.23	0.76	5.31
Terminal bronchi Bronchioles	SMALL AIRWAYS	8	256	0.186	0.64	6.95
		9	512	0.154	0.54	9.53
		10	1024	0.130	0.46	13.6
		11	2048	0.109	0.39	19.1
		12	4096	0.095	0.33	29.0
		13	8192	0.082	0.27	43.2
		14	16384	0.074	0.23	70.4
Terminal bronchioles Respiratory bronchioles Alveolar ducts Alveolar sacs	ACINUS	15	32768	0.066	0.20	112
		16	65536	0.060	0.165	185
		17	131907	0.054	0.141	300
		18	262144	0.050	0.117	534
		19	524288	0.047	0.099	944
		20	1489576	0.045	0.083	1600
21 - 23	15000000	0.042	0.060	~ 140m ²		

According to Girod 2007, the chronic characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema). Table 1 provides dimensions of the small airways. Particles larger than 10 µm are deposited in the upper airways. Particles 3 – 10 µm are deposited in the trachea and larger airways due to impaction. Smaller particles 0.5 – 3 µm are deposited in the terminal airways and in the alveoli. Ultra fine particles less than 100 nm are deposited in the alveolar region and the larger fraction is inhaled (Lindstrom, 2004). Based on this the current methods of sampling for respirable dust may not collect the dust fraction that may be responsible for COPD. Respirable dust sampling collects both the particle size fraction responsible for COPD and particles >3 µm which may not cause COPD. PM_{2.5} may provide a better indication of the risk when attempting to identify a dose response relationship.

4. CONCLUSION.

The mechanism by which respirable crystalline silica causes silicosis and COPD has been an area of considerable research. For crystalline silica particles to exhibit adverse health effects, particle size, shape, age of fractured surface, chemical activity and surface metals must be taken into account. Further research is required especially to identify the particle size distribution and shape that poses the greatest risk.

A number of sources claim that wetting respirable crystalline silica reduces the reactivity of the freshly cut crystalline silica but further research is required to confirm this theory. Wetting respirable crystalline silica will suppress airborne dust and fogging systems may possibly reduce the airborne dust fraction that is respirable. Trials are also required to confirm if these systems meet their objective.

The current method to sample respirable crystalline silica, using as 2985-2004, requires further evaluation. It may be that sampling for pm_{2.5} and analysis of this fraction may be a better estimate of the exposure risk.

5. REFERENCES.

- ADSTM Fog based dust control technology. Retrieved 30 October 2007 from http://www.raringcorp.com/ADS_Tech.htm
- Australian Safety and Compensation Council (ASCC) n.d, *Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment*, Retrieved 30 October 2007 from http://www.ascc.gov.au/NR/rdonlyres/317D25BA-E837-4F5B-AC65-24FE588888CA/0/ExposureStandards4AtmosphericContaminants_Nov06version.pdf
- Australian Safety and Compensation Council (ASCC) n.d., *Hazardous Substances Information System*, Retrieved 20 April, 2007 from <http://hsis.ascc.gov.au/Default.aspx>
- Brooke, T, Mossman, Churg, A, 1998, Mechanism in the Patogenesis of Asbestosis and Silicosis. *American Journal Respiratory Critical Care Medicine* Vol 157. pp 1666-1680, 1998.
- Buchanan, D, Miller, BG & Souter, CA 2003, *Quantitative relations between exposure to respirable quartz and risk of silicosis*, Institute of Occupational Medicine, Edinburgh, Scotland, UK.
- Champion, J, 2006, *Role of Target Geometry in Phagocytosis*, Department of Chemical Engineering, University of California, Santa Barbera, CA 93106.
- Churg A, Wright J, 2002, Airway Wall Remodelling Induced by Occupational Mineral Dusts and Air Pollutant Particles. *Chest* 2002;122;306-309
- Department of Mines and Energy (DME) 2006, Queensland Mining and Petroleum Industries, Explorations Operations and Developments. Retrieved 2 November 2007. http://www.nrw.qld.gov.au/mines/publications/min_pet_review/pdf/mp_ind_06.pdf
- Driscoll, K E (1995), The toxicology of crystalline silica studies in vitro. *Applied Occupational Environmental Hygiene*, 10, 1118 – 1125.
- Driscoll, T, Nelson, D, Steenland K, Leigh J, Marisol C, Fingerhut, M, Pruss-Ustun A, The global burden of non-malignant respiratory diseases due to occupational airborne exposures. *Journal of Industrial Medicine*, 2005.
- Driscoll, T 2006, *Review of Australian and New Zealand workplace exposure surveillance systems*, Office of the Australian Safety and Compensation Council and the National Occupational Safety and Health Advisory Committee.
- Dutch, S., 2002, *Quartz Structure*, Retrieved 30 October 2007, from <http://www.uwgb.edu/dutchs/PETROLOGY/QuartzStruc.HTM>
- Fenton's reaction. Retrieved 29 October 2007. <http://www.lenntech.com/Fenton-reaction.htm>
- Girod, C, Talmage, E, 2005, 'COPD: A Dust-Induced Disease', *Chest*, vol. 128, pp. 3055 - 3064. Health Information Home; Understanding COPD. Retrieved 30 October, 2007 from <http://www.cchs.net/health/health-info/docs/2400/2416.asp?index=8709>

- Health and Safety Executive (UK) 2002, *Respirable crystalline silica - phase 1: variability in fibrogenic potency and exposure-response relationships for silicosis. Hazard assessment document: Guidance note, environmental hygiene/EH75/4*, Health and Safety Executive, UK
- Health and Safety Executive (UK) 2005, *Occupational Respiratory Diseases (other than Asthma) work-related chronic obstructive pulmonary disease (COPD) evidence-base intervention and evaluation plans (DRAFT)*. Retrieved 20 April, 2007 from <http://www.hse.gov.uk/aboutus/hsc/iacs/acts/watch/051005/15annexe3.pdf>.
- HSE 2006, *Control of Substances Hazardous to Health – COSHH*, Retrieved 20 April, 2007 from <http://www.hse.gov.uk/coshh/>
- IARC 1987, *Monographs on the evaluation of carcinogenic risks to humans Vol 42, Silica and some silicates*. IARC, Lyon, France.
- IARC 1997, *Monographs on the evaluation of carcinogenic risks to humans Vol 68, Silica, some silicates, coal dust and para-aramid fibrils*.
- ISO 7708, *Air Quality – Particle size fraction definitions for health related sampling*. International Organization for Standardization
- Kim, TS, Kim, HA, Heo, Y, Park, Y, Park, CY & Roh YM 2002, 'Level of silica in the respirable dust inhaled by dental technicians with demonstration of respirable symptoms', *Industry Health*, vol.40, no. 3, pp.260 – 265.
- King, E. J., G. P. Mohanty, C. V. Harrison, and G. Nagelschmidt. 1953. The action of flint of variable size injected at constant weight and constant surface into the lungs of rats. *Br. J. Ind. Med.* 10:76–92.
- Lindstrom, M, 2004, *Particles in small airways: mechanisms for deposition and clearance and pharmacokinetic assessment of delivered dose to the lung*. From the Department of Public Health Science, division of Occupational Medicine, at Karolinska Institute of Stockholm and Childrens Hospital at Karolinska University Hospital, Huddinge, Sweden.
- Meijer, E, Kromhout, H & Heederik, D 2001, 'Respiratory effects of exposure to low levels of concrete dust containing crystalline silica', *American Journal of Medicine*, vol, 40, no. 2, pp.133-140.
- National Institute for Occupational Safety and Health (NIOSH) 2002, *NIOSH Hazard Review, Health Effects of Occupational Exposure to Respirable Crystalline Silica*, Retrieved 20 April, 2007 from <http://www.cdc.gov/niosh/02-129A.html>
- NOHSC 1995, *Guidance Note on the Interpretation of Exposure Standards for Atmospheric Contaminants in the Occupational Environment NOHSC 3008(1995)* 3rd Ed, Australian Government Publishing Service Canberra, Retrieved 30 April, 2007 from <http://www.ascc.gov.au/ascc/aboutus/publications/nationalstandards/guidancenoteontheinterpretationofexposurestandardsforatmosphericcontaminantsintheoccupationalenviron.htm>
- Office of the Environmental Health Hazard Assessment (OEHHA), 2005. Chronic Toxicity Summary – Silica (Crystalline, Respirable). http://www.oehha.ca.gov/air/chronic_rels/silica_final.html (accessed 29 October 2007).
- Standards Australia 2004, *Workplace atmospheres – Method for sampling and gravimetric determination of respirable dust*. AS2985:2004 Retrieved January 10, 2006 from Standards Australia Online database.
- Ulvestad, B, Bakke B, Eduard W, Kongerud J, Lund MB & Selmer, ASA 2001; 'Cumulative exposure to dust causes accelerated decline in lung function in tunnel workers', *Occup Environ Med*, vol.58, no. 10, pp.663-669.
- US Environmental Protection Agency, *Basic Concepts in Environmental Science*. Retrieved 30 October 2007. <http://www.epa.gov/eogapti1/module3/category/category.htm#pm2.5>

Vincent, J, 2001, *Industrial Characterisation of Coarse, Fine, Very Fine and Ultrafine Workplace Aerosols* Proceedings of 20th AIOH Annual Conference.

Wiessner, J. H., N. S. Mandel, P. G. Sohnle, and G. S. Mandel. 1989. Effect of particle size on quartz-induced hemolysis and on lung inflammation and fibrosis. *Exp. Lung Res.* 15:801–812.

UPDATE OF HEALTH WATCH STUDY FINDINGS 2007

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Abstract

Health Watch, a cohort study of Australian petroleum industry workers, was set up in 1980 to compare types of cancer and causes of death in these workers with that of the general population. In 2007, the cohort was matched to the national death and cancer registries and the findings compared to age standardised national rates. This paper presents findings from the update. Only a quarter of the cohort remains in employment in the industry. The healthy worker effect remains strong however with an all cause Standardised Mortality Ratio (SMR) of 0.72 for men and 0.65 for women.

The overall cancer incidence in male members of the Health Watch cohort is similar to that of Australian men of the same age distribution. Male lung cancer incidence is lower than expected but the incidence of mesotheliomas and melanomas remain elevated.

3-yearly serial analyses of melanoma, leukaemia, NHL and multiple melanoma SIRs show that all these have fallen since 1987

As the cohort has aged, the effects of smoking have become more pronounced and the dose-response relationship has become stronger. Quitting reduces these risks, especially that of heart disease.

Introduction

Health Watch is a prospective cohort study of about 18,000 employees, who have worked for more than 5 years in the Australian petroleum industry. It commenced in 1981 with a face-to-face survey and this survey was repeated in 1986, 1991 and 1996. At the surveys, subjects provided demographic details, health status information and details of their work history. All Australian petroleum company employees were invited to participate except those employed at head offices and at sites with fewer than 10 employees. About 95% of eligible employees have participated. (Gun et al. 2000) The cohort thus consists of employees from offices, upstream extraction and processing sites, refineries, terminals and airports all over Australia. Subjects have been followed by matching with the Australian national death and cancer registries on a regular basis. The latest match was carried out in 2007.

Methods

This update of the *Health Watch* cohort is based on searches of the National Death Index (NDI) to 30th November 2004 and the cancer registrations of the National Cancer Statistics Clearing House (NCSCCH) and the Victorian Cancer Registry (VCR) to 31st December 2002. The death rates and cancer incidence in *Health Watch* are then compared to national mortality data and cancer incidence data provided by the Australian Institute of Health and Welfare (AIHW). (Australian Institute of Health and Welfare (AIHW) 2007) 16623 men and 1375 women are included in this analysis.

In the early 1980s, all jobs in the Health Watch cohort were ranked by industry hygienists according to increasing potential for exposure to total hydrocarbons. In 1994-6 the rankings were reviewed and amended in the light of additional job history information. They have not been revised since. These rankings are a crude measure of exposure and were used to carry out risk estimates within the cohort, grouping workers by their highest ranked job or the rank of their longest held job.

Results and Discussion

1473 men and 34 women in the cohort had died by the end of 2004. *Health Watch* has now accumulated 289,275 person-years of observation in men and 19,347 person-years in women. (Glass et al. 2007) The age-adjusted death rate in men and women is significantly less than in the general Australian population. The strong healthy worker effect identified in previous studies continues to be observed. The chance of contracting cancer is similar for men and women in this industry to that of all Australians. However, the mortality from cancer is reduced for *Health Watch* members, significantly so for men (Table 1).

Table 1 Overall mortality and cancer incidence rates for Health Watch Cohort adjusted for age and calendar period of follow-up, compared to the Australian population

Sex	Overall SMR (95% C.I.)	Cancer SIR (95% C.I.)	Cancer SMR (95% C.I.)
Male	0.72 (0.68-0.76)	0.99 (0.94-1.04)	0.81 (0.75-0.88)
Female	0.65(0.45-0.91)	0.89 (0.68-1.15)	0.88 (0.54-1.34)

Of the 34 female cohort members who have died, 21 deaths were from cancer. The overall cancer SMR and SIR in women is similar to that of the Australian female population. The small number of women in the *Health Watch* program precludes further detailed analyses.

For men, death rates in all major disease categories were also significantly lower than for the corresponding Australian population (Table 2). A significant reduction in all cause mortality is seen among men in each workplace type e.g. refinery, terminal.

There is no trend of increasing mortality with increasing duration of employment. There is evidence of a trend of increasing overall mortality by time since first employment and period of first employment. This may be because the most recently employed men have particularly low mortality and the healthy worker effect is stronger in those most recently employed. (Applebaum et al. 2007)

Table 2: Mortality by major cause for men, adjusted for age and calendar period of follow-up, compared to the Australian population

Cause	SMR (95% C.I.)
Cancer (Malignant)	0.81 (0.75-0.88)
Ischaemic heart disease	0.77 (0.69-0.85)
Stroke	0.60 (0.46-0.77)
Respiratory disease	0.73 (0.59-0.89)
All diseases of the digestive system	0.57 (0.42-0.77)
External Causes (e.g. accidents, violence, suicide)	0.64 (0.53-0.77)
All other causes	0.55 (0.47-0.64)
All causes	0.72 (0.68-0.76)

There is no evidence of increasing cancer incidence or increasing cancer mortality with any of the following:

- increasing duration of employment;
- increasing time since first employment;
- time period of first employment.

Two cancers – mesothelioma and melanoma - have been and still are occurring at significantly higher rates than in the general population. These are the only cancers in significant excess. Cancer of the prostate SIR 1.09 (0.98-1.22) and bladder cancer SIR 1.11 (0.85-1.43) were in excess in the previous report (Gun et al. 2005) but are no longer in excess. Mortality from colon SMR 0.66 (0.48-0.89) and lung cancer SMR 0.65 (0.54-0.77) are significantly lower than that of the general population.

Twenty-three mesotheliomas have occurred in the cohort, 16 in refinery maintenance workers and operators, SIR 1.76 (1.12-2.65). It is possible that these cancers are related to asbestos exposure in refineries, mostly before the 1970s, although some are likely to have resulted from asbestos exposure occurring prior to entering the oil industry.

Asbestos exposure is a known cause of lung cancer. Some overseas studies have reported a higher rate of lung cancer in refinery maintenance workers compared with other refinery workers. (Schnatter et al. 1992; Tsai et al. 1996; Divine et al. 1999; Sorahan 2007) When the risk for lung cancer among maintenance workers was compared to that among non-maintenance workers (adjusted for age group, calendar year and ever vs never smoking), the incidence rate in the two groups was similar (RIR 1.10, 95% C.I. 0.48-2.54). The increased rate in maintenance workers is not statistically significant. This suggests that very few asbestos-related lung cancers have occurred from working in the Australian petroleum industry, particularly so since the overall lung cancer rate in the *Health Watch* cohort is so low. Smoking rates in the cohort are lower than that in the general population.(Gun et al. 2000)

There was a statistically significant increase in the incidence of melanoma in men, SIR 1.29 (1.13-1.48). The rate does not increase with increasing duration of employment, time since first employment or period of first employment. The overall melanoma SIR in the cohort has fallen over time (Table 3) and varies by state probably as a result of differences in sun exposure. When compared to state melanoma rates there is a significant excess in New South Wales.

Table 3: Melanoma incidence over time for men in the *Health Watch* cohort

Melanoma	1987	1990	1993	1996	1999	2002
Cases	23	47	80	123	173	222
SIR	1.64	1.20	1.52	1.48	1.46	1.29
(95% C.I.)	(1.0-2.5)	(0.9-1.6)	(1.2-1.9)	(1.2-1.8)	(1.2-1.7)	(1.1-1.5)

Table 4: Melanoma incidence compared to state specific rates for men by state of last employment

State	SIR (95% C.I.)
NSW	1.45 (1.14-1.82)
QLD	1.37 (0.98-1.86)
WA	1.27 (0.86-1.80)

Although an increased incidence of bladder cancer was reported in the 12th *Health Watch* report (Gun et al. 2005), this updated analysis shows only a small elevation in incidence which is not now statistically significant. However our analysis confirmed the known association between bladder cancer and smoking. Smoking (ever smoked vs never smoked) significantly increased the risk of bladder cancer (RR 3.98, 95% C.I. 1.81-8.75).

While analyses in the early years of the cohort found an excess leukaemia in the cohort, (Christie et al. 1990; Bisby and Adams 1993; Bisby and Adams 1999) there is now no significant excess of leukaemia SIR 0.92 (0.65-1.27). Acute non-lymphocytic leukaemia (ANLL), which is the leukaemia most strongly associated with benzene exposure, is not in significant excess in the cohort SIR 0.85 (0.43-1.53). There were no new ANLL cases since the last report. The incidence of leukaemia, non-Hodgkins lymphoma (NHL) and multiple myeloma (MM) have reduced over time (Figure 1).

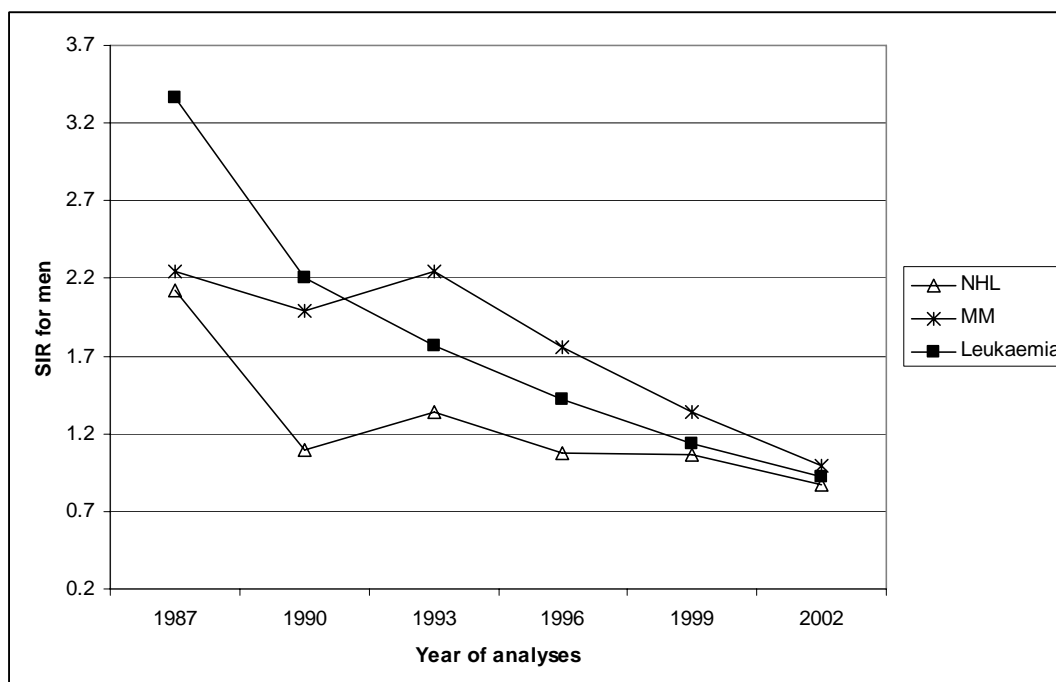


Figure 1: SIRs for leukaemia, NHL and MM in men plotted at 3-year intervals

Health Watch carries out analyses of members in some particular occupational groups, and a small and non-significant overall cancer excess was found in tanker drivers. However, there were no specific cancer types with significantly increased rates among drivers. Cancer of the kidney was raised among drivers in the previous report but is now no longer a statistically significant excess SIR 1.79 (0.95-3.07). The number of kidney cancer cases ($n=13$) in drivers in *Health Watch* is too low to do any meaningful analysis of any work-related cause in this group. However, analyses of the kidney cancer cases in the whole cohort were suggestive of an association between hydrocarbon exposure and cancer of the kidney. The possibility of an association between cancer of the kidney and hydrocarbon exposure warrants further study with more refined exposure assessment perhaps as a nested case-control study.

Smoking related diseases; lung cancer incidence and mortality (SIR 0.74(0.62-0.87); SMR 0.65 (0.54-0.77), incidence of cancer of the lip, oral cavity and pharynx SIR 0.77 (0.60-0.98), ischaemic heart disease mortality SMR 0.77 (0.69-0.85) and chronic obstructive pulmonary disease mortality, SMR 0.61 (0.45-0.80), are lower in *Health Watch* members, than in the general population. However, within the cohort, there is a clear pattern that increasing smoking category is associated with increasing risk of all-cause mortality, heart disease mortality and overall cancer mortality (Figure 2) and of lung and bladder cancer incidence. Furthermore it is clear that rates of mortality and cancer incidence are greatly reduced for ex-smokers compared with smokers. Altogether smoking is estimated to have contributed to about 40% of all male deaths in the cohort. The risk of lung cancer for those who smoke 30+ a day is 50 times that of non-smokers (up from 30 times 3 years ago), while the risk of dying of lung cancer in this group is close to 120 times greater than for non smokers. The risk of death is 4 fold for this group (up from 3 fold 3 years ago), the risk of death from ischaemic heart disease is more than 5 fold that of non smokers.

Heavy drinking, (more than 35 alcoholic drinks per week) is associated with increased overall mortality. Moderate drinking is associated with reduced mortality.

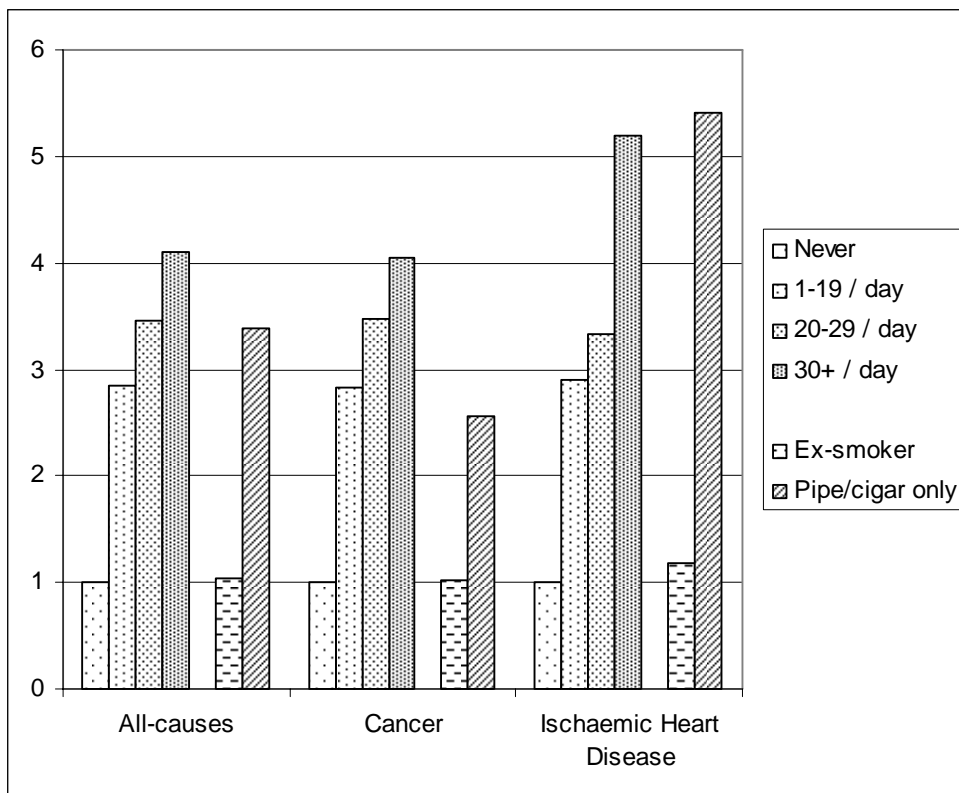


Figure 2: Relative risk of dying from any cause, of cancer or of ischaemic heart disease among men for different smoking categories, compared to those who never smoked.
(The RMRs are adjusted for age and calendar year.)

Conclusions

The age-adjusted death rate in men and women remains significantly lower than in the general Australian population. The strong *healthy worker effect* identified in previous studies continues to be observed.

The chance of contracting cancer is similar for men and women in this industry as for all Australians. However, the mortality from cancer is reduced for *Health Watch* members, significantly so for men.

There is no evidence of increasing mortality, cancer incidence or increasing cancer mortality with any of the following:

- increasing duration of employment;
- increasing time since first employment;
- time period of first employment.

The SIRs for melanoma, leukaemia, NHL and MM in the cohort, have been falling since 1987. Smoking related diseases are lower in *Health Watch* members, than in the general population. However, within the cohort, there is a clear pattern that increasing smoking category is associated with increasing risk of all-cause mortality and cancer. Smoking-related diseases are becoming more evident as the cohort ages. Quitting cigarette smoking greatly reduces the risks.

Acknowledgements

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We are indebted to the contact persons in each of the participating companies. *Health Watch* is dependent on them for follow-up information. Finally we wish to thank the many employees who participated and assisted the team.

References

- Applebaum, K. M., E. J. Malloy and E. A. Eisen (2007). "Reducing healthy worker survivor bias by restricting date of hire in a cohort study of Vermont granite workers " Occupational & Environmental Medicine **64**: 681-687.
- Australian Institute of Health and Welfare (AIHW). (2007). "Cancer Incidence Data." Retrieved May 2007, 2007, from <http://www.aihw.gov.au/cancer/datacubes/index.cfm>.
- Divine, B. J., C. M. Hartman and J. K. Wendt (1999). "Update of the Texaco mortality study 1947-93: part II. Analyses of specific causes of death for white men employed in refining, research, and petrochemicals." Occupational and Environmental Medicine **56**: 174-180.
- Glass, D., A. Del Monaco, K. Giuliano, et al. (2007). Health Watch Thirteenth Report 2007. Melbourne, Australia, Monash Centre for Occupational and Environmental Health (MonCOEH), Department of Epidemiology and Preventive Medicine, Monash University 87.
- Gun, R. T., L. Pilotto, P. Ryan, et al. (2000). Health Watch Eleventh Report 2000. Adelaide, Australia, Department of Public Health, Adelaide University.
- Gun, R. T., P. Ryan, D. Roder, et al. (2005). Health Watch Twelfth Report 2005. Adelaide, Australia, Department of Public Health, Adelaide University.
- Schnatter, R. A., G. Theriault, A. M. Katz, et al. (1992). "A retrospective mortality study within operating segments of a petroleum company." American Journal of Industrial Medicine **22**: 209-222.
- Sorahan, T. (2007). "Mortality of UK oil refinery and petroleum distribution workers, 1951-2003 " Occupational Medicine **57**(3): 177-185.
- Tsai, S. P., L. C. Waddell, E. L. Gilstrap, et al. (1996). "Mortality among maintenance employees potentially exposed to asbestos in a refinery and petrochemical plant." American Journal of Industrial Medicine **29**(1): 89-98.

DEVELOPMENT OF A SAMPLING PROTOCOL FOR ASSESSMENT OF FIREFIGHTER EXPOSURE TO AIR TOXICS ON THE FIREGROUND

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ABSTRACT

It is well known that the bushfire fireground contains a range of hazards that makes firefighting an extremely risky activity. In particular, bushfires generate a range of air toxics that are potentially harmful to human health. These toxics include Carbon Monoxide, Aldehydes, Volatile Organic Carbons [VOC's], and Particulates of various sizes, all of which may present a significant hazard to firefighter health and safety. While there have been studies on the different toxics contained in bushfire smoke, and of the exposure of firefighters in Northern America, there have been limited studies conducted on the exposure of firefighters in Australia, and of the air toxics produced by Australian vegetation. As Australian vegetation may emit air toxics of different composition and concentrations to those found in Northern America studies, and as firefighting strategies are different, the studies conducted in the US are not applicable to Australia.

As there are no industry specific exposure standards, based on extended shifts, level of activity or intensity of fire, established for active firefighter exposure, there is little established "Best Practice" sampling protocols available. If occupational exposure standards are to be applied for firefighter exposure a rigorous sampling protocol needs to be established. This investigation looks a possible method for monitoring firefighters including the challenges posed by sampling in such a dynamic and hazardous environment.

The development process involved work monitoring firefighter exposure during two wildfires in 2006/2007 and at prescribed burning activities conducted in Eastern Australia. Atmospheric sampling focused on two main areas:

1. personal monitoring, with samples collected in the breathing zone of active firefighters, and
2. local environment samples, collected with vehicle mounted instrumentation.

WHERE THERE'S FIRE, THERE'S SMOKE: AIR CONTAMINANTS AT RESIDENTIAL FIRE INVESTIGATION SCENES

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ABSTRACT

Fire investigations are typically conducted in environments that present a number of physical hazards such as confined spaces, working at heights, electrical and structural stability issues. However, risk management strategies also need to encompass the inhalation hazards associated with post-fire atmospheres. Scientific literature relating to air contaminants in post-fire residential structure environments is extremely limited. Making informed decisions relating to appropriate respiratory protective equipment for personnel working in these areas has therefore been very difficult.

In order to address this gap in knowledge, a joint study was conducted between Queensland Fire and Rescue Service Scientific Unit, Queensland Fire and Rescue Service Fire Investigation and Research Section, Queensland Health Forensic and Scientific Services and the School of Physical and Chemical Sciences, Queensland University of Technology. Air samples were collected during three fire investigations conducted at residential premises in the Brisbane area. These samples were analysed for the presence of volatile organic compounds and carbonyl compounds, with both groups of chemicals being associated with adverse health effects when occupational exposure occurs at excessive levels.

A number of compounds detected in the post-fire environments in this study were chemicals for which adverse short-term and/or long-term health effects have been documented. However, at the fire investigation scenes attended, all of the compounds detected were below the Australian Exposure Standards. The results of this study have been used to develop an atmospheric monitoring procedure for fire investigations at residential premises.

1. INTRODUCTION

Even in the absence of hazardous materials, the concentrations of toxic organic compounds released in residential fires can be high (Ruokojärvi et al., 2000). In addition, the complexity of the combustion process is such that the materials present and conditions of combustion greatly affect the products of that process (Lees, 1995). This results in a work environment characterised by harsh and uncontrolled conditions that may include high concentrations of chemicals, where the only feasible means of exposure control is in the form of personal protective equipment (Lees, 1995). Persons who may be exposed to post-fire atmospheres in the course of their work include members of fire and police services, specialist advisors such as electrical safety investigators, insurance company representatives, and repair or demolition crews.

The earliest research into the toxicity of combustion products in firefighting environments focussed on the effect of elevated levels of carbon monoxide (Barnard and Weber, 1979), with later research including substances such as sulfur dioxide, hydrogen chloride, hydrogen cyanide, nitrogen oxides, ketones, aldehydes and aromatic compounds (Burgess and Crutchfield, 1995; Lees, 1995). Studies have been mostly confined to conditions during fire suppression, which is defined as covering both the "extinguishment" or "knockdown" phase (in which the fire is brought under control) (Austin et al., 2001c; Jankovic et al., 1991; Lowry et al., 1985) and the "damping down" or "overhaul" phase (in which fire suppression is complete, and firefighters are searching the structure for hidden fire

(Austin et al., 2001a; Bolstad-Johnson et al., 2000; Brandt-Rauf et al., 1988; Gold et al., 1978; Jankovic et al., 1991; Treitman et al., 1980). Very little literature is available on air contaminants present in post-fire environments, with surface deposition sampling techniques commonly used instead of air sampling (Rao and Brown, 1990; Wobst et al., 1999).

Significant difficulties associated with collecting air contaminants from real fire scenes have been previously identified, including the complex nature of combustion products, overloading of sampling methods normally used to quantify low-level industrial exposures, the inability to schedule sampling, and the inability to begin monitoring within a uniform time interval after fire ignition or suppression (Austin et al., 2001b; Bolstad-Johnson et al., 2000; Brown, 2005; Lees, 1995). However, simulated fire scenes have limitations in accurately representing fire load in terms of variety of furniture and interior decorating material involved, while surface wipe samples result in a distorted combustion product profile (Ruokojärvi et al., 2000). Personal breathing zone measurements and area measures in actual working fires are considered the most reliable estimates of firefighter exposure to air contaminants (Lees, 1995). The present study used direct-reading air monitoring and air sampling techniques to investigate the contaminants present during fire investigations conducted in residential premises.

2. METHOD

The locations for this study were selected on the basis of the occurrence of a residential fire in the Greater Brisbane Region that required the attendance of personnel from the Fire Investigation and Research Section of the Queensland Fire and Rescue Service during 2006. Incidents involving fatalities were excluded from the study. In order to avoid interference with the fire investigation process, permission to enter any scene was obtained from the incident controller, and entry to any part of a fire-affected structure was made under supervision of personnel from either Queensland Fire and Rescue Service (QFRS) or Queensland Police Service (QPS). Placement of sampling equipment and timing of sample collection was subject to constraints imposed by either QFRS or QPS personnel.

Fire Scene 1

This fire scene involved a single-storey timber dwelling with tiled roof. The dwelling was well alight on the arrival of first attending fire crews. Fire service communications records indicated that the duration of extinguishment operations was approximately 50 minutes, with damping down continuing for a further 90 minutes. Ventilation of the premises was undertaken by the fire crews during the damping down phase. Fire investigation personnel arrived at the scene nine and a half hours after firefighting operations ceased. Fire damage was primarily confined to one bedroom of the structure, and this was the room in which sampling occurred. Internal walls were wooden, and the floor in the area of sampling had been carpeted. The fuel load provided by furnishings in this location was considered to be low. Approximately two-thirds of the roof was missing in this area, and all windows in this room were broken.

Fire Scene 2

The premises involved in this fire was a single-storey weatherboard residence with aluminium sheet roofing. The dwelling was well involved in fire at the time of fire crews' arrival. Extinguishment and damping down phases of firefighting had durations of approximately 35 minutes and 65 minutes respectively. Fire investigation personnel arrived at the scene approximately 13 hours after the cessation of firefighting operations. Sampling at this scene occurred in a bedroom identified by fire investigators as the likely area of origin. Ventilation of this scene prior to the arrival of fire investigators was considered to have been high, since the roof and all four walls of the building had sustained such heavy fire damage that the structure was essentially open. Building partitioning and ceilings had been of masonite construction, with timber floor and doors. The fuel load in the premises was rated as high by fire investigators based on the description of furnishings provided by the occupants of the premises.

Fire Scene 3

This fire occurred in a raised weatherboard dwelling with iron roof. Fire extinguishment operations were of approximately 35 minutes duration. This was followed by a damping down process, with the final fire crew departing one hour and 45 minutes after extinguishment operations ceased. Fire investigators arrived approximately three hours after the departure of the final firefighting crew. Sampling at this scene occurred in a bedroom that was identified by fire investigators as a possible area of origin. The area was characterised by timber walls and carpeted floor. Only the two doorways leading to other parts of the house provided ventilation to the sampling space, since the walls and ceiling were all intact.

Identification of sampling sites

Placement of sampling equipment within the working space of fire investigators during the investigation was designed to capture any contaminants stirred up during the process of determining the cause of the fire. The longest duration and the most physically demanding work of a fire investigator will usually occur in the area in which the fire was believed to have started. Therefore, if an area was identified early in the investigation to be a potential area of origin of the fire, sampling occurred in this area.

There were a number of limitations on placement and timing of sample collection. Firstly, it was important to ensure that fire scene integrity was not compromised. In the early stages of a fire investigation it may not be clear whether or not the cause of the fire was accidental or deliberate in nature. At some fire scenes it was therefore necessary to delay sample collection until initial observations had been made, and some restrictions were placed by QFRS and QPS personnel on location of sampling equipment, in order to avoid sensitive areas. Secondly, sampling equipment could only be set up in a location where safe access and egress could be achieved. At Fire Scenes 1 and 2, damage to floorboards was sufficient to require substantial caution in moving in and out of the area in which fire investigators were working. Thirdly, sampling occurred at least one metre from any still-existing interior or exterior wall, in order to avoid obstacles to airflow which could result in the re-sampling of the same atmosphere fraction. The usual recommendation for sampling in unobstructed areas is a distance of 2 metres from obstacles (Center for Environmental Research Information, 1999b), but this proved impossible in the post-fire environment.

Meteorological information

Information relating to the temperature, relative humidity, wind speed, atmospheric pressure and precipitation levels was obtained from the Bureau of Meteorology website for the weather station geographically closest to the location of each fire. These values were collated for both the time interval between the extinguishment of the fire and arrival of fire investigators, and the period of fire investigation itself (Table I). These parameters were considered to be important as they may influence the dissipation of combustion products, particularly where the integrity of the roof of the structure has been compromised.

Table I Summary of weather conditions prior to and during atmospheric sampling

Fire Scene Number	Weather Conditions		
		Interval Before Sampling	During Sampling
1	Temp (°C)	20 – 23	23 – 26
	Rel. Humidity (%)	81 – 96	63 – 81
	Wind Speed(m/s)	3 - 7	4 – 7
	Atm. Pressure (kPa)	101.4 – 101.7	101.5 – 101.6
	Precipitation (mm)	1.4	0
2	Temp (°C)	10 – 17	17 – 22
	Rel. Humidity (%)	66 – 94	41 – 58
	Wind Speed (m/s)	0 – 4	3 – 5

	Atm. Pressure (kPa)	100.9 – 101.1	101.0 – 101.1
	Precipitation (mm)	0	0
3	Temp (°C)	18 – 25	25 – 27
	Rel. Humidity (%)	n/a	n/a
	Wind Speed (m/s)	3 – 4	3 – 4
	Atm. Pressure (kPa)	101.6 – 101.7	101.5 – 101.7
	Precipitation (mm)	0	0

Air monitoring and sampling procedures

Real-time air monitoring was undertaken at each site for oxygen content, flammability, carbon monoxide and hydrogen sulfide levels (RAE Systems MultiRAE Plus) and volatile organic compounds (RAE Systems MiniRAE 2000 photoionisation detector).

Details of air sampling are summarised in Table II. Variation in flow rates and sampling times arose due to the uncharacterised nature of fire investigation scenes, and the consequent need to obtain balance between sampling sufficient air volumes and avoiding breakthrough, as well as limitations placed on sampling due to requirements of investigating officers.

Table II Summary of fire scene sample collection

Fire Scene Number	VOC (Tenax/Carboxen 569 tubes)	Carbonyls (DNPH cartridges)	Whole air samples (6 L air canisters)
1	200 mL/min; 10 minutes 200 mL/min; 30 minutes	200 mL/min; 30 minutes	n/a
2	250 mL/min; 32 minutes	1250 mL/min; 50 minutes	1 canister
3	250 mL/min; 30 minutes	Unknown; 18 minutes (pump failure)	1 canister

In order to sample for volatile organic compounds, air from the scene was pumped through stainless steel tubes containing 150 mg of Tenax in series with 100 mg of Carboxen 569. These samples were analysed by GC/MS in accordance with United States Environmental Protection Agency Method TO-17 (Center for Environmental Research Protection, 1999e).

Carbonyl samples were collected using cartridges containing chromatographic grade silica of particle size 150 to 250 µm, coated with 2,4-dinitrophenylhydrazine. Ozone scrubbers were attached in front of the cartridges to avoid negative interference. Samples were analysed using HPLC in accordance with United States Environmental Protection Agency Method TO-11A (Center for Environmental Research Information, 1999a).

Whole air samples were also collected at the scene, using 6 litre evacuated air canisters. These were analysed in accordance with United States Environmental Protection Agency Methods TO-14 and TO-15 (Center for Environmental Research Information, 1999c, 1999d). All samples were analysed at a laboratory accredited by NATA to the ISO17025 standard for these tests.

3. RESULTS

Real time direct monitoring

The results of the real-time monitoring conducted during the fire investigation process using direct-reading instrumentation are presented in Table III. Carbon monoxide, hydrogen sulfide and

atmospheric flammability did not occur at dangerous levels at any of the scenes during the fire investigations, and the oxygen content of each atmosphere was within the accepted normal range of 19.5% to 23.5%. No significant increase in volatile organic compounds above background levels were detected in the breathing space of fire investigators at any of the fire scenes, although elevated readings in non-breathing space areas were detected for volatile organic compounds at two of the fire investigations. Since these relate to the specific characteristics of the fire investigations, they are outside the scope of this project and have been excluded.

Table III Results of monitoring using direct-read instrumentation

	Fire Scene 1	Fire Scene 2	Fire Scene 3	Exposure Standard
Oxygen	20.9%	20.9%	20.9%	19.5% (min) 23.5% (max)
Flammability	3%	0%	0%	
Carbon monoxide	0 ppm	1 ppm	0 ppm	30 ppm
Hydrogen sulfide	0 ppm	0 ppm	0 ppm	10 ppm
VOCs	0 ppb	0 ppb	0 ppb	

Volatile Organic Compounds

A summary of the quantitative results of the GC/MS analysis of the Tenax/Carboxen 569 tubes used for collection of air samples at the three fire scenes is shown in Table IV. The exposure standard values (converted to $\mu\text{g}/\text{m}^3$ assuming standard temperature and pressure conditions) are also shown for comparison. Since the sampling volume for the various samples was not constant, the reporting limits for the various samples are also not constant. Compounds detected as atmospheric contaminants at more than one fire scene included benzene, toluene, xylenes and trimethyl benzenes. Overall, the greatest number of contaminants and highest levels of air contamination were detected at Fire Scene 3. This was not unexpected, as the investigation at this fire scene commenced within three hours of the departure of fire crews and thus ventilation effects would be expected to have been reduced. It is also possible that some or all of the compounds only detected as atmospheric contaminants at Fire Scene 3 (n-hexane, n-heptane and methyl ethyl ketone) may be from materials that were not present at Fire Scene 2 (which had a similar sampling volume) or as a result of different combustion conditions. Even at this fire scene, however, concentrations of individual compounds do not approach exposure standard levels.

Table IV Results of GC/MS analysis of volatile organic compounds sampled at fire scenes

Compound	Retention Time (minutes)	Measured Concentration (mg/m^3)				Australian Exposure Standard (converted to mg/m^3)
Benzene	10.8	< R.L.	< R.L.	0.010	0.031	3.2
Toluene	14.8	0.030	0.030	0.0064	0.066	189
Ethyl benzene	18.0	< R.L.	< R.L.	< R.L.	0.010	434
Xylenes (total)	18.6	0.036	0.041	< R.L.	0.056	350
Trimethyl benzenes	21.7	0.020	< R.L.	< R.L.	0.022	123
Methyl cyclohexane	13.5	< R.L.	< R.L.	< R.L.	< R.L.	1600
n-Hexane	8.7	< R.L.	< R.L.	< R.L.	0.0068	72
n-Heptane	12.5	< R.L.	< R.L.	< R.L.	0.0074	1640

<i>n</i> -Octane	16.1	< R.L.	< R.L.	< R.L.	< R.L.	1400
<i>n</i> -Nonane	19.1	< R.L.	< R.L.	< R.L.	< R.L.	1050
<i>n</i> -Decane	21.8	< R.L.	< R.L.	< R.L.	< R.L.	Not available
Dichloromethane	6.2	< R.L.	< R.L.	< R.L.	< R.L.	174
1,1,1-Trichloroethane	10.1	0.010	< R.L.	< R.L.	< R.L.	546
Trichloroethene	12.2	< R.L.	< R.L.	< R.L.	< R.L.	54
Tetrachloroethene	16.5	< R.L.	< R.L.	< R.L.	< R.L.	340
Methyl ethyl ketone	8.0	< R.L.	< R.L.	< R.L.	0.0067	440
Ethyl acetate	8.8	< R.L.	< R.L.	< R.L.	< R.L.	720
Methyl isobutyl ketone	13.6	< R.L.	< R.L.	< R.L.	< R.L.	205
2-Butoxyethanol	18.9	< R.L.	< R.L.	< R.L.	< R.L.	96.9
Reporting Limit (R.L)		0.0083	0.025	0.0063	0.0063	
Fire Scene		1	1	2	3	
Sample volume		6.0 litres	2.0 litres	8.0 litres	7.5 litres	

Carbonyls

The results of High Performance Liquid Chromatography analysis of the extracts from the DNPH cartridges used for carbonyl sampling are shown in Table V. For each fire scene, the total amount of each target carbonyl present in the extract is presented. Due to the analysis process, each target compound has a different reporting limit (Kratzmann, 2005). Consequently, values less than the reporting limits for the individual chemicals have been expressed in Table V as being less than specific values, rather than a generic reporting limit. At the first fire scene, the limitations on sampling resulted in carbonyl quantities being below the reporting limit for all target compounds. At the third fire scene, pump failure during sampling and subsequent identification of the need to recalibrate the sampling rate resulted in the actual volume of air sampled being unknown. Therefore, only the results from Fire Scene 2 have been expressed both as total compound quantities and atmospheric concentrations.

Table V Results of HPLC analysis of carbonyl compounds sampled at fire scenes

Compound	Amount of Compound Present in Sampling Cartridge (μg)			Measured Concentration (mg/m^3)	Australian Exposure Standard (converted to mg/m^3)
	1	2	3		
Fire Scene	1	2	3	2	
Sample volume	6.0 litres	62.5 litres	Unknown	62.5 litres	
Formaldehyde	< 0.61	0.895	1.18	0.0143	1.2

Acetaldehyde	< 0.32	2.44	7.95	0.0390	36
Acrolein	< 0.28	0.68	0.805	0.011	0.23
Acetone	< 0.43	1.09	1.605	0.0174	1185
Propionaldehyde	< 0.89	< 0.89	< 0.89	< 0.014	Not available
Crotonaldehyde	< 0.34	< 0.34	< 0.34	< 0.005	5.7
Methacrolein	< 0.57	< 0.57	< 0.57	< 0.009	Not available
2-Butanone	< 0.51	< 0.51	< 0.51	< 0.008	445
Butyraldehyde	< 0.53	< 0.53	< 0.53	< 0.008	Not available
Benzaldehyde	< 0.37	< 0.37	0.64	< 0.006	Not available
Valeraldehyde	< 1.05	< 1.05	< 1.05	< 0.0168	176
p-Tolualdehyde	< 0.37	< 0.37	< 0.37	< 0.006	Not available
Hexaldehyde	< 0.32	0.845	0.68	0.0135	Not available

Whole air samples

Table VI presents the results from the analysis of the whole-air canister samples collected at Fire Scenes 2 and 3 (due to difficulties encountered during sampling, no whole-air canister sample was collected at Fire Scene 1). Results for Fire Scene 2 were the averaged results from two injections into the gas chromatograph, while those for Fire Scene 3 were from a single injection. The majority of target compounds in this analysis were not detected at or above the reporting limit. Compounds detected at the fire scenes included acrylonitrile, benzene, toluene, styrene, xylene and trimethylbenzene. Concentrations for each of the detected compounds were substantially below the relevant Australian Exposure Standards.

4. DISCUSSION

The various compounds detected during this study of air contaminants present during fire investigations at residential premises include a number of chemicals for which occupational exposure standards exist in Australia, due to short-term and/or long-term health effects from exposure to these chemicals at particular levels (National Environment Protection Council, 2003). Compounds consistently detected across multiple residential fire scenes included benzene, toluene, xylene, styrene, formaldehyde, acetaldehyde, acetone, acrolein, and hexaldehyde. Other compounds, such as hexane, heptane, methyl ethyl ketone, acrylonitrile and trimethylbenzene, have also been found in low concentrations at one of the fire investigations covered by this study. However, all of the compounds detected were considerably below the Australian Exposure Standards for occupational exposure at all fire investigation scenes. These results therefore support current Queensland Fire and Rescue Service practices relating to respiratory protection used at fire investigations at residential premises. Although no levels of concern were detected during direct-reading measurements at the fire investigations, it is recommended that direct-reading measurement of oxygen levels, atmospheric flammability, carbon monoxide, hydrogen sulfide, acid gases and volatile organic compounds be used for safety.

Several significant factors should be considered in the interpretation of the results of this study. Firstly, the results of this study suggest that fire investigations conducted within a short time interval after the completion of firefighting duties are those in which higher levels of contaminants could be expected to be present. This is likely to be due to less time for ventilation to have occurred

after extinguishment activities have ceased. However, a larger study involving more fire investigation scenes would be required to properly investigate the interplay between the factors of quantity and identity of fuel loads and pre-investigation ventilation intervals in determining levels of air contaminants.

Secondly, measurements of air contaminant concentrations cannot be taken as direct measures of fire investigator exposure, since fire investigators do not spend all their time at an incident within the area subject to air contamination. However, this study does provide quantitative data regarding the levels of a range of air contaminants present after fires in residential structures. This allows informed decisions to be made regarding appropriate respiratory protective equipment for personnel working in these areas, and also regarding the handover of premises to owners, occupants or other organisations.

Thirdly, there are other potential contaminants generated at fire scenes that have not been investigated in this study, such as polycyclic aromatic hydrocarbons (PAHs). This class of possible air contaminant at fire investigation scenes remains unquantified, and is of considerable interest in terms of occupational health due to the known carcinogenic or mutagenic properties of some of these compounds (Lim et al., 2005; National Environment Protection Council, 2003). It is therefore recommended that further study into this aspect of fire investigation atmospheres be conducted in the future.

Finally, minimal research appears to have been conducted worldwide on air contaminants present in post-fire atmospheres in other types of buildings (for example, commercial premises) and other types of fires (for example, vegetation and vehicle fires). It is important that the identities and levels of air contaminants present in these situations also be quantified, so that more complete information on occupational exposures of fire investigation personnel can be obtained.

REFERENCES

- Austin, C. C., Dussault, G. and Ecobichon, D. J. (2001a) Municipal firefighter exposure groups, time spent at fires and use of self-contained breathing apparatus. *American Journal of Industrial Medicine* **40**, 683-692.
- Austin, C. C., Wang, D., Ecobichon, D. J. and Dussault, G. (2001b) Characterisation of volatile organic compounds in smoke at experimental fires. *Journal of Toxicology and Environmental Health, Part A* **63**, 191-206.
- Austin, C. C., Wang, D., Ecobichon, D. J. and Dussault, G. (2001c) Characterization of volatile organic compounds in smoke at municipal structural fires. *Journal of Toxicology and Environmental Health, Part A* **63**, 437-458.
- Barnard, R. J. and Weber, J. S. (1979) Carbon monoxide: a hazard to fire fighters. *Archives of Environmental Health*, 255-257.
- Bolstad-Johnson, D. M., Burgess, J. L., Crutchfield, C. D., Storment, S., Gerkin, R. and Wilson, J. R. (2000) Characterization of firefighter exposures during overhaul. *American Industrial Hygiene Association Journal* **61**, 636-641.
- Brandt-Rauf, P. W., Fallon, L. F., Tarantini, T., Idema, C. and Andrews, L. (1988) Health hazards of fire fighters: exposure assessment. *British Journal of Industrial Medicine* **45**, 606-612.
- Brown, S. (2005) Personal exposure of firefighters to air toxics and OHS risk management strategies. Bushfire CRC, Brisbane.
- Burgess, J. L. and Crutchfield, C. D. (1995) Tucson fire fighter exposure to products of combustion: a risk assessment. *Applied Occupational and Environmental Hygiene* **10**, 37-42.
- Center for Environmental Research Information (1999a) *Compendium Method TO-11A: Determination of formaldehyde in ambient air using adsorbent cartridge followed by High Performance Liquid Chromatography (HPLC)*. United States Environmental Protection Agency, Cincinnati.
- Center for Environmental Research Information (1999b) *Compendium Method TO-13A: Determination of polycyclic aromatic hydrocarbons (PAHs) in ambient air using gas*

- chromatography/mass spectrometry (GC/MS)*. United States Environmental Protection Agency, Cincinnati.
- Center for Environmental Research Information (1999c) *Compendium Method TO - 14A: Determination of Volatile Organic Compounds (VOCs) in Ambient Air Using Specially Prepared Canisters with Subsequent Analysis by Gas Chromatography*. United States Environmental Protection Agency, Cincinnati.
- Center for Environmental Research Information (1999d) *Compendium Method TO - 15: Determination of Volatile Organic Compounds (VOCs) in Air Collected in Specially Prepared Canisters and Analyzed by Gas Chromatography / Mass Spectrometry (GC / MS)*. United States Environmental Protection Agency, Cincinnati.
- Center for Environmental Research Information (1999e) *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air: Second Edition: Compendium Method TO-17 - Determination of Volatile Organic Compounds in Ambient Air Using Active Sampling Onto Sorbent Tubes*. United States Environmental Protection Agency, Cincinnati.
- Gold, A., Burgess, W. A. and Clougherty, E. V. (1978) Exposure of firefighters to toxic air contaminants. *American Industrial Hygiene Association Journal* **39**, 534-539.
- Jankovic, J., Jones, W., Burkhart, J. and Noonan, G. (1991) Environmental study of firefighters. *Annals of Occupational Hygiene* **35**, 581-602.
- Kratzmann, S. (2005) *The determination of formaldehyde and other carbonyls in ambient air as per USEPA TO-11A*. Queensland Health Pathology and Scientific Services ed. Queensland Government, Brisbane.
- Lees, P. S. J. (1995) Combustion products and other firefighter exposures. *Occupational Medicine: State of the Art Reviews* **10**, 691-706.
- Lim, M. C. H., Ayoko, G. A. and Morawska, L. (2005) Characterization of elemental and polycyclic aromatic hydrocarbon compositions of urban air in Brisbane. *Atmospheric Environment* **39**, 463-476.
- Lowry, W. T., Juarez, L., Petty, C. S. and Roberts, B. (1985) Studies of toxic gas production during actual structural fires in the Dallas area. *Journal of Forensic Sciences* **30**, 59-72.
- National Environment Protection Council (2003) *Impact Statement for the National Environment Protection (Air Toxics) Measure*.
- Rao, H. V. and Brown, D. R. (1990) House fire: a source of dioxins. *Risk Analysis* **10**.
- Ruokojärvi, P., Aatamila, M. and Ruuskanen, J. (2000) Toxic chlorinated and polyaromatic hydrocarbons in simulated house fires. *Chemosphere* **41**, 825-828.
- Treitman, R. D., Burgess, W. A. and Gold, A. (1980) Air contaminants encountered by firefighters. *American Industrial Hygiene Association Journal* **41**, 796-802.
- Wobst, M., Wichmann, H. and Bahadir, M. (1999) Surface contamination with PASH, PAH and PCDD/F after fire accidents in private residences. *Chemosphere* **38**, 1685-1691.

SMOKE EXPOSURE DURING BUSHFIRE FIREFIGHTING OPERATIONS

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Abstract

Bushfire firefighters face many hazards and the risk of injury, whether it is directly related to the fire front or as a result of an accident during firefighting tasks, can be extremely high. There is also a growing concern regarding the effect of bushfire air toxic emissions on firefighter health, both short-term and long-term. It is not known whether exposures to air toxics in bushfire smoke might impair the firefighters' abilities to perform tasks both mentally and physically and potentially affect their safety on the fire ground. Current research on firefighters' exposure to bushfire smoke is limited and has primarily been carried out in the USA. In Australia, there is a lack of knowledge of firefighters' exposures to air toxics and results from the USA may not necessarily be applicable for Australian firefighters due to differences in vegetation type and firefighting operations. In addition, no standard of work practice for bushfire firefighting specifying either short-term or long-term safe levels of exposure exists in Australia or overseas. As a result current constraints on how the fire agencies can manage their operations to comply with Occupational Health and Safety regulations are the lack of air toxics exposure knowledge for Australian firefighters (both career and volunteers) and a lack of suitable occupational exposure standards. Current occupational exposure standards need to be adjusted to take into account extended work shifts, heavier workload and exposure to a mixture of air toxics. Furthermore, no standard is available for smoke particles from any source.

Over the last three years, air samples were collected within the breathing zone of firefighters during a range of prescribed burns and bushfires across Australia. The exposure assessment has shown that the primary air toxics of concern are carbon monoxide, respirable particles and formaldehyde, and that work activity is a major factor influencing exposure levels. These measurements have provided crucial information on magnitude, extent and frequency of personal exposure levels and identified a range of situations that may potentially cause occupational standards to be exceeded.

Introduction

Bushfire firefighters face a range of hazards in their occupation, and procedures are generally in place to manage many of them. However, one hazard for which there is currently little control is their exposure to bushfire smoke. Bushfire smoke is a complex mixture of gases and particles (Brauer, 1999; Ward, 1999), and when inhaled has the potential to cause adverse health effects. These health effects can be acute, resulting from short-term (usually high) exposure, or chronic, resulting from long-term (often low level) exposure over a period of time (Dost, 1991; Larson & Koenig, 1994). Some of the short-term health effects that firefighters may experience on the fire ground include respiratory or eye irritation, nausea, headaches, dizziness, or reduced work capacity. Those symptoms generally disappear once the hazard is removed. Chronic effects such as lung damage, heart disease or cancer may not occur for many years and causes of such diseases are often hard to identify.

Previous research on firefighters' exposure to bushfire smoke is mostly limited to extensive personal exposure studies of forest firefighters in the USA (Materna *et al.*, 1992; McMahon & Bush, 1992; Reh & Deitchman, 1992; Kelly, 1992a; Kelly, 1992b; Materna *et al.*, 1993; Reh *et al.*, 1994; McCammon & McKenzie, 2000; Reinhardt & Ottmar, 2000; Reinhardt *et al.*, 2000; Reinhardt & Ottmar, 2004). In Australia, exposure assessments of bushfire firefighters are very scarce, and the only available study, (Brotherhood *et al.*, 1990), concentrated solely on carbon monoxide (CO) exposures measured in exhaled breath analysis. However, to date there have been

no Australian studies that have evaluated exposure levels of bushfire firefighters to air toxics present in the bushfire smoke. Results from the USA may not necessarily be applicable for Australian firefighters due to differences in vegetation cover and firefighting operations. Therefore it is critical to assess exposure levels within the Australian context to determine what primary factors influence exposure levels and whether those exposures could result in health problems.

The present study focused on environmental monitoring, leading to a better characterisation of the bushfire firefighting work environment, in particular understanding and quantifying exposure levels to toxic air pollutants in bushfire smoke. The dose ingested by firefighters can then be determined by combining physiological data, such as ventilation rates and work activity levels, with monitored atmospheric composition data. Further toxicological input is then necessary to assess potential adverse health effects as a result of exposure to bushfire smoke. The goal is to create a safe and healthy work environment for firefighters by developing a strategy for controlling firefighter exposure to bushfire smoke to safe/acceptable levels. The findings from this study along with a better understanding of the bushfire environment will allow us to review accepted exposure indices such as those presented by the Australian Safety and Compensation Council, and to establish a work code for bushfire firefighting in Australia.

Occupational exposure standards

Firefighting is an occupational activity and therefore is covered by Occupational Health and Safety regulations relevant to any other workplace. However, there are specific features of firefighting that differentiate it from most other work activities, and so the Australian legislative requirements need to be reviewed in light of these features.

Existing occupational exposure standards (OES) relevant to the bushfire air toxics research are presented in Table I (NOHSC, 1995). They are presented as (1) time-weighted average (TWA) concentration, which is the average airborne concentration of a particular substance calculated over a normal eight hour working day for a five day working week for a sedentary work activity and (2) short-term exposure limit (STEL) which is a 15 minute average exposure. Neither STEL nor TWA should be exceeded.

The standards need to be adjusted to take into account the different work environment of bushfire firefighters, e.g. longer and irregular work shifts, heavier workload, exposure to a mixture of air toxics that may have interactive health impacts. While the list of OES (Table I) is extensive and identifies several types of respirable particles (RP), it does not provide OES for smoke particles from any source. Since the biological effects of particles are dependent upon the chemical composition and physical characteristics of particles, a better characterization of bushfire smoke particles is essential to determine a suitable exposure standard.

Table V Occupational exposure standards (NOHSC, 1995)

Air Toxic	TWA	STEL	Carcinogen Category ¹
Carbon monoxide (CO)	30 ppm	400 ppm (0 min) 200 ppm (15 min) 100 ppm (30 min) 60 ppm (60 min)	
Formaldehyde (HCHO) ²	1 ppm 0.3 ppm (proposed)	2 ppm 0.6 ppm (proposed)	2
Acrolein	0.1 ppm	0.3 ppm	
Acetaldehyde	20 ppm	50 ppm	
Benzene	1 ppm / 3.2 mg/m ³	-	1
Respirable particles (RP)			
Carbon black	3 mg/m ³		
Graphite dust	3 mg/m ³		
Wood dust (hardwood)	1 mg/m ³		
Wood dust (softwood)	5 mg/m ³		
Fumed silica	2 mg/m ³		
Talc dust	2.5 mg/m ³		

¹ Category 1 Carcinogens – established human carcinogens; there is sufficient evidence of a causal association between human exposure and development of cancer

Category 2 Carcinogens – probable human carcinogen; there is sufficient evidence to provide a strong presumption that human exposure might result in development of cancer

² The exposure level of 1 ppm should prevent any acute symptoms (eye & respiratory tract irritation). The recommended standard of 0.3 ppm will provide adequate protection against discomfort of sensory irritation and also provides a high level of protection for cancer

Experimental

Over the last 3 years, monitoring has been carried out at prescribed burns in Victoria, South Australia, Tasmania, Queensland and the Northern Territory and included fuel reduction burns, experimental burns and slash or heap burns in eucalypt or pine plantations. Monitoring was also conducted at bushfires in Victoria for the 2005/2006 and 2006/2007 bushfire season. The air toxics that were monitored included CO, RP, aldehydes and volatile organic compounds (VOCs); these are primary air toxics released during bushfires and could potentially impact firefighters' health if present at elevated levels.

At each site, the aim was to monitor the personal exposure of 3 to 6 firefighters according to their tasks. Firefighters selected usually included those who volunteered to participate in the research study. The selection was also made to ensure that different work activities were represented. No differentiation was made for age, gender, work experience on the fire ground or smoking habits. Some of the recruited firefighters may have endured higher smoke exposures or may have altered their normal duties to gain maximum smoke exposure, although this was not encouraged by researchers. The firefighters were asked to wear up to five sampling devices monitoring CO, RP, aldehydes and VOCs, such as benzene, for the full duration of their work shifts. The equipment consisted of a data logging sensor for CO and a micro pump with an adsorbent tube for collection of VOCs, both devices being placed in the top pocket of the firefighter's personal protective clothing. Air monitoring devices for RP and aldehydes were carried in a small 'camelpack' style backpack with an inlet tube placed on the harness within the breathing zone.

All data collected during the field monitoring program were combined to estimate personal exposures and risk potentials to firefighters for different tasks and fire types as a result of exposure to bushfire air toxics.

Results and Discussion

Personal breathing zone measurements collected at burns and bushfires between 2005 and 2007 included 114 average and peak measurements of CO, 58 average measurements of formaldehyde, acetaldehyde and acrolein, 40 average measurements of VOCs and 62 average measurements of RP.

Work shift assessment

Work shift durations at prescribed burns vary considerably (half-day to one-day shifts) and depend strongly on weather conditions, size and location of the burn, availability of resources and objectives of the burn. At bushfires, work shifts ranged on average from 6 hours up to 12 hours. Due to difficulties in getting access to firefighters and the limited comfort of wearing sampling devices, some of the sampling times did not represent the entire work shift. At prescribed burns, 61% of samples covered the entire work shift, 32% of samples covered a fraction of the work shift considered to be representative of the shift, and 7% of the samples covered a fraction of the shift that was not necessarily representative of the work shift.

Exposure assessment

Major air toxics identified in the personal breathing zone measurements included CO, RP, formaldehyde, acetaldehyde, acrolein, benzene, toluene, acetic acid and phenol. The primary air toxics of concern were CO, RP and formaldehyde, due to their potential to cause adverse health effects at the levels measured within the breathing zone. Even though benzene and other VOCs were present, their concentrations remained low. The results were in agreement with those reported in personal exposure studies of bushfire firefighters in the USA.

The environmental monitoring has shown that the majority of exposures are below OES (Figure 1, shown for average and maximum CO levels, RP and formaldehyde). In 3-6% of cases, exceedences were observed for TWA exposures to CO and RP. Formaldehyde levels remained below the current OES of 1.0 ppm, but exceeded the proposed limit of 0.3 ppm in 10% of the samples. However it should be noted that four aldehyde samples were overloaded and therefore exposure levels for those firefighters may have been much higher than reported, and potentially exceeding the current OES.

The data show a strong correlation between CO and RP, as highlighted by the strong correlation of the linear regression for average exposures to these pollutants ($r^2 = 0.86$, which is significantly different from the null hypothesis (no correlation) at $p=0.001$). Correlations with the same significance were also observed between CO and formaldehyde ($r^2 = 0.63$), CO and acetaldehyde ($r^2 = 0.49$) and RP and formaldehyde ($r^2 = 0.50$). No correlation was observed between CO and benzene ($r^2 = 0.06$), which is due to non-fire benzene emissions such as vehicle exhaust and fumes from the drip torches used to ignite prescribed burns.

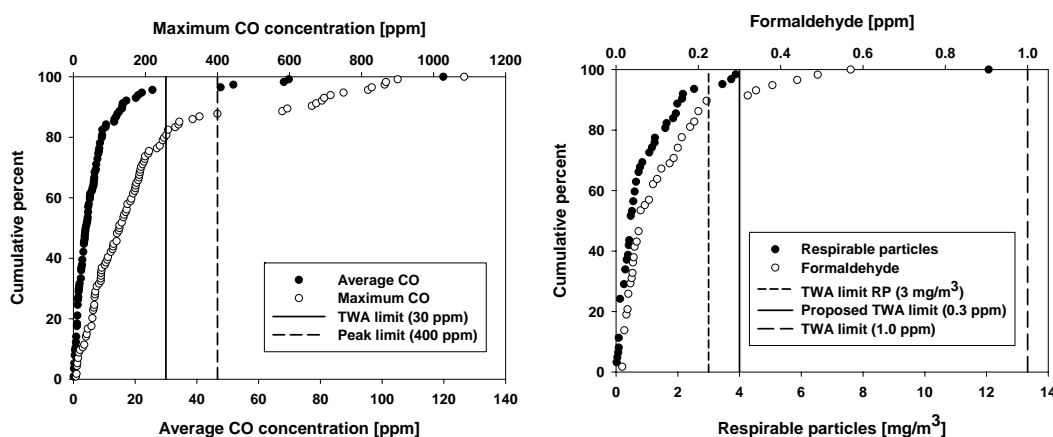


Figure 3 Distribution of personal exposure levels to carbon monoxide (average and peak), respirable particles and formaldehyde

The use of data-logging sampling devices for CO and RP enabled a better assessment of the extent and frequency of short-term exposure peaks in addition to provide an overall average exposure level over the duration of the work shift. Figure 2 shows the data-logging records of a firefighter's exposure to CO during fuel reduction burns and highlights the daily variability over a work shift. One of the records shows an overall average CO exposure level below 30 ppm but with a few excursions above 400 ppm. Those short-term peaks were measured during spot-fire suppression and usually lasted no longer than 1 minute. The other record displays an average CO exposure level largely exceeding the TWA exposure limit of 30 ppm and the STEL limit of 200 ppm (maximum 15-minute exposure measured at 310 ppm). The peak limit of 400 ppm has also been exceeded over a 4-5 min exposure time and at several occasions during the duration of the work shift.

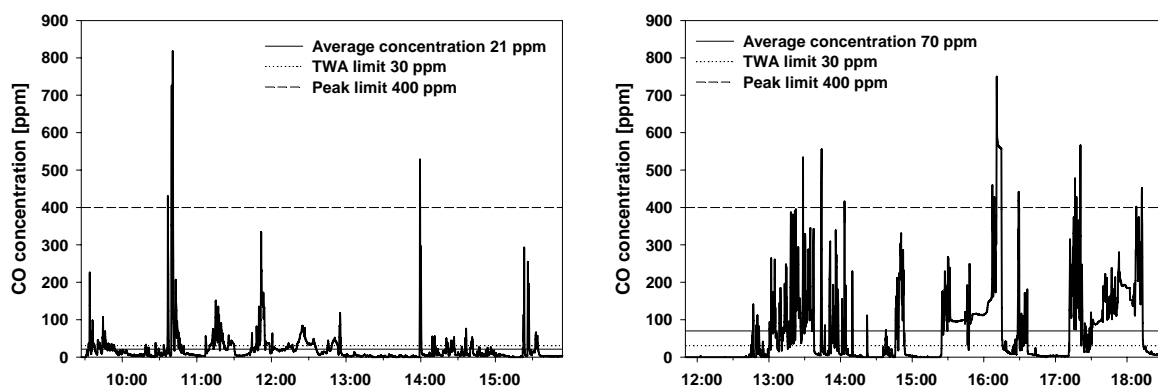


Figure 4 Data-logging records of firefighter's exposure to carbon monoxide

Work activity was a major factor influencing exposure levels. At prescribed burns, exposure levels were highest for crews involved in patrol and suppression, with lowest levels observed for lighting crews, who were primarily working upwind of the smoke (Figure 3). Due to VOCs emitted from the drip torches, exposure levels to total VOCs were highest for lighting crews. Short-term peak exposures were observed during suppression of spotfires, and in general, high risk situations that lead to elevated exposures to air toxics included suppression of spotfires, holding the fire line, and patrolling at the edge of a burn area in the urban interface.

At bushfires, primary work activities that were monitored included driving tankers on the fire ground, blacking out (e.g. making a fire safe after it has been controlled by extinguishing or removing burning material along or near the fireline), backburning and active fire suppression. Average CO and RP exposures were similar for drivers and firefighters working at the end of the hose. However firefighters involved in suppression and blacking out activities had higher short-term excursions compared to drivers.

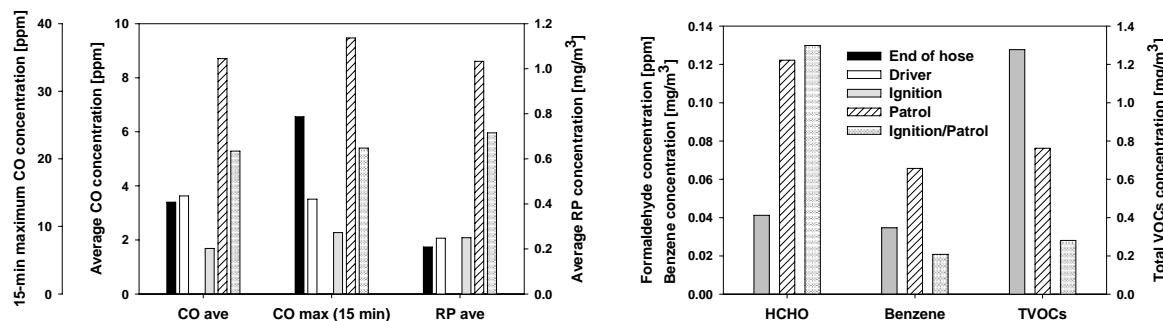


Figure 5 Personal exposures to air toxics in bushfire smoke according to work activities

Potential health impacts and control strategies

The environmental monitoring provided essential information on magnitude, extent and frequency of personal exposure levels and identified a range of situations that may potentially cause

exceedences of occupational standards. Primary air toxics of concern included CO, RP and formaldehyde.

Potential health impacts

CO is a colourless and odourless gas which when inhaled binds to haemoglobin in the blood. Carboxyhaemoglobin (COHb) is produced leading to reduced transport, delivery and utilisation of oxygen. CO has a half life of about 4-5 hours, and COHb levels will return to background levels once CO exposures are eliminated. Exposure to elevated levels of CO can result in cognitive impairment, reduced work capacity, dizziness and nausea. The risk for CO induced symptoms depends on each individual. The question is whether ongoing exposure to low levels of CO with occasional exposure to elevated levels of CO is likely to cause headaches, dizziness or behavioural effects. In order to minimise potential symptoms from overexposure to CO, it is recommended to keep COHb levels below 5%. Regular monitoring of firefighters COHb levels using exhaled breath will provide a good indication whether it is safe to keep a firefighter on the fire ground or whether additional time in a low CO environment is necessary.

Acute effects that could arise from exposure to elevated levels of RP and formaldehyde include irritation to eyes and the respiratory tract, breathing difficulty and coughing. In general these symptoms are likely to disappear once the hazard is removed. Low concentrations of respiratory irritants may also trigger asthmatic attacks in sensitised individuals.

Long term health effects that may possibly result from elevated exposure levels to RP include permanent lung damage. The risk for long-term health effects depends upon the magnitude and frequency of exposure, the duration of exposure (in years), exposure to other pollutant sources (e.g., vehicle exhaust, cigarette smoking), and the health status of the individual. Decrease in lung function could be assessed by measuring lung function at the beginning of the fire season and then again at the end of the fire season. In order to determine whether lung damage has been permanent, measuring lung function at the start of the next fire season will determine whether lung function has returned to initial value. It would be necessary to evaluate the individual's exposures to other pollutants potentially affecting the lung over the time period between fire seasons.

There is also a concern that some of the air toxics in bushfire smoke are carcinogenic. Formaldehyde has recently been classified by the International Agency for Research on Cancer (IARC) as a known human nasal carcinogen (Cogliano *et al.*, 2005) and its exposure levels should be kept as low as feasible. For average exposure levels of 0.3 ppm formaldehyde, the occupational risk for respiratory tract cancers after repeated exposure to formaldehyde by inhalation is likely to be low (NICNAS, 1996).

RP may contain potentially carcinogenic compounds such as polycyclic aromatic hydrocarbons (PAHs). Further analysis needs to be conducted to determine the chemical composition of bushfire smoke particles and assess their potential carcinogenicity. Classification as a carcinogen, which would require a lower TWA, need not apply if it can be shown that bushfire smoke particles contain less than 0.005 % w/w benzo[a]pyrene (NOHSC, 2004).

Control strategies

The findings from the field monitoring program will help in developing control strategies to minimize potential health impacts. Potential control strategies include policies and procedures for safe work practices such as frequent rotations among lighting and patrol crews, reducing exposure time in dense smoke, and hazard awareness training. In case high exposure situations cannot be avoided, wearing of respiratory protection may be necessary. However, since CO is not filtered out by respiratory masks, it is recommended that the amount of time spent in dense smoke be limited.

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References

- Brauer, M. (1999). Health impacts of biomass air pollution. In "Health Guidelines for Vegetation Fire Events: Background Papers". p.186-255. World Health Organization, Geneva.
- Brotherhood, J. R., Budd, G. M., Jeffery, S. E., Hendrie, A. L., Beasley, F. A., Costin, B. P. and Wu, Z. E. (1990). Fire Fighters Exposure to Carbon Monoxide during Australian Bushfires. *Am. Ind. Hyg. Assoc. J.* 51(4): 234-240.
- Cogliano, V. J., Grosse, Y., Baan, R. A., Straif, K., Secretan, M. B. and El Ghissassi, F. (2005). Meeting report: summary of IARC monographs on formaldehyde, 2-butoxyethanol, and 1-tert-butoxy-2-propanol. *Environ. Health Perspect.* 113(9): 1205-1208.
- Dost, F. N. (1991). Acute Toxicology of Components of Vegetation Smoke. *Rev. Environ. Contam. Toxicol.* 119: 1-46.
- Kelly, J. (1992a). Health hazard evaluation report, U.S. Department of Interior, National Park Service, Gallatin National Forest, Montana. HETA 91-312-2185. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and National Institute for Occupational Safety and Health. Cincinnati, OH.
- Kelly, J. (1992b). Health hazard evaluation report, U.S. Department of Interior, National Park Service, New River Gorge National River, West Virginia. HETA 92-045-2260. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and National Institute for Occupational Safety and Health. Cincinnati, OH.
- Larson, T. V. and Koenig, J. Q. (1994). Wood Smoke - Emissions and Noncancer Respiratory Effects. *Annu. Rev. Public Health* 15: 133-156.
- Materna, B. L., Jones, J. R., Sutton, P. M., Rothman, N. and Harrison, R. J. (1992). Occupational Exposures in California Wildland Fire Fighting. *Am. Ind. Hyg. Assoc. J.* 53(1): 69-76.
- Materna, B. L., Koshland, C. P. and Harrison, R. J. (1993). Carbon Monoxide Exposure in Wildland Firefighting: A Comparison of Monitoring Methods. *Appl. Occup. Environ. Hyg.* 8(5): 479-487.
- McCammon, J. and McKenzie, L. (2000). Health hazard evaluation report 98-0173-2782. Colorado Department of Public Health and Environment.
- McMahon, C. K. and Bush, P. B. (1992). Forest Worker Exposure to Airborne Herbicide Residues in Smoke from Prescribed Fires in the Southern United-States. *Am. Ind. Hyg. Assoc. J.* 53(4): 265-272.
- National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Department of Health and Ageing (1996) Formaldehyde, Priority Existing Chemical Assessment Report No. 28. (<http://www.nicnas.gov.au>)
- National Occupational Health and Safety Commission (1995) Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment. [NOHSC:1003(1995)] (<http://www.ascc.gov.au>)
- National Occupational Health and Safety Commission (1995) Guidance Note on the Interpretation of Exposure Standards for Atmospheric Contaminants in the Occupational Environment [NOHSC:3008(1995)] 3rd Edition
- National Occupational Health and Safety Commission (2004) Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)]
- Reh, C. M. and Deitchman, S. D. (1992). Health hazard evaluation report, U.S. Department of Interior, National Park Service, Yellowstone National Park, Wyoming. HETA 88-320-2176. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and National Institute for Occupational Safety and Health. Cincinnati, OH.
- Reh, C. M., Letts, D. and Deitchman, S. (1994). Health hazard evaluation report, U.S. Department of Interior, National Park Service, Yosemite National Park, California. HETA 90-0365-2415.

- U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and National Institute for Occupational Safety and Health. Cincinnati, OH.
- Reinhardt, T. E. and Ottmar, R. D. (2000). Smoke exposure at western wildfires. USDA Forest Service Pacific Northwest Research Station Research Paper (525).
- Reinhardt, T. E. and Ottmar, R. D. (2004). Baseline measurements of smoke exposure among wildland firefighters. *J. Occup. Environ. Hyg.* 1(9): 593-606.
- Reinhardt, T. E., Ottmar, R. D. and Hanneman, A. (2000). Smoke exposure among firefighters at prescribed burns in the Pacific Northwest. USDA Forest Service Pacific Northwest Research Station Research Paper (526): U1-45.
- Ward, D. E. (1999). Smoke from wildland fires. In "Health Guidelines for Vegetation Fire Events: Background Papers". p. 70-85. World Health Organization, Geneva.

SILICA TOXICITY AND EXPOSURE

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Abstract

The health hazards of airborne dust, including silicosis, have long been known. In the early part of the 1900's silicosis was widespread in miners in the gold mines of South Africa. Accelerated silicosis was common, with symptoms appearing after only 3-10 years of exposure.

Following much outcry, a number of dust control measures including adequate ventilation, wet-drilling and restricted blasting, began to be implemented and the average dust levels dropped from 20 mg/m³ in 1909 to about 1 mg/m³ in 1924.

However, despite a great deal of research and improvements in mining technology over the next 40 to 50 years, there has been little evidence of any meaningful decline in respirable dust levels.

This period of complacent neglect was possibly caused, or exacerbated by, the migrant labour policies of the time which often saw affected miners being sent home at the end of their contracts to rural areas, where medical resources to identify silica-related diseases were scant. In addition, the extremely low levels of compensation paid to black mineworkers during this period and the correspondingly low compensation levies paid by mines provided little financial incentive for employers to tackle dust problems in the mines.

The levels of silicosis in South African mine workers remains at an unacceptably high level of up to 20% in current long-service employees and up to 30% in retired miners. Exposure to silica dust is known to reduce resistance to lung infections, particularly tuberculosis. South Africa has amongst the highest tuberculosis rates in the world. It also has seen the emergence of an HIV/AIDS epidemic. The risk of tuberculosis in workers with both silicosis and HIV infection is multiplicative and estimated to increase the susceptibility to tuberculosis by 15 fold.

Black South African gold miners have a tuberculosis incidence of around 3 000 per 100 000, in comparison with rates of around 30 in Cuba and the United States.

1. Introduction

1.1 Silica Toxicity

Approximately 28% of the earth's crust is comprised of silicon. Silica contains an atom of silicon and two atoms of oxygen (SiO₂). Many minerals contain SiO₂ combined with other elements and these are known as silicates. The various forms of asbestos, for example, are silicates. Silica, uncombined with other elements, is sometimes called 'free silica'. The most common crystalline form is quartz. It is silica's peculiar crystalline structure that is believed to be responsible for causing lung damage.

The mechanism for the development of silicosis and lung tumours in man and animals exposed to crystalline silica is still unclear. Silica particles are non-toxic in the ordinary sense of the word, but they breach the defence mechanisms of the lung and provoke the growth of excessive amounts of fibrous connective tissue. The dust-clearing function of the lungs involves macrophages (free-lying defensive tissue cells). Usually the enzymes of the macrophages are able to absorb and destroy foreign bodies. Silica particles however, seem to be able to both resist the enzymes and to alter them. When the macrophage dies the silica particles and the altered enzymes are released back into the lung tissue. Fresh macrophages then congregate to attack the foreign bodies, only to be altered

and destroyed in turn. Chronic inflammation results, and ironically the healing process is fibrosis. This fibrosis obliterates some air sacs and blood capillaries and diminishes the aerating surface area of the lung and the quantity of blood coming into contact with the air.

1.2 Health effects

Crystalline silica was classified as a human carcinogen in 1997 by the International Agency for Research into Cancer (IARC). Other diseases associated with respirable crystalline silica include:

- Pneumoconiosis
 - Chronic silicosis (10-20 yrs of exposure, often no symptoms)
 - Accelerated silicosis (3-10 yrs of exposure)
 - Alveolar lipoproteinosis (sometimes after only a few months of intense short exposures)
 - Progressive massive fibrosis (PMF)
- Chronic bronchitis
- Emphysema
- Mineral dust airways disease (MDAD)
- Mycobacterial disease
 - Pulmonary and extra-pulmonary tuberculosis
 - Non-tuberculous mycobacterial (NTM) disease
- Lung cancer
- Autoimmune diseases
- Scleroderma
 - Systemic lupus erythromatosis
 - Rheumatoid arthritis
- Renal disease

Silicosis is a progressive, irreversible disease that often takes years to develop. It progresses even after exposure ceases and is often only diagnosed radiologically, before any symptoms are observed. It is one of the oldest known occupational illnesses. Agricola documented the role of dust in producing 'suppuration' of the lungs in his famous work, *De re Metallica*, published in 1556.⁽¹⁾

Silicosis is a form of pneumoconiosis. Pneumoconiosis means 'dust-containing lung'. It is an incurable, often fatal, occupational disease caused by prolonged exposure to dust containing fine silica particles. In its simple form, chronic silicosis, it is a slow developing disease but there is also a swiftly advancing version called accelerated silicosis. Neither is infectious nor contagious, but both reduce the sufferer's resistance to lung infections, particularly tuberculosis.

The aetiology of silicosis includes many complex variables, including the duration of dust exposure, the percentage of silica in the dust and the intensity of the dust dose. The shape and size of the dust particles are also important. The development of the disease is just as complex and is still the subject of some controversy.

1.3 Prevalence

Silicosis is not just a South African disease; at least 1,7 million workers in the United States are potentially exposed to crystalline silica.⁽²⁾ However, up to 20% of older, in-service gold miners in South Africa have silicosis and higher prevalences are found in ex-miners.⁽³⁾⁽⁴⁾⁽⁵⁾ The South African mining industry had 493 000 employees in June 2007.⁽⁶⁾ The gold industry is the largest employer, responsible for more than 50% of total employment.⁽⁷⁾

The prevalence of silicosis has been decreasing in high-income countries. However, in contrast, low income countries such as South Africa have large populations at risk, poor capacity to control dust and to enforce legislation, and many competing health needs, including traumatic injury and infectious diseases.

2. History of silicosis in South African mines

2.1 1886-1939 Recognition

Gold was discovered in 1886 where Johannesburg is now and the mining industry rapidly developed. Silicosis and other dust-related diseases were recognized very early. A Commission on Miners Phthisis reported in 1903 on the hazards of airborne dust and in 1911 a medical commission found that about 26% of working miners were affected by silicosis.

There was a general outcry about the unhealthy working conditions in the mines. In 1913 Reynold's Newspaper reported "*It is obvious that the gold mines of the Transvaal are little better than charnal houses ... Men are being choked by the dust from the rock drills and then flung away as pitilessly as if they were mere bundles of old clothes.*"

Dust monitoring was in place in these early days. However early dust-measuring devices consisted of a tube through which a fixed amount of dust-laden air was sucked into a dust trap. At first cotton wool was used which was then incinerated so that the dust in the ash could be measured. Later sugar was used and the sugar was dissolved in water, filtered and the filter burnt and weighed – known as the 'sugar tube'.

In 1913 Dr John McCrae, a South African chemist, found that only particles less than twelve micrometres were found in silicotic lungs and therefore large particles presumably did not play an important part in silicosis. In general, the greater the mechanical forces applied, the more likely is the generation of small particle (respirable) dust. The increase in mechanical drilling in this period, along with rock blasting, contributed to small particle exposure. Mechanised rock drills were already in use by more than 90% of mining companies in 1895. The dust concentrations generated by these drills were such that it was often impossible to make out a man standing 2 metres away.

The konimeter became the preferred sampling device for respirable dust particles. This collected only the smaller particles on to a glycerine-coated glass slide where they could be counted under a microscope.

The Miners' Phthisis Act, passed in 1912, was a far-reaching piece of legislation. The act made provision for adequate ventilation, wet-drilling and restricted blasting. Pre-employment fitness medicals were introduced and silicosis, with or without tuberculosis, became a compensable disease. This was 15 years before similar legislation was introduced in the United States.

In 1909 samples contained around 20 mg/m³ dust but by 1924 the average level had dropped to about 1 mg/m³. [REF]

As a result, a dramatic decrease in the incidence of accelerated silicosis was observed and the length of time that miners were exposed before silicosis was detected demonstrated this improvement.⁽⁸⁾

1918-1920	9 years
1929-1932	13 years
1934-1935	16 years

In 1929 Dr A Mavrogordato, Fellow in Industrial Hygiene at the South African Institute for Medical Research (a forerunner to the National Institute for Occupational Health), and Dr LG Irvine, chairman of the Miners' Phthisis Medical Bureau, gave a good account of silicosis on the Witwatersrand in the early days of mining.⁽⁹⁾ They were also instrumental in setting up the first International Silicosis Conference, which was held in Johannesburg in 1930 under the auspices of the League of Nations.

2.2 1940 – 1996 Complacent neglect

The period between 1940 and 1996 was a time of intensive research into silicosis. A Pneumoconiosis Research Unit (PRU), another forerunner to the National Institute for Occupational Health (NIOH), was formed in South Africa in 1956. Scientists from many countries were invited to Johannesburg to advise the PRU research team on their research projects. This resulted in the second International Conference on Pneumoconiosis held in Johannesburg, South Africa in 1959.

Modern principles of dust sampling for hazard assessment are founded on three main recommendations adopted at the 1959 Conference, and have remained largely unchanged to the present day:

- (i) measurement of the respirable dust fraction
- (ii) gravimetric assessment of the collected dust
- (iii) long-period sampling to give average dust levels over a shift.

A third International Conference on Pneumoconiosis was held in Johannesburg in 1969.

Despite this period of continued research there appears to have been little in the way of real dust control improvements. During 1995 to 1997, 26 000 dust samples were collected from 48 gold mines. Only 8 of these mines were able to show that all of their respirable crystalline dust levels were below the 0.1 mg/m^3 exposure limit.⁽⁸⁾

This period of complacent acceptance was possibly caused or exacerbated by the demand for ever greater production and the migrant labour policies of the time which often saw affected miners being sent home at the end of their contracts to rural areas, where medical resources capable of identifying silica-related diseases were scant. A 1997 Committee of Inquiry into a National Health and Safety Council in South Africa by Benjamin and Greef reported that a contributing factor was the extremely low levels of compensation paid to black mineworkers and the correspondingly low compensation assessment paid by mines. This provided little financial incentive for employers to tackle dust problems in the mines.

Larger mines were also permitted to monitor dust levels to determine their compensation levies. In the case of small mines, the state performed this function. The method of measurement adopted (gravimetric sampling) did not provide any meaningful feedback to control hazardous dust levels effectively. The result was a system in which more was spent on determining air quality indices for mines for the purpose of calculating contributions than on either controlling or rectifying hazardous conditions.

This period is aptly summed up in the findings of Justice RN Leon in the 1994 report of the Leon Commission of Inquiry into Safety and Health in the Mining Industry; "*... on the basis of dust measurements made between 1956 and 1960, ... there was little evidence of a decline in dust levels between 1938 and 1969.*"

2.3 1996 – 2007 Reawakening to the threat?

In response to the establishment by the joint International Labour Organisation, World Health Organisation (ILO/WHO) International Programme on the Global Elimination of Silicosis in 1995⁽¹⁰⁾, the South African Department of Minerals and Energy has set a national target to reduce occupational silica exposure by 2008, so that 95% of respirable dust measurements in their operations are below 0.1 mg/m^3 , and by 2013 no new cases of silicosis in previously unexposed workers are diagnosed.

The Mine Health and Safety Act (1996) and the occupational hygiene regulations published under this act (2002), have been developed by the South African Department of Minerals and Energy partly to improve legislation requiring mining houses to address dust.

The South African Department of Minerals and Energy has set an exposure limit (OEL) of 0.1 mg/m³ for respirable crystalline silica dust. However this has been shown not to be protective.⁽¹¹⁾ The risk of silicosis following a working lifetime of exposure at 0.05 mg/m³ is estimated to be as high as 20-40%.⁽¹²⁾ Simply reducing the exposure limit is unlikely to achieve the desired goal as South Africa has limited capacity to reliably measure respirable crystalline silica even at 0.1 mg/m³.

Despite the suspected under-reporting of respirable dust exposures by the mining industry, investigations by the NIOH into the Department of Minerals and Energy (DME) dust exposure database indicate that many industry exposure results are still in excess of the 0.1 mg/m³ limit.

According to figures tabled before the South African Parliament's Minerals and Energy Portfolio Committee in October 2007 by Thabo Gazi, the Chief Inspector of the Mine Health and Safety Inspectorate, mine doctors submitted 1 536 cases of silicosis to the Medical Bureau of Occupational Diseases in 2006 - an increase of almost 50% on the year before.

It must be noted that silicosis, except in the advanced stages, may not produce any disability and there may be no symptoms. Even with a suspected degree of under-reporting, the above disease figures would probably not elevate the elimination of silicosis to a priority in South Africa, where there are many occupational and general health problems, were it not for the association between silica exposure and tuberculosis.

This association between silicosis and TB has been known for a long time, but recent findings show that exposure to silica, even without silicosis, may also predispose individuals to tuberculosis.⁽¹³⁾ The increased risk is life-long even if exposure ceases. Tuberculosis rates in persons with simple silicosis can be up to three-fold higher than those in the same workforce without silicosis.⁽¹⁴⁾

South Africa has amongst the highest tuberculosis rates, including the multi-drug resistant forms, in the world. Black South African gold miners have an incidence of around 3 000 per 100 000, in comparison with rates of around 30 in Cuba and the United States.⁽¹⁴⁾

The emergence of the HIV/AIDS epidemic in South Africa has also contributed to the tuberculosis rate. The risk of tuberculosis in workers with both silicosis and HIV infection is more than the sum of the risks conferred by the two risk factors. The risk is multiplicative and is estimated to increase the susceptibility to tuberculosis by 15 fold.⁽¹⁵⁾

3. Challenges ahead

From an occupational hygiene perspective, there are many challenges to reducing the toll on miners' health caused, directly and indirectly, by exposure to respirable crystalline silica dust. Obviously exposure levels have to be reduced but methods to reliably and cost-effectively measure silica dust exposures below 0.1 mg/m³ are not readily available in South Africa. The outcomes of the deliberations of the International Standard Organisation (ISO) Working Group TC146/SC2/WG7 Silica which is addressing this issue, is awaited with interest.

In order to have credibility, the dust exposure results reported by the mines need to be verified by an independent authority. This would involve more than rechecking the analytical results. The entire measurement programme needs to be validated. This could include the procedure for the selection of the miners to be monitored, the sampling equipment used, the diligent recording of observations during sampling, the preparation and transport of the filters, the analysis and

calculation of the exposure, and the interpretation and presentation of the results in a meaningful way.

4. Conclusion

Silicosis has been a major occupational health issue in the mining industry for over 100 years. Despite numerous plans, commissions of enquiry, improvements in legislation, and many educational and training initiatives, it remains the mining industry's most important occupational health concern.

The contribution of silicosis and silica exposure to tuberculosis is relatively minor compared with the HIV/AIDS epidemic in South Africa. However the very large number of miners with silicosis and continued silica exposure has contributed to the high rate of tuberculosis in the mining industry and the communities surrounding mines.

5. References

- (1) Agricola, G. (1950). *De Re Metallica* (H. C. Hoover and L. H. Hoover, trans.). Dover, New York.
- (2) National Institute for Occupational Safety and Health. Hazard review: health effects of occupational exposure to respirable crystalline silica. Cincinnati, OH, USA: NIOSH, 2002.
- (3) Churchyard GJ, Ehrlich R, teWaterNaude JM, et al. Silicosis prevalence and exposure-response relations in South African goldminers. *Occup Environ Med* 2004;61: 811-816.
- (4) Steen TW, Gyi KM, White NW, et al. Prevalence of occupational lung disease among Botswana men formerly employed in the South African mining industry. *Occup Environ Med* 1997; 54: 19-26.
- (5) Trapido AS, Mqoqi NP, Williams BG, et al. Prevalence of occupational lung disease in a random sample of former mineworkers, Libode District, Eastern Cape Province, South Africa. *Am J Ind Med* 1998; 34: 305-313.
- (6) Stats SA, Quarterly employment statistics (QES) June 2007, as accessed on 9 November 2007 at <http://www.statssa.gov.za/PublicationsHTML/P0277June2007/html/P0277June2007.html>
- (7) MBendi, South Africa: Mining – Overview, as accessed on 9 November at <http://www.mbendi.co.za/indy/ming/af/sa/p0005.htm>
- (8) Rees D, et al. Occupational health indicators for South Africa; Part II. NCOH report, 1999: No. 1/99.
- (9) Irvine LG, Mavrogordato A. Miners' phthisis on the Rand. *J Chem Metal Mining Soc S Afr*, Nov 1929, pp 163-168.
- (10) Fedotov I. Global elimination of silicosis: the ILO/WHO international programme. *Asian-Pacific Newsletter on Occupational Health and safety* 1997; 4(2).
- (11) Steenland K. One agent, many diseases: exposure-response data and comparative risks of different outcomes following silica exposure. *Am J Ind Med* 2005; 48: 16-23.
- (12) Greaves IA. Not-so-simple silicosis: a case for public health action. *Am J Ind Med* 2000; 37: 245-251.
- (13) Hnizdo, E. & Murray, J. Risk of pulmonary tuberculosis relative to silicosis and exposure to silica dust in South African gold miners. *Occup Environ Med* 1998 55: 496-502.
- (14) Cowie RL. The epidemiology of tuberculosis in gold miners with silicosis. *Am J Respir Crit Care Med* 1994; 150: 1460-1462.
- (15) Churchyard G J, et al. Silicosis prevalence and exposure-response relations in South African goldminers. *Occup Environ Med* 2004, 61: 811-816.

CASE STUDIES IN HAZARDOUS SUBSTANCES AUDITING, NAMELY ASBESTOS CONTAINING MATERIALS

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ABSTRACT

A series of case studies will be the basis of this presentation and paper. These are taken from the author's activities over the past 5 years undertaking audits and remediation plans to decontaminate sites.

The author is finding that the focus on auditing building spaces is overlooking a higher risk hazard of friable asbestos contamination in soils.

A management approach to guide users of open spaces (eg. mainly schools and church groups) has been developed and examples will be presented for discussion.

The author has been associated with the management of asbestos related health risks since the mid 1970's and has lived through the changes that have occurred with the banning of the use of asbestos containing materials. However risk of exposure has changed from the factory environment to the community.

A zero risk policy for school children will be outlined.

ASBESTOS IDENTIFICATION LEAP OF FAITH

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Abstract

The presence/absence of asbestos in bulk materials must be based on sound scientific information if the best interest of those involved and the general public is to be served.

Polarised Light Microscopy (PLM) is the universally accepted test method for asbestos identification. It is adequate for most commercial samples. It is also universally accepted that certain sample types (e.g. vinyl tiles) are “*difficult or impossible to analyse*” by PLM because the asbestos fibres are very fine and intimately dispersed within organic matrix.

Test reports from NATA accredited laboratories for these sample types often say:

“No asbestos was detected by polarised light microscopy (PLM) but identification may not be possible due to adhering resins. Confirmation by another analytical technique is advised”

In Australia most laboratories do not confirm their findings by an independent method and this paper argues that reports with unconfirmed results are incomplete and should not be accepted by the industry. By and large the practice in Australia has been to ignore the need for confirmation and classify samples with doubtful results as not containing asbestos

Organisations like Australia Standard and NATA do not demand that testing laboratories using PLM have additional protocols when dealing with samples known to be problematic by this technique.

The distinction between “*asbestos not detected*” because of method limitations and “*asbestos not detected*” because asbestos is not present is self-evident. It is a leap of faith to assert the contrary, and it could have devastating potential legal ramifications.

There are several independent analytical techniques that could be used. It is a joint responsibility of the testing laboratories and their clients to make sure that potential “*false negatives*” are confirmed by an independent technique. This paper will concentrate on X-ray Diffraction (XRD) technique because of its ease of use and cost effectiveness.

1. Introduction

It is in the best interest of professionals working in asbestos related fields, laboratories that undertake asbestos identification work, companies engaged in asbestos surveys and asbestos registers, workers involved in asbestos removal, and the public in general that decisions as to the presence/absence of asbestos in bulk materials are based on sound scientific information.

2. Discussion

NATA endorsed Asbestos Identification reports issued in Australia often contain disclaimers similar to these.

“No asbestos was detected by polarised light microscopy (PLM) but identification may not be possible due to adhering resins. Confirmation by another analytical technique is advised”.

or

“Even after disintegration it can be difficult to detect the presence of asbestos in some asbestos-containing bulk materials using PLM and dispersion staining. This is due to the low grade or small length or diameter of the asbestos fibres present in the material, or to the fact that very fine fibres have been distributed intimately throughout the materials. Vinyl/asbestos floor tiles, some asbestos containing epoxy resins and some ore samples are examples of these types of material, which are difficult to analyse”.

Page 17 of the Supplementary Requirements for Accreditation in the Field of Chemical Testing by NATA in relation to asbestos testing states:

“If identification is not possible due to adhering resins or cements or because of degradation of the fibres, an explanatory note to that effect must be included on the report.”

In Australia, the mandatory method for the identification of asbestos in bulk samples is AS 4964-2004. The Preface states:

“The standard sets out relatively simple aspects of sample preparation and PLM that enable a large proportion of commercial samples to be identified, even though some samples will be difficult or impossible to analyse. These samples may require the use of an independent confirming technique such as infrared spectroscopy, X-ray diffraction, scanning electron microscopy or transmission electron microscopy, if PLM fails to give an unequivocal identification, or they require more complex sample preparation”

The Standard recognises that some samples are **“difficult or impossible to analyse”** and suggests the use of independent confirmatory techniques. Failure to see asbestos fibres by PLM in **“difficult or impossible”** samples must, therefore, fail to give **“unequivocal identification”**.

The Note in Section 8.3.2 of AS 4964-2004 provides indication of the materials it considers difficult or impossible to analyse by PLM. The Note states:

“Even after disintegration it may be difficult to detect the presence of asbestos in some asbestos-containing bulk materials using PLM and DS. This is due to the low grade or small length or diameter of the asbestos fibres present in the material, or to the fact that very fine fibres have been distributed intimately throughout the materials. Vinyl asbestos floor tiles, some asbestos containing sealants and mastics, asbestos-containing epoxy resins and some ore samples are examples of these types of material, which are difficult to analyse”.

Despite these known limitations, evidences and warnings, it is extremely unusual in Australia for laboratories testing aforementioned materials to confirm **“asbestos not detected”** by an independent method. In effect, reports dealing with these materials and without confirmation are incomplete and should not be accepted by the industry. The sole purpose of submitting samples for analysis is to ascertain the presence/absence of asbestos. Answers which essentially say **“we are not sure”** fail to satisfy this purpose.

There is a clear distinction between **“not detected”** because test method limitations and **“not detected”** because the sought material/compound is present at very low levels or not at all.

The Australian Code of Practice for the Management and Control of Asbestos in Workplaces (NOHSC:2018 (2005)) in Part 9.3 requires asbestos registers to contain **“the results of any analysis that has confirmed a material in the workplace is or is not an ACM (Asbestos Containing Material).”**

This specific requirement cannot be met if analysis reports upon which asbestos registers are based state that asbestos was not detected because the test method had difficulties in seeing asbestos. In the absence of confirmation analysis reports these samples should be “*assumed*” to contain asbestos and be classified accordingly. In reality these materials are inevitably classified as non-ACM. Unconfirmed test results are not acceptable in many fields, and should not be acceptable in this industry, particularly given the potential consequences of incorrect classification.

The Standard lists several independent analytical techniques to resolve asbestos identification problems by PLM. Taking the additional step of confirming results by an independent method would clearly place laboratories issuing PLM test reports, and professionals that base their decisions on those reports, into the ‘safe’ ground of having acted to the best of their knowledge and as far as the available technologies permitted them.

It is self-evident that statements such as “*asbestos not detected*” in inspection reports and asbestos registers are **meaningless and potentially dangerous** if reports upon which they are based are qualified with disclaimers **AND** there are no confirmation reports. It is a leap of faith to assume that “*asbestos not detected*” due to method/samples difficulties means “*asbestos not present*”. **The legal ramifications of such leap of faith could represent a fertile ground for litigation.**

Warnings in the AS 4964-2004 reflect warnings in the US EPA Method EPA/600/R-93/116 which lists the interferences in PLM as follows:

“fibrous and nonfibrous, organic and inorganic constituents of bulk samples may interfere with the identification of the asbestos mineral content. Binder/matrix materials may coat fibres, affect colour, or obscure optical characteristics to the extent of masking fibre identity. Fine particles of other materials may also adhere to fibres to an extent sufficient to cause confusion in identification”.

The earlier method EPA-600/M4-82-020 combines PLM and X-ray Diffraction (XRD) to resolve identification doubts. The method was issued in 1982 and was the **compliance** monitoring technique for EPA programs. By 1993, following further research, the EPA published an expanded protocol (EPA/600/R-93/116) that, in addition to XRD, included analysis by Transmission Electron Microscopy (TEM) as confirmatory techniques.

The National Occupational Health and Safety Commission in April 2005 published its “Guidance Note on the Membrane Filter Method for Estimating Air borne Asbestos Fibres” (NOHSC:3003(2005)). This document contains the following warning:

“It must be also recognised that the use of the Membrane Filter Method (MFM) has limitations when applied to monitoring samples containing plate-like or acicular particles (e.g. vermiculate, talc, gypsum and certain other mineral fibres), and consequently should not be implemented without a full qualitative understanding of the sampling environment.”

Amongst the “analytical methods” that provide a greater understanding of complex samples, PLM is listed along with XRD, SEM and TEM.

It follows that, irrespective of whether a laboratory is engaged in identifying asbestos in bulk materials or does air monitoring by MFM, the onus is on the laboratory to make sure that all appropriate steps, including confirmation by an independent method, are taken in the effective identification of asbestos in samples.

It is inadequate from a scientific view point and from a customer service prospective to issue reports with unconfirmed “*not detected*” results for samples types that have been identified as problematic. Competent laboratories close the service loop by supplying customers with confirmed results for

such samples. This provides surety to customers that all possible steps have been taken to effectively identify the presence of asbestos in submitted samples. Warning clients that there are unresolved problems with the information provided does not suffice if clients need the information to make defensible and appropriate decisions.

In submitting samples known to present problems by the PLM technique, e.g. vinyl tiles, customers should demand that the laboratory confirms all “*asbestos not detected*” results in such samples.

Australian Standard and NATA, having recognised the difficulties with PLM for certain samples, should make mandatory that laboratories engaged in asbestos identification analysis have in place protocols to confirm “*asbestos not detected*” results in such samples.

3. Principles of XRD

The distance between atomic layers in a crystal is of a similar magnitude of X-ray wavelengths. As a result, when X-rays enter a crystal, they strike atoms in the crystal lattice and are reflected. X-rays that pass through the first layer may be reflected by atoms in the second layer, or the next layer and so on. If the reflected x-rays from the various layers are out of phase they would cancel each other out and a detector to which these reflected rays are focused would produce no signal. However, conditions may exist when reflected waves from the various layers are in phase. Under these conditions they reinforce each other, i.e. constructive interferences, and the same detector would now detect a signal.

For constructive interference to exist, Bragg, in 1914, postulated that the difference in path length, and hence the distance the X-ray has to travel, has to be an integer multiple of the X-ray wavelength, and the incident angle must equal the reflecting angle.

Consider the diagram in Figure 1, two layers of atoms in a crystal lattice are separated by a distance (d). The incident beam contains rays that are in phase and parallel. When two such beams enter the crystal lattice, one strikes atom z while the 2nd beam travels to the 2nd layer and strikes atom B. Both beams are scattered and for them to be in phase and parallel, the 2nd beam has to travel the additional distance AB + BC. This distance must be a multiple integer (n) of the incident wavelength (λ).

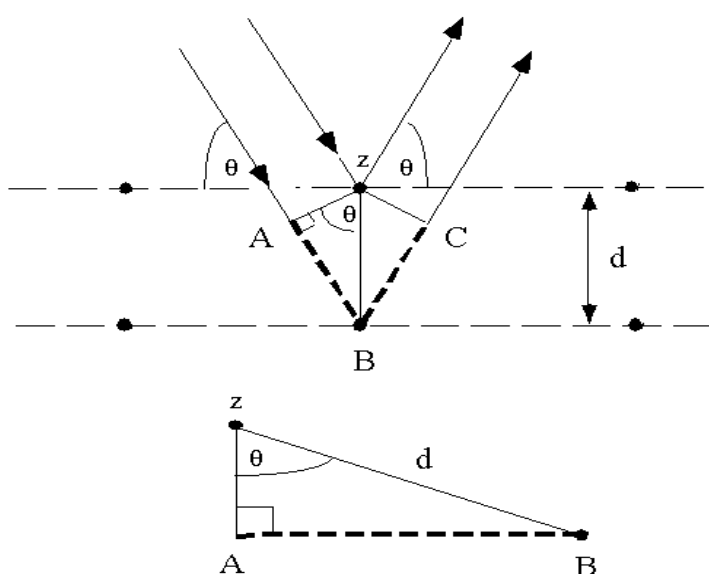


Figure 1: Derivation of Bragg's Law

$$n\lambda = AB + BC \quad (1)$$

The distance (d) is the hypotenuse of the right angle triangle ABz. Using trigonometry we can resolve AB in relation to d and the incident angle θ .

$$AB = d \sin\theta \quad (2)$$

Since $AB=BC$, substituting into (1) yields

$$n\lambda = 2 AB = 2 d \sin\theta \quad (3)$$

Bragg's law (Equation 3) earned Bragg and his son the 1915 physics Noble Prize.

The λ of the incident X-ray is known precisely, the angle at which constructive interferences occur is easily measured and hence d spacing of crystals can be determined with relative ease.

In practice, for each crystalline material there is a series of 2θ angles at which, under specific conditions, constructive interferences occur. When examining an unknown, identification is done by comparing found diffraction angles against published data.

4. XRD Application in Asbestos Identification

By orienting a sample in relation to an incident X-ray beam it is possible to collect a diffraction pattern which, in most cases, is unique and could be used to identify crystalline materials.

All forms of asbestos, i.e. *“the fibrous form of mineral silicates belonging to the serpentine and amphibole groups of rock-forming minerals”* are crystalline and hence have distinct X-ray diffraction patterns.

A sample containing asbestos, no matter how well dispersed or how fine the fibres are, will produce characteristic XRD diffraction patterns. Major diagnostic peaks and their relative intensities are shown in Table I.

Table I: A Guide of Principal Lattice Spacing of Asbestiform Minerals

Mineral	Principal d-spacing (Å)	Relative intensity	JCPDS File Ref.
Chrysotile	7.37	100	21-543
	3.65	70	
	4.57	50	
Amosite	8.33	100	17-745
	3.06	70	
	2.756	70	
Crocidolite	8.35	100	27-1415
	3.10	55	
	2.72	35	
Tremolite	8.38	100	13-437
	3.12	100	
	2.705	90	
Actinolite	2.72	100	25-157
	2.54	100	
	3.40	60	

Source: International Centre for Diffraction Data Powder Diffraction

A major advantage of XRD over other techniques, e.g. TEM and SEM, is the simplicity of sample preparation. The analysis is conducted at room temperature and pressure. A small sample of a few square centimetres with a freshly exposed face is held in a holder and rotated in relation to an X-ray

beam of known wavelength. The intensity of the signal is plotted against the 2θ angles and this is compared to the diffraction pattern produced by pure standards under identical conditions. The sample isn't destroyed and can be re-examined several times at various depths and orientation.

5. Sensitivity of XRD Method

The sensitivity of asbestos analysis by XRD is dependent on many factors, including matrix effect, interferences and absorption. Sanchez and Gunter (2006) reported using XRD to quantify amphibole content of expanded vermiculite down to 0.1%. Going to such low levels isn't commonly necessary. For example, bulk materials with less than 1% asbestos are not subject to regulation and aren't classified as ACM (USEPA 2006). Materials containing at least 1% asbestos produce strong XRD patterns. Table II lists the approximate asbestos content of various materials.

Table II: Asbestos Content of Various Materials

Product	% Asbestos	Product	% Asbestos
Vinyl Asbestos Tiles	21	Insulation Board	30
Asbestos Cement Sheet	20-50	Roofing Felt	10-15
Joint Compounds	3-5	Roofing Tiles	20-30
Textured Paint	4-15	Roofing Shingles	20-32
Vinyl Wallpaper	6-8	Putty	>20
Asphalt Asbestos Tiles	26-33	Cement Pipe & Fittings	>20
Resilient Sheet Flooring	30	Clapboards	12-15

An example of the power of XRD is shown in examining tiles marked A and B in Figure 2. Despite magnifying the edge of these two tiles some 100 times, no visible bundle of fibres is visible. Incinerating these tiles produced an amorphous material even more difficult to view under a microscope than the original sample. These two samples would be reported as “*asbestos not detected*” by PLM with qualification notes described earlier.

XRD analysis revealed that sample A was asbestos free and sample B contained chrysotile and several mineral fillers (Figure 3).



Figure 2: Two asbestos vinyl tiles and their magnified edges.

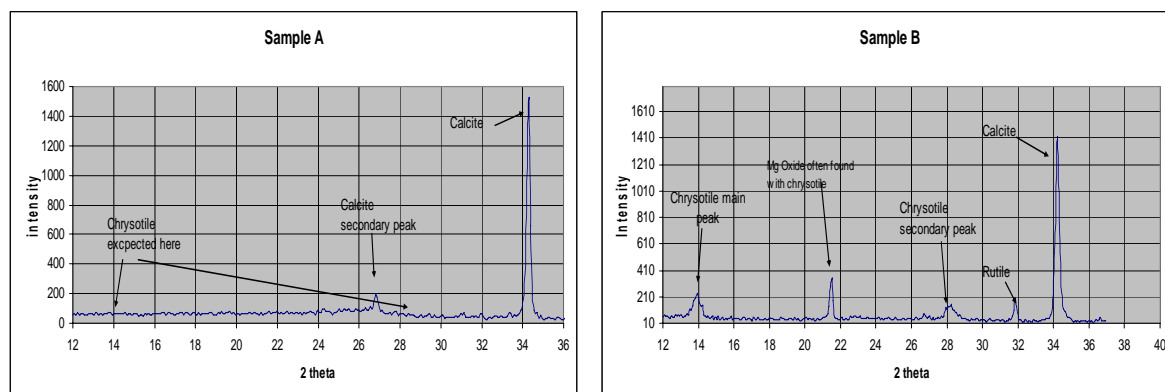


Figure 3: XRD scan of Samples A & B

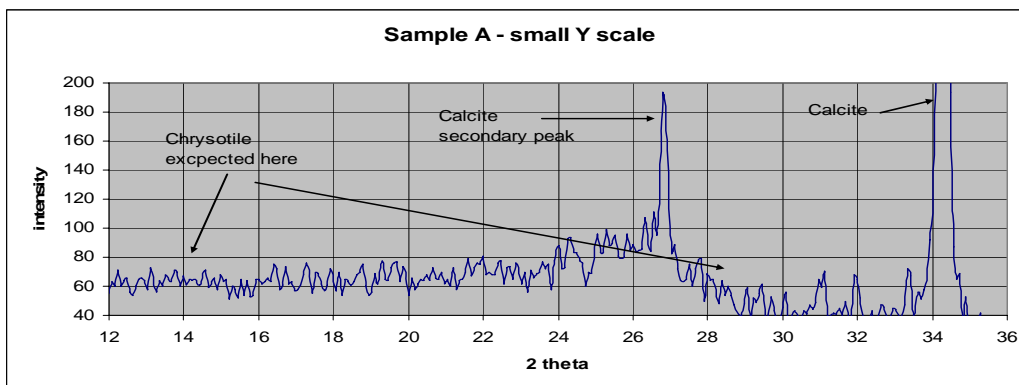


Figure 4: XRD scan of Sample A plotted on narrow y-axis

An advantage of instrumental analysis is that it allows for the signals to be inspected post data collection. Figure 4 is Sample A signal replotted on a narrower Y axis. The regions where chrysotile peaks are expected contain nothing more than baseline signals. The ability to store original data for review by more experienced analysts is very advantageous, facilitating the identification of asbestos in ‘difficult’ samples.

6. Interferences and Limitations

No analytical technique, no matter how sophisticated, is interference free. Interferences that influence the application of XRD for asbestos identification are well known (EPA-600/M4-82-020, Section 2.3) and can be divided into 2 categories:

- a. interferences due to non-fibrous forms of serpentine and amphibole minerals
- b. presence of other crystalline minerals with diffraction patterns that overlap that of asbestos minerals.

Interference from category (a) can’t be easily overcome. Optical methods of analysis have the ability to determine crystal morphology, XRD cannot, therefore the presence in a product of only non-fibrous forms of serpentine and amphibole minerals will lead to a false positive identification. The likelihood of the presence of only non-fibrous asbestos analogues in a manufactured product is very unlikely. Normally such interferences occur when examining naturally occurring products, e.g. during mineral exploration studies. It is unlikely that non-fibrous asbestiform minerals would be added to manufactured products where asbestos is normally used. When this type of interference occurs, XRD analysis will classify the product as ACM although it isn’t. This is a “fail safe” situation with no associated health risk as a result of incorrect identification. It is evident that it is considerably more risky to classify a product as non-ACM when the PLM fails to identify asbestos despite its presence.

Interferences of the (b) category are well known. They are summarised in Table III. If these minerals are present at sufficiently high levels then XRD will incorrectly classify a sample as an ACM. In these instances the ‘fail safe’ situation argued above also applies.

Table III: Interferences in XRD analysis of asbestiform minerals

Asbestiform Mineral	Primary Diagnostic Peak d-spacing (A)	Interference
Chrysotile	7.4	Chlorite Kaolinite Gypsum
	3.7	Chlorite Halloysite Cellulose
Amosite		Carbonates (>5%)

Anthophyllite Crocidolite Tremolite	3.1	Talc
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7. Benefits of XRD in Asbestos Identification Analysis

EPA method EPA/600/R-93/116, although recognising the limitations and interferences associated with XRD analysis of asbestos, states:

“When used in conjunction with optical methods such as PLM, XRD techniques can provide a reliable analytical method for the identification and characterisation of asbestiform minerals in bulk materials”.

And further

“This XRD method is applicable as a confirmatory method for identification and quantitation of asbestos in bulk material samples that have undergone prior analysis by PLM or other optical methods”.

XRD analysis is reasonably rapid and requires minimum sample preparation.

The major benefit of XRD analysis is that samples showing no peaks in the region where asbestiforms normally have peaks can be definitively classified as non-ACM. Decisions based on confirmed asbestos identification by XRD have the assurance that all materials containing >1% asbestos (or asbestiform analogues) are classified as ACM.

In the event that XRD has given false positive asbestos identifications due to interferences, then ‘fail safe’ decisions will be made with no adverse consequences to the health of those that may come into contact with the material.

8. Conclusions

XRD is a very valuable adjunct to PLM methods for the correct classification of products that may contain asbestos. Confirming doubtful “*not detected*” results in problematic samples by XRD provides greater surety to anyone dealing with asbestos. The usefulness of XRD in asbestos identification has been widely recognised worldwide and should be integrated into all asbestos related work.

9. References

1. AS 4964-2004, Method for the Qualitative Identification of Asbestos in Bulk Samples, (2004)
2. Cambridge Physics: *X-ray Diffraction*, <http://www-outreach.phy.cam.ac.uk>
3. Center for High Pressure Research: *Bragg’s Law and Diffraction: How waves Reveal the Atomic Structure of Crystals*, <http://www.eserc.stonybrook.edu>
4. Dunn H.W.& Stewart J.H Jr., *Determination of Chrysotile in Building Materials by X-ray Diffractometry*, Anal Chem., 54 (7), 1122-1125, 1982
5. NATA Supplementary Requirements for Accreditation in the Field of Chemical Testing (2005)
6. National Occupational Health and Safety Commission: *Code of Practice for the Management and Control of Asbestos in Workplaces (NOHSC:2018 (2005))*, Canberra, April 2005

7. National Occupational Health and Safety Commission: *Guidance Note on the Membrane Filter Method for Estimating Airborne Asbestos Fibres (NOHSC:3003 (2005))*, Canberra, April 2005
8. Sanchez M.S.& Gunter M.E.: *Quantification of Amphibole Content in Expanded Vermiculite Products from Libby, Montana U.S.A Using Powder X-ray Diffraction*, American Mineralogist, Aug 2006, v. 91, no 8-9, p 1448-1451.
9. U.S Environmental Protection Agency: *15USC2642(4)*, June 2006.
10. U.S Environmental Protection Agency: *Amphibole Fiber Concentration Determination for a Series of Community Air Samples: Use of X-ray Diffraction to Supplement Electron Microscope Analysis*, Dec. 2005.
11. U.S Environmental Protection Agency: *EPA-600/M4-82-020*, Dec. 1982
12. U.S Environmental Protection Agency: *EPA/600/R-93/116*, July 1993

INVESTIGATION INTO THE BEHAVIOUR OF ASBESTOS CONTAINING MATERIALS IN BUILDING FIRES

Cameron Hunter

Noel Arnold & Associates

ABSTRACT

Community awareness of asbestos in buildings has raised concerns of health risks due the liberation of asbestos fibre during building fires. Key stakeholders include neighbouring residents, emergency services personnel and government authorities.

In response to stakeholder concerns, the Department of Human Services commissioned Noel Arnold & Associates to conduct an investigation into the effect of fire on asbestos containing building materials. The objectives of the investigation were to:

1. To determine the type and prevalence of dominant materials containing asbestos,
2. To determine the effect of elevated temperatures on the materials with particular regard to whether such temperatures result in:
 - a) the release of respirable fibres,
 - b) the possible modification of such fibres due to heat (denaturing) and
 - c) the effect of heat on the post-fire characteristics of materials (friability)
3. To determine the likely concentrations of respirable fibres in smoke dispersed by the fire to locations adjacent to the fire
4. The investigation utilised the expertise of occupational hygienists together with fire engineering to predict the behaviour of asbestos containing building materials in real fires. Combining with the Centre for Environmental Safety and Risk Engineering (CESARE) at Victoria University, the behaviour of materials was simulated in a scale model fire. Asbestos fibre within the plume and residual ash was then analysed. Computer modelling was then undertaken to predict plume dispersion and the health risks posed to various potentially exposed populations.

Results of the investigation were found to be compelling and valuable in advising stakeholder groups of the risks posed due to fire disturbance of asbestos in buildings.

PREVENTION OF NOISE INDUCED HEARING LOSS – THE NEXT STEP

Kelly Sutherland

Alcoa

ABSTRACT

Noise can affect us physically, psychologically and socially. The most obvious physical effect of noise is hearing loss. Intense noise or long stays in noisy environments will most likely cause hearing damage which cannot be repaired.

Short term exposure to high noise causes a temporary shift in the threshold of hearing. Provided that the noise is not too loud or the exposure too long, normal hearing returns after a period of rest. However, when hair cells close to the oval window (3000-6000Hz frequencies) become worn out from repeated exposure to high noise levels, noise induced hearing loss (NIHL) begins to occur.

The benefits of a holistic approach to hearing conservation management have been well documented. However, an important, and often overlooked part of an effective hearing conservation program is the fitting of hearing protection devices. Evaluation of effectiveness in the past has been a qualitative approach, for both the occupational hygienist and management.

The research in this paper is a summary of the benefits and findings of a quantitative assessment program for insert type hearing protective devices. The study involves a target group of 400+ workers in the mining industry. The report analyses the effectiveness of introducing a quantitative assessment process to an already well established hearing conservation program, to support ongoing prevention of noise induced hearing loss.

COAL TAR PITCH VOLATILE EXPOSURE IN AN ALUMINIUM SMELTER GREEN CARBON FACILITY

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Abstract – Operators and maintenance craftsmen in an aluminium smelter green carbon facility are exposed to coal tar pitch volatiles (CTPV). The aim of this study is to determine if the CTPV exposure presents a health risk to the personnel in the green carbon similar exposure groups (SEGs) and if the controls are appropriate. Personnel volunteered to participate in a personal air sampling and biological monitoring programme to determine occupational exposure to airborne Benzo- α -pyrene (BaP) and Benzene Soluble Material (BSM), including pre- and post-shift urinary excretion of 1-hydroxypyrene (1-OHP). BaP exposure for the operator SEG varied from < 0.5 to $9.7 \mu\text{g}/\text{m}^3$, and for BSM from < 0.02 to $0.98 \text{mg}/\text{m}^3$. BaP exposure for the maintainer SEG varies from < 0.4 to $6.7 \mu\text{g}/\text{m}^3$, and for BSM from < 0.02 to $0.44 \text{mg}/\text{m}^3$. The concentration of urinary 1-OHP for pre- and post-shift samples for the operator SEG varied from none detected to $3.76 \mu\text{mol}$ 1-OHP per mol creatinine, and for the maintainer SEG from none detected to $5.37 \mu\text{mol}$ 1-OHP per mol creatinine. The personal airborne results indicate the BSM and BaP exposures for both the operators and maintenance craftsmen have the potential to be unacceptable, depending upon the tasks undertaken. The operators urinary 1-OHP results were all below the UK Benchmark Guidance Value (BGV) of $4 \mu\text{mol}$ 1-OHP per mol creatinine, whilst there were two urinary 1-OHP results for the maintenance craftsmen that exceeded the BGV. The urinary 1-OHP results indicate that even though the airborne exposure levels have the potential to be unacceptable at times, the controls used during normal operation are effective, with the level of health risk considered acceptable. Maintenance activities have the potential for unacceptable exposure with an increased health risk, hence, the investigation and use of additional controls is required to reduce this risk.

1.0 INTRODUCTION

This study investigated the potential for exposure to Coal Tar Pitch Volatiles (CTPV) within a Green Carbon environment at an aluminium smelter.

The production of the green anodes requires a mixture of crushed used butts, graded petroleum coke (which is sieved and crushed in a continuous ball mill) and hot liquid pitch (160°C) brought together in mixers, which have balanced Local Exhaust Ventilation systems fitted. This semi-solid mixture is fed into the anode block formers, which are vibrated and pressed, forming green anodes. Although the process is automated, employees are frequently exposed to polycyclic aromatic hydrocarbon (PAH)-containing coal tar pitch volatiles, pitch and coke. The green anodes are cooled in a cooling pond for approximately one hour, followed by the baking process in a gas fired baking furnace at 1200°C for 160 hours with a moving fire train. The baked anodes are cooled over ten days, mated with rods, sprayed with aluminium, and are finally suspended within centre-break pre-bake electrolytic cells.

The environmental conditions in the green carbon facility have deteriorated over the last few years and personal exposure monitoring has not been maintained (airborne or biological). Therefore, total exposure to CTPV via inhalation and skin absorption is currently not fully understood or characterised in the Green Carbon Facility. By obtaining current data for CTPV exposure, controls can be investigated and potentially planned for and implemented enabling total CTPV exposure to be reduced to acceptable and insignificant levels.

CTPV inhaled exposure was determined by the collection of airborne contaminants and analysing for the Benzene Soluble Material (BSM) and Benzo- α -pyrene (BaP). Actual CTPV uptake, including inhalation and skin absorption exposure, was estimated by the analysis of 1-hydroxypyrene (1-OHP) in urine.

2.0 METHODOLOGY

2.1 Study Design

Prior to the commencement of this study, the sampling and reporting strategy was discussed with the Green Carbon Management team:

1. Determine current personal CTPV (Benzene Soluble Material – BSM and Benzo(α)pyrene – BaP) exposures.
2. Determine current 1-hydroxypyrene levels in urine of the employees working in Green Carbon, including a control group of employees with no known occupational exposure to CTPV, covering both smokers and non-smokers.
3. Recommend appropriate controls to reduce CTPV exposure for personnel working within the green carbon facility.

2.2 Personal Air Sampling

The air samples were collected using constant flow air sampling pumps (SKC 224-PCXR8), which were calibrated using a Bios DryCal DC-lite calibrator at 2.0 ± 0.1 L/minute prior to sampling. Flow rates were again checked immediately after sampling. Samples were considered invalid if the post-sampling flow rate varied from the pre-sampling flow rate by more than 5%. All calibrations were performed with the filter and sorbent tube in-line. Field blanks were collected on each sampling day. Personal air samples were collected by positioning the filter and tube train in the breathing zone of the employee, while the sampling pump was worn attached to their belt, and collected for at least 70% of the working shift.

Total particulate was collected onto filters, which were 37 mm, 2 μ m pore size, PTFE membrane filters. The filters were loaded into 37 mm 2 section cassettes, with a 37 mm cellulose support pad. The volatile compounds were collected with XAD-2 sorbent tubes (front = 100 mg; back = 50 mg). The sorbent tube was connected to the filter with a minimum length of Tygon tubing, forming a sample train. Immediately after sampling and post calibration, the cassette containing the filter and the sorbent tube (which had been capped) were wrapped in aluminium foil and refrigerated.

BaP was analysed according to NIOSH Method 5506 (NIOSH, 1998), with a reporting limit of 0.5 μ g. The units for BaP are μ g/L. BSM was analysed according to NIOSH Method 5042 (NIOSH 1998a), with a reporting limit of 0.02 mg. The units for BSM are mg/L. The analytical laboratory was Clark Laboratories, a division of Clark Testing Group, Jefferson Hills, PA, U.S.A.

The BSM and BaP exposure results will be compared to agreed Rio Tinto Aluminium (RTA) company occupational exposure limits of 0.2 mg/m³ for BSM and 0.2 μ g/m³ for BaP (Rio Tinto Aluminium, 1997).

2.3 Biological Sampling

During the working week, urine samples were collected from 3 sets of employees prior to each shift commencing and immediately at the completion of each shift:

1. Green Carbon Operators – regular exposure to CPTV;
2. Carbon Maintenance Craftsmen – irregular exposure to CTPV; and
3. Control Group – with no known occupational exposure to CTPV.

Smokers were in both the operators and craftsmen groups, therefore, smokers were also included in the control group.

The urine samples were collected in 30 mL plastic universal containers and refrigerated prior to being sent the Health and Safety Laboratory (HSL) for funded analysis as part of the Health and Safety Executive's (HSE) Disease Reduction Programme (DRP). The DRP is part of HSE's "Fit for work, Fit for life, Fit for tomorrow" Strategic Programme (HSE, 2006). The DRP Cancer project is profiling chemical carcinogens to enable a better understanding of the extent of the risk associated with them. The site used in this study was initially included in a DRP in 2000 to help fulfil a commitment for the Health and Safety Commission's Working Group on the Assessment to Toxic Chemicals (WATCH) programme (WATCH, 2000; Scobbie, *et.al.*, 2000). During 2006-2007, the site was revisited by HSE as part of the DRP to determine if exposure to PAHs had improved, assisting in developing a method of reducing the risk of exposure to chemical carcinogens and reducing cases of work related ill health. As part of the DRP, the HSE funded the 1-Hydroxypyrene in urine analysis at the HSL.

At the HSL, the urine samples were analysed for 1-hydroxypyrene by high performance liquid chromatography (HPLC) with fluorescence detection after enzymatic hydrolysis of conjugates (HSL, 2005; Scobbie, *et.al.*, 2000), using the HSL's standard operating procedure OTOP09). The urine samples were also analysed for creatinine, using the HSL's standard operating procedure SOP23HE. The method's detection limit is 1 nmol/L. The 1-hydroxypyrene results were divided by the concentration of creatinine to correct for urine dilution, taking into account variations due to fluid intake, diet, and insensible losses (e.g. sweating). The units for 1-hydroxypyrene in urine are $\mu\text{mol/mol}$ creatinine (Scobbie, *et.al.*, 2000).

The urinary 1-OHP results were compared to the UK Benchmark Guidance Value (BGV) as an end-of-shift urinary 1-OHP concentration of $4.0 \mu\text{mol/mol}$ creatinine on the basis of the 90th percentile of available biological monitoring results collected from a representative sample of relevant workplaces with good occupational hygiene practices (WATCH 2000; Jongeneelen 2004; ICMM, 2007; HSE, 2002; Unwin, *et.al.*, 2006). The UK BGVs are non-statutory, are not health based but are practical achievable levels (ICMM, 2007; HSE, 2002) which could be considered as the "action level" (Angerer, *et.al.*, 1997).

3.0 RESULTS

3.1 Personal Air Exposures

Detailed Personal Air CTPV Sample Results are summarised below in Tables I and II:

BaP ($\mu\text{g}/\text{m}^3$)								
Job Group	n	AM	GM	Minimum	Maximum	SD	GSD	95%ile
Green Carbon Operator	27	1.18	0.44	< 0.5	9.7	2.31	3.19	3.0
Carbon Maintenance Craftsmen	13	0.90	0.20	< 0.4	6.7	1.80	3.08	2.4
OEL	$0.2 \mu\text{g}/\text{m}^3$							

Table I: Summary of Personal Air CTPV Sample Results – BaP

BSM (mg/m^3)								
Job Group	n	AM	GM	Minimum	Maximum	SD	GSD	95%ile
Green Carbon Operator	27	0.13	0.04	< 0.02	0.98	0.24	4.62	0.50
Carbon Maintenance Craftsmen	13	0.11	0.05	< 0.02	0.44	0.14	4.14	0.50
OEL	$0.2 \text{mg}/\text{m}^3$							

Table II: Summary of Personal Air CTPV Sample Results – BSM

3.2 Biological Monitoring

Urine samples from the Green Carbon Operators, Maintenance Craftsmen and Control Group were collected for the analysis of 1-hydroxypyrene (1-OHP) are summarised below in Tables III and IV:

1-Hydroxypyrene (1-OHP) in Urine								
Job Group	n	AM	GM	Minimum	Maximum	SD	GSD	95%ile
Pre-Shift (With at least 48 hours break from work environment) Samples								
Control Group	15	0.05	0.02	< 0.01	0.26	0.07	4.72	0.25
Green Carbon Operator	9	0.28	0.23	0.07	0.62	0.17	1.97	0.70
Carbon Maintenance Craftsman	15	0.11	0.03	< 0.01	0.35	0.13	6.59	0.71
All Pre-Shift Samples								
Control Group	73	0.13	0.05	< 0.01	1.48	0.21	5.59	0.77
Green Carbon Operator	48	0.72	0.42	< 0.01	2.65	0.63	4.15	4.35
Carbon Maintenance Craftsman	49	0.42	0.17	< 0.01	5.31	0.79	5.23	2.54
UK BGV	4 µmol 1-OHP / mol creatinine							

Table III: Summary of Biological Monitoring - 1-OHP in Urine (Pre-Shift Samples)

1-Hydroxypyrene (1-OHP) in Urine								
Job Group	n	AM	GM	Minimum	Maximum	SD	GSD	95%ile
Post-Shift Samples								
Control Group	73	0.18	0.05	< 0.01	1.97	0.29	6.85	1.14
Green Carbon Operator	47	1.17	0.85	< 0.01	3.76	0.77	2.89	4.87
Carbon Maintenance Craftsman	49	0.72	0.48	0.09	5.37	0.95	2.27	1.86
UK BGV	4 µmol 1-OHP / mol creatinine							

Table IV: Summary of Biological Monitoring - 1-OHP in Urine (Post-Shift Samples)

4.0 DISCUSSION OF RESULTS

4.1 Personal Air Exposures

Each personal air sample was analysed for BSM and BaP, covering particulate and volatile compounds. BSM or BaP levels were not detected within the XAD tube; therefore, they were not present as volatile compounds, only in the particulate form. For all of the valid samples collected, the presence of CTPV was confirmed to be present.

4.1.1 Green Carbon Operator

Each of the Green Carbon Operators rotate through the various activities and tasks within and across the shifts – Former, Analysis and General Duties. The Green Carbon facility is operated from the ground floor control room. The only access to the control room is from outside the green carbon process area. Under normal conditions, the Forming Operator is present within the control room to monitor the Green Carbon process via visual computer based monitoring screens. At times, the operator is required to enter the process area to reset control units near the formers. Sampling and analysis of the production materials is required for quality control. The Sample Laboratory is located on the second floor within the Green Carbon facility. The Analysis Operator collects samples from designated locations within Green Carbon, and analyses them within the laboratory.

The housekeeping within the laboratory is of an acceptable standard. The door leading into the process area is kept closed, resulting in restricting contamination from entering the laboratory area. The General Duties Operator duties include driving forklift trucks moving anodes to the storage yards, reloading crushed material back into the process, fault finding (e.g. dust collector, mixers), housekeeping (including the use of the super sucker vacuum truck) and relieving the Forming Operator. When the Green Carbon Facility is shutdown for scheduled preventative maintenance and cleaning, the Green Carbon Operators undertake the duties of cleaning and housekeeping, including the operation of the super-sucker vacuum truck. The housekeeping standard within the Green Carbon facility is considered poor and is compounded by the number of breakdowns and leaks within the process system. The flooring within the building is a mixture of concrete floors and grid mats, so if a large leak occurs, the dust can be distributed throughout the building across the various levels.

The arithmetic mean (AM) for BSM was calculated as 0.13 mg/m^3 , with a 95th percentile at 0.50 mg/m^3 . The arithmetic mean (AM) for BaP was calculated as $1.18 \text{ } \mu\text{g/m}^3$, with a 95th percentile at $3.0 \text{ } \mu\text{g/m}^3$. Therefore, there is the potential for each of the Green Carbon Operators to have CTPV / PAH airborne exposures that are unacceptable.

4.1.2 Carbon Maintenance Craftsmen

The Carbon Maintenance Craftsmen include fitters, electricians and welders and cover the whole Carbon department (Green Carbon, Bakes, and Anode Supply). There is potential for CPTV / PAH exposure in each of these areas, with exposure within Green Carbon expected to be the highest levels and deemed unacceptable.

The arithmetic mean (AM) for BSM was calculated as 0.11 mg/m^3 , with a 95th percentile at 0.50 mg/m^3 . The arithmetic mean (AM) for BaP was calculated as $0.90 \text{ } \mu\text{g/m}^3$, with a 95th percentile at $2.4 \text{ } \mu\text{g/m}^3$.

Due to the variety of tasks and nature of work undertaken, it is observed that the CTPV / PAH exposure for the mechanical craftsmen (fitters and welder) is higher than that for the electrical craftsmen. Electrical work tends to be more often within control rooms where the main electrical panels are located. Mechanical work tends to be more often associated with process equipment where there is potential for increased work environment contaminant exposure, for example, screw conveyors, bucket elevators, flop gates, mixers, secondary butts equipment. There are increasing opportunities within the Carbon Maintenance crews for multi-discipline functions, where electricians assist fitters within the work environment. Therefore, the potential for unacceptable CTPV / PAH exposure within the Carbon Maintenance area exists across each of the craft disciplines.

4.2 Biological Monitoring

The concentration of 1-OHP in urine for the control group volunteers (employees with no contact to CTPV / PAH containing material), Green Carbon Operators and Carbon Maintenance Craftsmen in the pre and post-shift samples was creatinine corrected.

Both smokers and non-smokers were in each of the sample groups. Caution is required with small sample numbers when comparing the differences between the 1-OHP concentration in urine of smokers and non-smokers. Several authors have found that smokers do not have a statistically significant higher 1-OHP concentration in urine when compared with that for non-smokers.

339 urine samples were collected – 170 pre-shift samples and 169 post-shift samples. The HSL analysed the urine samples for 1-OHP as part of HSE's DRP, which covered the analytical cost.

Only three of the 339 samples were above the UK HSE BGV for 1-OHP in urine of 4 µmol/mol creatinine. This indicates that the CTPV exposure is generally well in control during normal operating conditions, except during housekeeping and maintenance activities.

4.2.1 Pre-Shift Samples

The pre-shift samples were broken into two groups:

1. Pre-shift samples where there was at least 48 hour break away from the work environment; and
2. All pre-shift samples.

The arithmetic mean and 95th percentile for the identified groups pre-shift samples following the break away from the work environment were all well below the UK BGV for 1-OHP in urine of 4 µmol/mol creatinine. The exposed groups of Green Carbon Operators and Carbon Maintenance Craftsmen have a slightly higher pre-shift level of 1-OHP in urine when compared with the Control Group. This may be due to a slowly excreting component with a half-life of 16 days (Buchet, *et.al.*, 1992).

For the exposed groups, only one sample had a pre-shift 1-OHP concentration in urine above the UK BGV of 4 µmol/mol creatinine at 5.31 µmol/mol creatinine. This was from a Carbon Maintenance Craftsman (number 3) who was a fitter who undertook preventative maintenance on the green carbon former over two days. Unfortunately, the pre-shift sample for the fourth sample day was not provided, as it would have been interesting to see if the 1-OHP concentration in urine was also high following the second day of preventative maintenance in green carbon area.

The higher pre-shift 1-OHP in urine result can be explained as the half-life for the urinary excretion of 1-OHP in occupationally exposed workers is known to vary from 6 to 35 hours from the time of CTPV exposure (Jongeneelen, *et.al.*, 1990; Brandt, *et.al.*, 2003; Health Council of the Netherlands, 2006; Buchet, *et.al.*, 1992).

4.2.2 Post-Shift Samples

The only samples with a 1-OHP concentration (4.03 and 5.37 µmol/mol creatinine) in urine above the UK BGV of 4 µmol/mol creatinine was from the same Carbon Maintenance Craftsman (number 3) who was a fitter who undertook preventative maintenance on the green carbon former over two days. As the plant is shutdown during preventative maintenance activities limiting the concentration of airborne CTPV / PAH, the craftsman's exposure may be mainly dermal.

Due to variation in 1-OHP in urine sample results for the Green Carbon Operator, there is potential for this SEG to have employees with 1-OHP levels in their urine to be above the UK BGV of 4 µmol/mol creatinine.

PAH in the diet, environment and smoking may contribute to the change in urinary 1-OHP for the non-occupationally CTPV / PAH exposed control group.

4.3 Control of CTPV Exposure

In general, the control of CTPV / PAH exposure is the use of PPE – clothing, gloves, and respirators. This method of control has been chosen as the only practicable form of control given the nature of the tasks and process. Eating, drinking and smoking are prohibited within the Green Carbon facility.

COSHH (HSE, 2002) requires that where it is not reasonably practicable to prevent exposure to a carcinogen, the employer is required to implement the following measures in addition to those previously indicated:

- totally enclose the process and handling systems unless this is not reasonably practicable;
- the prohibition of eating, drinking and smoking in areas that may be contaminated by carcinogens;
- cleaning of floors, walls and other surfaces at regular intervals and whenever necessary;
- designating those areas and installations which may be contaminated by carcinogens and using suitable and sufficient warning signs; and
- storing, handling and disposing of carcinogens safely, including the use of closed and clearly labelled containers.

Various controls are currently in place to reduce CTPV exposure of the Green Carbon Operators and Carbon Maintenance Craftsmen:

- The company provides personal protective clothing – trousers, shirts, jackets, long sleeved vests, and disposable Tyvek coveralls (also known on the site as “catsuits”), and gloves;
- The company provides a contractor laundering service for the trousers, shirts and jackets, which prevents CTPV / PAH soiled clothing being taken home;
- Effective respiratory protection equipment programme, which provides various models and sizes of respirator (including half and full face negative pressure respirators, and power assisted positive pressure respirators fitted with organic and particulate filters); annual quantitative fit testing for negative pressure respirators; education and training; maintenance, cleaning and storage requirements);
- Provision of and encouragement to use water based barrier creams;
- No smoking, eating or drinking within the CTPV contaminated areas;
- Signage erected at the entry points of the Green Carbon facility indicating the required personal protective equipment;
- Documented work procedures known as Best Current Practices (BCPs);
- CTPV awareness presentations to personnel working within the Green Carbon facility (including the definition of CTPV / PAH, exposure pathways, effects of exposure, potential health issues – skin, bladder and lung cancer, exposure standards, personal protective equipment, barrier creams, washing and showering, personal hygiene, sunscreen);
- Washing of hands prior to eating, smoking or use of the toilet;
- Showering at the end of the shift and if necessary, during the shift following dirty tasks with high CTPV dermal exposure;
- Clean and dirty sides of the change rooms in the Carbon amenity block;
- Encouragement of regular fluid intake which not only hydrates employees, but ensures regular flushing of the bladder;
- Provision of and encouragement to use UV protection creams on all skin surfaces that may have been contaminated with CTPV prior to being exposed to sunlight;
- Annual health surveillance including spirometry / lung functions, skin examination for unusual growths or changes in spots, and urinalysis;
- Regular biological monitoring (urinary 1-OHP);
- Regular personal and static airborne exposure monitoring (BSM and BaP).

5.0 CONCLUSION AND RECOMMENDATIONS

The Green Carbon work environment CTPV levels in the air can exceed the BSM and BaP OELs. The biological monitoring of urinary 1-OHP indicates that the current control measures reduce the body burden to below the UK BGV of 4 $\mu\text{mol/mol}$ creatinine during the operation of the Green Carbon facility, except during some maintenance activities. The use of PPE, such as gloves, coveralls and respirators and administrative measures, are the main controls used to reduce personal CTPV exposure levels. The employees working in the Green Carbon facility, operators and

maintenance craftsmen, have good understanding of the hazards and control measures required to reduce their CTPV exposure. Additional use of controls, such as improved extraction systems, improved housekeeping and cleaning regimes, is required to reduce the CTPV exposure to the lowest practical levels.

The following are recommended to reduce and manage CTPV exposure for personnel working with the Green Carbon facility:

- Improving the extraction systems (i.e. ensuring the scheduled preventative maintenance is undertaken; balancing of the air flow; cleaning of the ductwork which will reduce the amount of CTPV build up);
- Continue the LEV testing, using an accredited contractor, at least every 14 months, in accordance with the COSHH regulations;
- Removal of carpet tiles in the control room and laying of vinyl tiles, aiding in floor cleaning and reducing the amount of residual CTPV in the environment;
- Preventative maintenance programme for the control room and analysis laboratory air conditioners, including regular filter changes;
- Installation of a hand washing station located outside the control room;
- Availability of barrier creams and dispensers within the control room;
- Availability of UV protection creams in the control room and amenity blocks;
- Inclusion of barrier and UV protection creams as stores stock items, increasing the ready available across the shifts and crews, instead of just being available from the Occupational Health Centre;
- Review of gloves and disposable coveralls (various gloves and coveralls are currently being trialled);
- Improved housekeeping and cleaning standards;
- 100% attendance at the Occupational Health Centre for annual health surveillance;
- 100% participation in biological monitoring programmes (i.e. 1-OHP in urine);
- Regularly scheduled personal and static air exposure monitoring of BSM and BaP;
- Documented attendance at annual CTPV awareness presentation session; and
- Regular review of documented COSHH risk assessments associated with Green Carbon operations and maintenance activities.

Primary considerations should be given to reducing CTPV exposure by engineering controls and modification of work practices. While personal protective equipment and administrative controls are necessary to provide reduced exposure to employees while other controls are being investigated and implemented, it is recognised that these are not acceptable long term solutions.

It is also important for each employee to maintain a high level of personal hygiene whilst working in a CTPV contaminated environment.

There is a significant role for biological monitoring of exposure to CTPV when used in conjunction with airborne contaminant monitoring. The two types of monitoring complement each other, covering CTPV routes of exposure – ingestion, dermal and inhalation. The use of biological monitoring can also be used to assess the efficiency of a respiratory protection programme.

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REFERENCES

- Angerer, J., Mannschreck, C. and Gündel, J., *Occupational Exposure to Polycyclic Aromatic Hydrocarbons in a Graphite-Electrode Producing Plant: Biological Monitoring of 1-Hydroxypyrene and Monohydroxylated Metabolites of Phenanthrene* 1997, *Int Arch Occup Environ Health*, 69:323-331.
- Buchet, J.P., Gennart, J.P., Mercado-Calderon, F., Devavignette, J.P., Cupers, L. and Lauwerys, R., *Evaluation of Exposure to Polycyclic Aromatic Hydrocarbons in a Coke Production and a Graphite Electrode Manufacturing Plant: Assessment of Urinary Excretion of 1-Hydroxypyrene as a Biological Indicator of Exposure* 1992, *British Journal of Industrial Medicine*, 49:761-768.
- Health and Safety Executive (HSE), *Disease Reduction Programme (DRP)*, 2006, (<http://www.hse.gov.uk/drp/index.htm>).
- Health and Safety Executive (HSE), *Occupational Exposure Limits 2002 (EH40/2002)* 2002, HSE Books, Sudbury, UK.
- Health and Safety Executive (HSE), *The Control of Substances Hazardous to Health (COSHH), Statutory Instrument 2002 No. 2677*, 2002, ISBN 0 11 042919 2, The Stationery Office Limited.
- Health and Safety Laboratory (HSL), *Guidance on Laboratory Techniques in Occupational Medicine*, 2005, 10th Edition, Health and Safety Laboratory, an agency of the Health and Safety Executive, UK.
- International Council on Mining and Metals (ICMM), *The Setting and Use of Occupational Exposure Limits: Current Practice* 2007, ICMM, London, UK.
- Jongeneelen, F.J., *Guidelines for Biological Monitoring of Workers in Aluminium Production Facilities for Urinary 1-Hydroxypyrene (1-Pyrenol)*, *J Environ Monit* 2004, 6: 61-65.
- Jongeneelen, F.J., van Leeuwen, F.E., Oosterink, S., Anzion, R.B.M., vanderLoop, F., Bos, .P. and VanVeen, H.G., *Ambient and Biological Monitoring of Cokeoven Workers, determinants of the Internal Dose of PAH*, *British Journal of Industrial Medicine* 1990, 47:454-461.
- NIOSH Manual of Analytical Methods (NMAM), *Method 5042 – Benzene-Soluble Fraction and Total Particulate (Asphalt Fume)*, 4th Edition, Issue 1, 15 January 1998.
- NIOSH Manual of Analytical Methods (NMAM), *Method 5506 – Polynuclear Aromatic Hydrocarbons by HPLC*, 4th Edition, Issue 3, 15 January 1998a.
- Rio Tinto Aluminium, *Rio Tinto Aluminium Smelting Standard for Coal Tar Pitch Volatile (CTPV) Risk Management*, Draft 8, 2007.
- Scobbie, E., Cocker, J., Whitely, S., Guiver, R. and Chambers, H., *Survey of occupational exposure to polycyclic aromatic hydrocarbons*, HSL Report No. JS2000329, Sheffield, HSL, 2000.
- Unwin, J., Cocker, J., Scobbie, E. and Chambers, H., *An Assessment of Occupational Exposure to Polycyclic Aromatic Hydrocarbons in the UK* 2006, *Ann. Occup. Hyg.*, 50:4:392-403.
- WATCH, WATCH-subcommittee of Health and Safety Commission of UK, *CTPV and PAH*, Paper for meeting at HSE, London, Watch/21/2000, May 2000.

FARMERS AND PPE: WHAT FACTORS PREDICT SAFETY BEHAVIOURS?

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Objectives: To investigate patterns of use of personal protective equipment (PPE) to reduce pesticide exposure amongst a sample of Australian farmers and to assess associations between PPE use and possible predictive factors.

Methods: A cross-sectional survey of 1 102 farmers recruited through the Victorian Farmers Federation (VFF) was conducted. The written questionnaire was filled out by participants at VFF meetings attended by a visiting research assistant. Non-attending members were recruited by farm visits. Participants answered questions about frequency of pesticide use and PPE items they usually used when doing two different pesticide-related tasks, mixing and application, of each of four classes of pesticides. They also answered questions about personal characteristics, farm characteristics, farming activities, career and health.

Results: Nearly all surveyed farmers had ever used pesticides, and over 87% had used Herbicides or Animal Health Products in the previous 12 months. Non-use of PPE was frequently reported, with up to 10-40% of farmers routinely using no PPE at all when using pesticides. Across all pesticide classes, PPE was more commonly reported for pesticide mixing than for application. In multivariate analyses PPE use appeared to be most strongly and consistently associated with younger age and farm chemical training.

Conclusions: PPE use across all pesticide classes was poor, indicating the possibility of clinically significant pesticide exposure in many farmers. Given that PPE use was found to be associated with farm chemical training, training is likely to be an important intervention for reducing farmers' pesticide exposure. Poor uptake of farm chemical training by farmers and the aging farming workforce are causes for concern in the light of these findings.

Q FEVER IN A SECONDARY MEAT PROCESSING WORKPLACE

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Abstract

Four employees at a Victorian workplace contracted Q fever as a result of processing semi-thawed ovine placentas. The employer had not identified Q fever as a hazard, and potential risk, and hence had not implemented appropriate controls. Once the risk was identified the employer implemented controls, including vaccinations, to prevent future cases of employees contracting Q fever. WorkSafe Victoria successfully prosecuted the employer with a resulting fine of \$20,000 plus costs.

Introduction

WorkSafe Inspectors visit workplaces across Victoria to address a range of occupational health and safety issues and to review and enforce compliance with legislation. The Occupational Hygiene Unit of WorkSafe routinely provides advice and assistance on occupational hygiene issues to WorkSafe inspectors across the State. In 2005 the Occupational Hygiene Unit was contacted by an Inspector requesting assistance in relation to a secondary meat processing workplace in Geelong where 4 employees had contracted Q fever. The workplace processed raw meat at its premises for use in food and cosmetics – this included ovine (sheep) placentas.

Q fever

Q fever is a bacterial infection caused by *Coxiella burnetii*. The organism is highly contagious and mostly concentrated in birth products such as amniotic fluid, placenta and uterus of an infected animal. Animals that may be infected include goats, cattle, sheep, other farm and domestic animals and some wild animals (including kangaroos, bandicoots and feral pigs), however it is most commonly associated with cattle outbreaks in abattoirs. It is an infectious disease contracted through the respiratory route after inhalation of the contaminated dust or aerosol. The incubation period is usually 19 – 21 days but can range from 2 weeks to 2 months. Person to person transmission is rare.

The onset of Q fever is usually acute and characterized by fever, chills, sweats, severe headache, weakness, anorexia, myalgia and cough. Abnormal liver function tests are common. It is similar in severity to a severe case of the flu. Chronic Q fever is uncommon – it occurs in about 1% of people who suffer from Q fever and may include granulomatous, hepatitis and endocarditis. The latter is a more serious concern as it occurs months to years after the acute illness.

There is a Q fever vaccine; immunisation is a primary prevention measure for those at risk.

Discussion

In May 2005 the Occupational Hygiene Unit of WorkSafe visited a Geelong secondary meat processing workplace to observe and review the systems of work at the workplace in order to assist in the prevention of any future Q fever outbreaks.

During the visit it was established that the workplace processed frozen ovine placentas into powdered form by mincing, cooking, drying and then grinding them. Processing of ovine placentas was a small part of the workplaces operations but had been performed for a few years.

In late 2004 early 2005 one of the workplaces clients requested that the foetus be separated from the placenta – the workplace had not previously performed this type of separation. In February 2005 the workplace undertook a trial of separating the foetus from the placenta. The ovine placentas were 'semi-thawed' in a bath overnight, the next morning the placenta and its components were

separated. A knife was used to cut open the placenta bag and to separate the foetus, the umbilical cord and the amniotic fluid into separate tubs. Handling the placentas in this form has the potential to generate airborne mist. Once separated the components were refrozen until enough of each (e.g. enough foetuses) was available to justify starting their processing. Their processing involved mincing them (in a frozen state), then cooking, drying and grinding them into a powder form.

This trial was the first time, for this workplace, that the ovine placentas were handled in a semi-thawed state and the components separated. Also around this time, some of the placentas used by the workplace were sourced from South Australia, where Q fever is known to exist.

Prior to commencing work with the ovine placentas, particularly the semi-thawed placentas, the workplace had not identified that Q fever was a hazard in processing the placentas and hence that there was a potential risk of employees contracting Q fever.

Information, from suppliers of the placentas, identifying hazards (such as Q fever), potential risks and recommended health and safety measures was not provided or requested.

There was no formal hazard identification or risk assessment conducted prior to commencing the process, particularly in the semi-thawed state. There appeared to be, naively, reliance on a quality certification of the meat product.

Employees handled the ovine placentas and their components in the open, the process was not enclosed or under localised extraction at the point where airborne dust or mist was likely to be generated. Personal protective clothing in the form of a half face disposable respirator was made 'available' to employees involved in the trial but its use was not enforced. Gloves, aprons and gum boots were standard protective clothing for employees processing the placentas. Employees were also advised to maintain good personal hygiene (e.g. washing hands when leaving the process room). Vaccination for Q fever was not made available to employees as management was not aware of the risk.

Information on the potential risk of contracting Q fever when handling placentas should have been provided to the workplace from the supplier of the placentas. Information on the potential risk of contracting Q fever when handling placentas should have been obtained by the workplace. This would have enabled:

- appropriate information to be provided to employees; and
- appropriate controls to be implemented, for example:
 - making vaccinations available to employees at risk;
 - enclosing the process;
 - implementing engineering controls to capture dust and mist generated from the process;
 - handling the product in the frozen, state not semi-thawed;
 - using methods that do not generate airborne mist or dust; and
 - using respiratory protective equipment to minimise the risk.

Conclusion

The potential risk of employees contracting Q fever was present from the time the workplace commenced processing ovine placentas, however as the placentas were in a frozen state the risk was low. Once the placentas were handled in a semi-thawed state the potential for inhalation of airborne mist increased and hence the risk increased.

An inadequate investigation into the hazards and potential risks associated with processing ovine placentas resulted in inappropriate controls for the handling of semi-thawed placentas which was the likely cause of the four employees contracting Q fever

WorkSafe Victoria issued proceedings against the employer under the Occupational Health and Safety Act 2004 for failing, so far as reasonably practicable, to provide and maintain for employees of the employer a working environment that is safe and without risks to health in relation to

- the provision or maintenance of plant or systems of work that are, so far as is reasonably practicable, safe and without risks to health; and
- the provision of such information, instruction, training or supervision to employees of the employer as is necessary to enable those persons to perform their work in a way that is safe and without risks to health.

The matters were successfully prosecuted. The employer pleaded guilty to the charges resulting in the company being fined \$20,000 plus costs.

After the prosecution WorkSafe Victoria sent a letter to 225 organisations in the meat, smallgoods and poultry manufacturing industries raising their awareness of the incident and to outline their duties as manufacturers of meat and animal products to supply adequate health and safety information about the products supplied and safety measures necessary to ensure the products do not cause a risk to health to those who transport, store or handle the product.

SPECIFICATION AND ASSESSMENT OF HIGH QUALITY INDOOR ENVIRONMENTS FOR SUSTAINABLE OFFICE BUILDINGS

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ABSTRACT

The quality of office indoor environments is considered to consist of those factors that impact occupants according to their health and well-being and (by consequence) their productivity. Indoor Environment Quality (IEQ) can be characterized by four indicators:

- Indoor air quality indicators
- Thermal comfort indicators
- Lighting indicators
- Noise indicators.

Within each indicator, there are specific metrics that can be utilized in determining an acceptable quality of an indoor environment based on existing knowledge and best practice. Examples of these metrics are: indoor air levels of pollutants or odorants; operative temperature and its control; radiant asymmetry; task lighting; glare; ambient noise. The way in which these metrics impact occupants is not fully understood, especially when multiple metrics may interact. While the potential cost of lost productivity from poor IEQ has been estimated to much exceed building operation costs, the level of impact and the relative significance of the above four indicators are largely unknown. However, they are key factors in the sustainable operation or refurbishment of office buildings.

This paper presents a methodology for assessing indoor environment quality (IEQ) in office buildings, and indicators with related metrics for high performance and occupant comfort. These are intended for integration into the specification of sustainable office buildings as key factors to ensure a high degree of occupant habitability, without this being impaired by other sustainability factors.

The assessment methodology was applied in a case study on IEQ in Australia's first 'six star' sustainable office building, Council House 2 (CH2), located in the centre of Melbourne. The CH2 building was designed and built with specific focus on sustainability and the provision of a high quality indoor environment for occupants. Actual IEQ performance was assessed in this study by field assessment after construction and occupancy. For comparison, the methodology was applied to a 30 year old conventional building adjacent to CH2 which housed the same or similar occupants and activities. The impact of IEQ on occupant productivity will be reported in a separate future paper.

Keywords: office, air, thermal comfort, noise, lighting

1. BACKGROUND

The CRC for Construction Innovation initiated a project "Regenerating Construction to Enhance Sustainability" in 2005, with the overall objective to 're-life' an office building to an "A Grade" standard using cost effective practices for ecologically sustainable design and best-practice technologies. The expected outcome was the delivery of superior refurbished **office** buildings according to a core set of four sustainability criteria:

- Eco-efficiency: minimising ecological footprint
- High indoor environment quality (IEQ): demonstrable improvement in key IEQ criteria, including thermal performance and indoor air quality
- Healthier and more productive working environment: as measured by the performance of

- occupants determined before and after refurbishment and
- Waste minimisation.

This report considers only the core criterion **high indoor environment quality**. A previous report (Brown 2006) reviewed available knowledge on IEQ in offices and developed a research plan for this project as follows:

- Identify and define **key indicators** for high quality indoor environments
- Specify **sampling and measurement protocols** for performance measures (metrics) of key IEQ indicators
- Specify reliable, **scientific procedures** by which performance measures can be evaluated
- Recommend **performance criteria** for each metric
- Consider **design and specification implications** of performance targets
- Document the application of the indicators in a **target building before and after refurbishment**.

These are presented here with the results from the application to a target building in Melbourne.

2. IEQ INDICATORS

IEQ indicators (Brown and Kivlighon 2005) were considered to be encompassed in the following indicators:

- indoor air pollutant levels
- thermal comfort
- lighting, and
- noise.

Building ventilation rate will have a significant impact if uncontrolled, but historically this has been tightly regulated in the Building Code of Australia (BCA) and was not *directly* included as an indicator assuming that ventilation performance was generally optimised. A key consideration in selecting the indicators was that they could be represented by performance metrics relevant to their potential impacts on occupant satisfaction and acceptance of office environments.

2.1 Indoor Air Quality Indicators

Poor indoor air quality (IAQ) can be a significant health, environment and economic problem, and has become a public health issue and liability for employers and building managers who fail to provide a 'safe' working environment. IAQ measures must determine how well indoor air (a) satisfies thermal and respiratory requirements of occupants, (b) prevents unhealthy accumulation of pollutants, and (c) allows for a sense of well-being. International research has established the occurrence of a range of building-related illnesses, many with identifiable and diverse causes. A subset of these illnesses - termed the 'sick building syndrome' (SBS) - includes mainly subjective symptoms (mild irritation of eyes, nose and throat, headaches, lethargy). SBS symptoms are believed to arise from multiple causes which, while not clearly understood, are associated mainly with air-conditioned office buildings. Australian studies have been limited, but indicate similar occurrence to other developed countries for building-related illnesses, SBS-like symptoms and dissatisfaction with office air environments (Brown 1997, 2005).

Regulatory actions related to *indoor* air quality in Australia are limited, especially in comparison to outdoor air quality and industrial workplace air, a situation also common overseas. Some guidance has been provided by the NHMRC (formerly recommended health-based advisory IAQ goals), the enHealth Council, the Australian Safety & Compensation Council (ASCC, occupational exposure standards), National Industrial Chemicals Notification & Assessment Scheme (NICNAS) and the World Health Organization (WHO, health-based environmental air quality guidelines for Europe). Some pollutants have been investigated in Australian buildings in detail, but for others few observations are available. Based on this limited data and international research, the most

appropriate strategies are to reduce exposures to health-based criteria by reducing pollutant sources, controlling moisture, and ventilating to current standards (Brown 2005). Based on this background, key metrics for IAQ *in offices* are recommended in Table 1. Note that an order of priority was assigned to each, according to the level of quality of indoor air that is likely to be achieved by their application in an office building where members of the public and children may have access.

Table 1. Key indicators for indoor air quality

Indoor air pollutant	Possible sources	IAQ criterion (averaging period)	Priority
Formaldehyde	Partitions, furniture, shelving, flooring	100 µg/m ³ (peak)	High
Total VOC (TVOC)	Building materials, furniture, office equipment	500 µg/m ³ (1 h)	High
VOC: benzene	As for TVOC, auto exhausts	10 µg/m ³ (1 y)	High
VOC: toluene	“	4100 µg/m ³ (24 h)	High
VOC: xylene isomers	“	1200 µg/m ³ (24 h)	Low
PM2.5	Auto exhausts	25 µg/m ³ (24 h)	High
Carbon monoxide	Auto exhausts	9 ppm (8 h)	High
Carbon dioxide	Exhaled breath	800ppm (1h)	High
Ozone: at equipment exhausts	Copiers, printers	0.1 ppm	Low
Micro-organisms	Persistently damp surfaces, mechanical ventilation system	Absent on inspection	High
Asbestos	Insulation, sheeting, flooring	Inspection + risk evaluation	Low-Medium

2.2 Thermal Comfort Indicators

Thermal comfort is commonly defined as that ‘condition of mind which expresses satisfaction with the thermal environment’ (ISO 1994). Since people vary greatly in physiological and psychological factors, it is accepted that it is impossible to satisfy the thermal comfort of all occupants. However, based on existing data it is possible to statistically define conditions that a specified proportion of office occupants will find thermally comfortable. As well as physical parameters - air temperature, radiant temperature, air speed, humidity - a person’s activity levels and the insulation received from clothing will also influence thermal comfort but these are generally specified at default values which are typical levels for office environments.

A significant factor to thermal comfort is whether a space is *mechanically* conditioned or *naturally* conditioned – these are known to require different conditions for thermal comfort since occupant expectations in the latter are shifted due to different thermal experiences and availability of individual control. Only mechanically conditioned offices will be considered here (see ASHRAE (2004) for guidance on naturally conditioned spaces). For given values of humidity and air speed, the thermal comfort zone can be defined in terms of operative temperature or in terms of combinations of air temperature and mean radiant temperature (ASHRAE 2004), defined as follows:

- operative temperature: the uniform temperature of an imaginary black enclosure in which an occupant would exchange the same amount of heat by radiation plus convection as in the actual non-uniform environment. In most practical cases, this can be calculated as the mean of the air temperature and the mean radiant temperature. Also, in the absence of radiant heating/cooling panels, heat generating equipment, envelope insulation and large window solar heat gain, the assumption that operative temperature equals air temperature is acceptable;
- air temperature: the temperature of air surrounding the occupant;
- mean radiant temperature: the uniform surface temperature of an imaginary black enclosure in which an occupant would exchange the same amount of radiant heat as in the actual non-uniform space.

The operative air temperature for buildings recommended by ISO (1994) was $22^{\circ}\text{C} \pm 2^{\circ}\text{C}$ for winter conditions and $24.5^{\circ}\text{C} \pm 1.5^{\circ}\text{C}$ for summer conditions, and these values were endorsed by the Australian Government (1995). ASHRAE (2004) specified operative air temperature according to two equivalent procedures: a simplified graphical method or a computer program based on a heat balance model; only the former will be presented. The graphical method may be applied to spaces where the occupants have activity levels between 1.0-1.3 met, where clothing provides 0.5 – 1.0 clo of thermal insulation, and air speeds are not greater than 0.2 m/s, *conditions that occur in most office spaces*. The range of operative temperatures presented in Figure 6 are for 80% occupant acceptability (based on 10% dissatisfaction for whole body- and 10% for partial body-comfort). Note that the thermal comfort zone extends across an operative T from 19°C to 28°C , the specific operative temperature depending on clothing, activity and humidity levels. The former are set at default values of 1.2 met activity and 0.5 clo (summer) /1.0 clo (winter) as specified in ISO (2005).

Other thermal comfort metrics are:

- Relative humidity (RH) which can lead to skin, eye and respiratory irritation (ASHRAE 1992) if too high or too low. ISO (2005) recommends that the relative humidity should be between 30% to 70% for summer and winter conditions. RH above approximately 70-80% can cause microbial growth and damage to surfaces within buildings, due to an increased likelihood of persistent surface condensation
- Air velocity should be within the range 0.05 – 0.2 m/s. ASHRAE (2004) specifies that air speed may be increased above 0.2 m/s to increase the maximum temperature for acceptability *if occupants are able to control the air speed*. The amount of increase is limited to 3°C with air speed to not exceed 0.8 m/s.

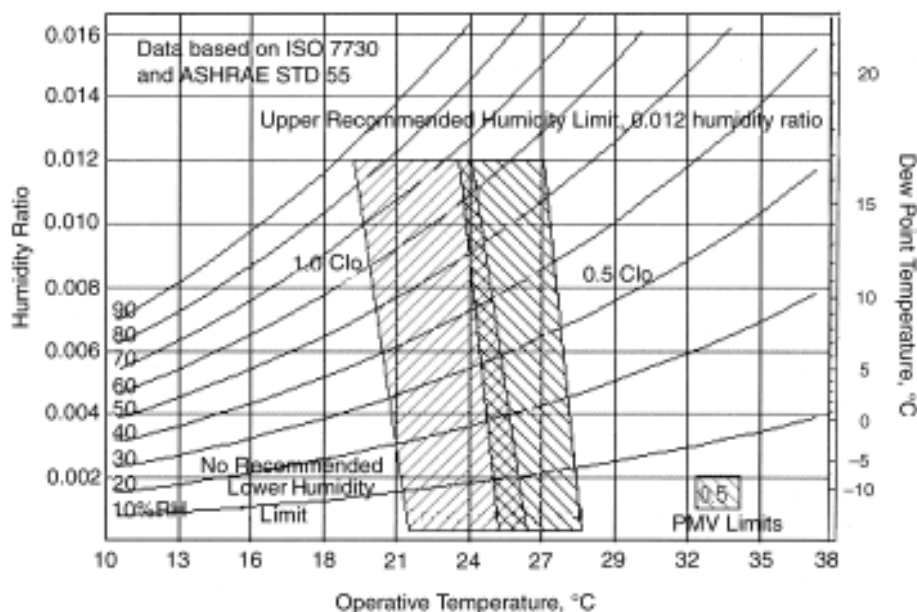


Figure 6. Acceptable ranges for operative temperature and humidity in ‘typical’ office spaces (ASHRAE 2004)

- Vertical and horizontal radiant temperature asymmetry, specified by ASHRAE (2004) as warm ceiling < 5°C, cool wall < 10°C, cool ceiling < 14°C, and warm wall < 23°C.

Currently, ASHRAE (2004) and ISO (2005) specify three performance levels for thermal comfort by these metrics, as presented in Tables 2 and 3, since in practice the levels attained will depend on technical, cost, environment and energy considerations.

Table 2. Three categories of thermal environment

Thermal state of the body as a whole				Local thermal discomfort (%PD caused by)		
Category	PPD %	Predicted mean vote (PMV)	Draught rate, DR %	Vertical air temperature difference %	Warm or cool floor %	Radiant temperature asymmetry %
A	< 6	- 0.2 < PMV < + 0.2	< 10	< 3	< 10	< 5
B	< 10	- 0.5 < PMV < + 0.5	< 20	< 5	< 10	< 5
C	< 15	+ 0.7 < PMV < + 0.7	< 30	< 10	< 15	< 10

Table 3. Criteria for operative temperature for typical buildings (Olesen 2004)

Type of building	Clothing (clo)		Activity (met)	Category	Operative Temperature (°C)	
	Summer	Winter			Summer	Winter
Office	0.5	1.0	1.2	A	24.5 ± 0.5	22.0 ± 1.0
				B	24.5 ± 1.5	22.0 ± 2.0
				C	24.5 ± 2.5	22.0 ± 3.0
Cafeteria / Restaurant	0.5	1.0	1.4	A	23.5 ± 1.0	20.0 ± 1.0
				B	23.5 ± 2.0	20.0 ± 2.5
				C	23.5 ± 2.5	20.0 ± 3.5
Department Store	0.5	1.0	1.6	A	23.0 ± 1.0	19.0 ± 1.5
				B	23.0 ± 2.0	19.0 ± 3.0
				C	23.0 ± 3.0	20.0 ± 4.0

Note in Table 3 that while the *mean* operative temperatures are the same for the different categories, the *allowable spread* of operative temperatures changes markedly across categories. Also, comparing these to earlier specification by ISO (1994) - 22°C ± 2°C for winter and 24.5°C ± 1.5°C for summer – it is seen that extra performance levels have been introduced above and below the earlier specification purely based on *operative temperature control*.

2.3 Lighting Quality Indicators

Lighting levels need to be of a quality that provides an environment in which it is easy to see so that office tasks can be safely performed without eye strain. During typical working hours, lighting inside offices tends to rely on a combination of both daylight from windows and electric lighting. There is little doubt that people prefer to work by daylight and enjoy a view outdoors. Also, this mixture of lighting enables a degree of flexibility which is a useful outcome. Windows can assist in avoiding or reducing eyestrain by allowing an individual to focus on distant objects rather than prolonged viewing of close objects such as computer screens. However, the use of windows needs to be balanced with respect to any adverse thermal effects or unwanted lighting effects such as glare.

Even though a task may be three dimensional, it is generally carried out in more or less one plane and it is common to provide illuminance on that plane (called the 'working plane'). Note that achieving illuminance on working planes will facilitate the task visibility but does not necessarily achieve the desired visual appearance or comfort of a space. In general, there are three key factors to task illuminance:

1. increasing the illuminance on a task produces an increase in performance following a law of diminishing returns
2. the illuminance at which performance levels off is determined by the visual difficulty of the task (the smaller or the lower contrast in a task, the higher the illuminance level)
3. it is not possible to bring a difficult visual task to the same level of performance as an easy task simply by increasing the illuminance (e.g. consider the improvement from using a magnifier for tasks difficult to the unaided eye).

The standard international unit that is used to measure the amount of light per unit of surface area, also known as illuminance, is lux (symbolized lx). Australian Standards for interior lighting for office and screen based tasks recommend a *minimum* of 160 lx on the working plane so that eyes are not strained due to a deficiency of light. Also, the uniformity of illuminance within a room should not be less than 0.7 (i.e. the minimum illuminance on a given plane should not be less than 70% of the average illuminance). Good task visibility depends on both the luminance of the task and its surroundings. The optimum levels recommended for the ratio of the luminances of task: immediate surrounds: general surrounds are approximately 10:3:1. Also, the average *initial* illuminances for office-based tasks provided by a lighting system will need to be significantly higher than the **recommended maintenance illuminance** in order to allow for the progressive loss of light due to lamp ageing and dust accumulation.

Standards Australia (1994) specifies recommended values for maintenance illuminance according to specific tasks and room types, from 160 lx for keyboards, 320 lx for reading/writing/typing, to 600 lx for tasks with fine details (e.g. draughting). Other key lighting metrics for IEQ are Glare, Colour temperature, and Flicker from electric luminaires (Brown 2006).

2.4 Noise Indicators

Sound level is defined in terms of the unit decibel (A) which is measured at the frequencies over which humans generally hear, 20 to 20 kHz, using an 'A' filter. Equivalent continuous A-weighted sound pressure level ($L_{Aeq,T}$) is a term that is used to indicate the sound level over a defined number of hours. For sound that is encountered during working hours, usually an 8 hour day, the continuous A-weighted sound pressure level is denoted by $L_{Aeq,8h}$. Background sound tends to be of a low intensity and is present for most of the time in any environment. Sources of background sound in an office include: computers, lights and ventilation systems. Excessive amounts of background sound can cause stress which can impede upon an individual's ability to work well. The UK's Sustainable Development Unit recommended that separate rooms/offices should have an $L_{Aeq,8h}$ value of less than 40 dBA and an open plan office less than 45 dB(A) (UK Government,

1999). Standards Australia (2000) recommended levels for different occupancies as a range from satisfactory noise level (for most) to maximum (unsatisfactory for most). These were to be measured with the building *unoccupied* but ready for occupancy, and varied from 35-40 dbA (private offices), 40-45 dbA (general offices), 45-50 dbA (computer rooms).

Impact sound is of a high intensity but lasts for only a short amount of time. Impact noise within in an office can come from sources such as electric staplers or doors slamming. High intensity impact noise can damage hearing, but it is considered highly unlikely to occur within an office environment, and so the averaged 8 hour noise level is considered to be the appropriate metric. Also, sound from short-term sources, such as printers and photocopiers, can be readily minimised by locating them in a separate room.

2.5 Occupant Questionnaire on Environmental Comfort

While the above air and physical metrics aim to assess the key indicators of IEQ, it is considered that the complexity of IEQ and the environment-occupant interaction are such that a direct feedback of occupant experience must also be part of IEQ assessment. Applied to a statistically significant but random sample of occupants (approx. 30), this can provide a direct measure of the comfort levels experienced by occupants. Occupant experience was assessed with a questionnaire developed in this project from the 'Office Environment Survey' by the UK Health & Safety Executive (Raw 1995). This was a two-page, self-administered questionnaire, applied to the occupants at the time of IEQ assessment. Key questions related to:

- Working conditions
- Discomfort from indoor climate in the preceding two months
- Symptoms or health complaints in the preceding two months specifically linked to office occupancy.

3. APPLICATION TO AN OFFICE REFURBISHMENT

It was considered that IEQ assessment of offices should be carried out with the following overarching guidelines:

- Assessment only during working hours with the building occupied
- Assessment both before and after refurbishment
- Each assessment to be duplicated for two seasons (summer and winter), the first being as close as possible to the building refurbishment,
- Provided that all levels of offices had a common air supply system and occupants had similar tasks/activities, measurements to be made on 3 levels (approximately bottom, mid- and top levels) over 5-8 consecutive work days,
- Measurements should be made at two distant locations on each level, with duplicate measurement of these locations on separate days.

A typical assessment plan for the above metrics, using specified sampling and measurement procedures (Brown 2006) is provided in Table 4.

Table 4. IEQ assessment plan for building with 6 office levels

IEQ Factor	Measurement location(s)			Sample time		Criteria for high performance
	levels	location s	out-door	sample	sample days	
IAQ						
TVOC	1,4,6	2	1	1h	2	500 µg/m ³ (1h)
Benzene	1,4,6	2	1	1h	2	10 µg/m ³ (1h)
Toluene	1,4,6	2	1	1h	2	4100 µg/m ³ (1h)
Formaldehyde	1,4,6	2	1	0.5h	2	100 µg/m ³ (0.5h)

PM2.5	1,6	2	1	8h	5	25 µg/m ³ (8h)
CO	1,6	2	1	8h	5	9 ppm(8h), 25ppm(1h)
CO2	1,6	2	1	8h	5	800 ppm (1h)
Microbial	1,6	20	-	-	-	none visible
<u>Ventilation</u>						
Effective air changes/h (ACH)	1,4,6	1	-	1h	2	< 1 ACH infiltration > 2 < 6 ACH w/mech.
<u>Thermal Comfort</u>						
Operative T	1,4,6	5+	1	8h	5	PPD<10%
Air velocity	1,4,6	5	-	2 min	5	0.05-0.2 m/s (2 min)
RH	1,4,6	2	1	8h	5	30-70% (1h)
Thermal gradient	1,4,6	5	-	2 min	2	<3°C (2 min)
Floor T	1,4,6	5	-	2 min	2	19-29°C (2 min)
Radiant T assym (0.1-0.6m)	1,4,6	5	-	2 min	2	warm ceiling < 5°C cool wall < 10°C cool ceiling < 14°C warm wall < 23°C
<u>Lighting</u>						
Illuminance (min)	1,4,6	6+	-	2 min	3	>160 lx
Task illuminance	1,4,6	8+	-	2 min	3	160-600lx (Aust Std)
Glare	1,4,6	4+	-	2 min	3	UGR < 19
<u>Noise</u>						
Sound level L _{Aeq,T}	1,4,6	5+	-	1h	3	Aust Std
Reverberation time	1,4,6	2	-	1h	1	Aust Std
<u>Occupant Comfort</u>	1,4,6	30	-	4h	1	<20% complaint rate
<u>Survey</u>						

Melbourne City Council had an office building in Melbourne city centre with two lower levels of carparking, six levels of offices occupied by its staff, and a plant room on the 9th level. This building, referred to as Council House 1 (CH1), was constructed in 1970 and *was* planned to be upgraded and refurbished in 2006. An IEQ assessment plan was developed for Levels 1, 4 and 6 of CH1 based on the above discussion, and these were carried out in July 2005 and February 2006. However the Council decided not to implement the refurbishment and so a replacement MCC building, Council House 2 (CH2), was used as a surrogate for CH1. The CH2 building was designed and built with a specific focus on sustainability and provision of a high quality indoor environment for occupants, similar to planning for CH1. CH2 consisted of shops at ground level (with own ventilation systems) and nine office levels housing approximately 540 staff, including approximately one-half of the staff from CH1. An IEQ assessment identical to that of CH1 was carried out for CH2 on Levels 2, 6 and 8. CH2 was designed as a benchmark sustainable building and was the first building in Australia to receive a 6-star design rating from the Australian Green Building Council. Key features of CH2 included:

- a sewer mining plant to deliver up to 100,000 litres of recycled water per day (note this had not yet started operating at the time of IEQ assessments)
- a low energy cooling system based on phase-change material
- vaulted concrete ceilings to improve air circulation, cooling and natural light, with ceiling mounted chillers
- automatic windows that open at night to cool the building in summer (these operated when the concrete slab ceilings exceeded 21°C and the outside temperature was ≥2 °C below that of the concrete ceiling)

- 100% fresh-air supply, nominally at 2 air changes per hour, from floor vents in a suspended floor plenum (operated on a timer with 1 h in front of occupant arrival and 1 h after departure)
- a CO₂ monitoring system to control ventilation rate to keep it below 800 ppm
- a facade of louvres to track the sun and shade the Western side
- roof-mounted wind turbines to draw hot air out of the building
- use of low-emission fit-out materials (the major interior surface was uncoated concrete, with some areas of paper gypsumboard painted with low-emission paint; mechanically fixed carpet tiles were used throughout; most office furniture was powder-coated low emission MDF and low-emission plywood sealed with a water-based lacquer)
- an open-plan office lay-out, common for both staff and managers.

CH2 was occupied by MCC staff in October 2006 and ‘tuning’ of the operation of the building was considered to be a requirement by the building designers over its first few months of occupancy. Hence CSIRO could not assess IEQ until March 2007 (summer) and August 2007 (winter), 5 and 10 months after occupancy. Note that the first assessment would ideally have been carried out within 1 month of occupancy when VOC and formaldehyde pollution are expected to be greatest (Brown 2002). Also of special significance was the upgrading of the lighting system the month before the August 2007 assessment, by adding extra strip lighting and adjusting light levels to suit workstations.

3.1 Key observations in CH1

General IAQ findings for CH1 were:

1. There was a high level of consistency found for both winter and summer assessments of IAQ and occupant perceptions of indoor environments
2. **Most IAQ measures were within the recommended criteria, with the exception of formaldehyde concentration and the occupant comfort survey, in particular on Level 6**
3. **Formaldehyde concentrations on Level 6 exceeded the IAQ criteria, especially in summer**, but no specific source for the formaldehyde could be identified; it was concluded that there were dispersed formaldehyde sources (e.g. office furniture, wall partitions) on this Level
4. CO₂ levels ranged from 560-710 ppm, much below the criterion 800 ppm, indicating that ventilation was adequate to remove occupant odours
5. VOCs, formaldehyde, fungi/bacteria and fine particles (PM_{2.5}) were present in CH1, while ozone from office equipment and carbon monoxide were below detection. Indoor air concentrations of VOCs and formaldehyde exceeded those outdoors, showing there were indoor sources for these pollutants. Fungi and PM_{2.5} were much lower indoors than outdoors, by a factor of 10- to 20-fold, showing there were no indoor sources and that significant cleaning of intake air occurred due to filtration by the mechanical ventilation system
6. Similar indoor air VOCs were observed in both seasons and there was no specific or consistent trend in the VOC concentrations according to the location sampled. This is considered to indicate that *VOC sources within CH1 were uniformly dispersed through the building*. Sources for the VOCs were not known, though several were considered to originate with outdoor air used to ventilate the building (e.g. benzene, hexanal, benzaldehyde and 1,2,4-trimethylbenzene), others (ethanol, acetone, and limonene) were clearly indoor source-related (probably from consumer products used by occupants), and some (toluene and xylene) were contributed by indoor sources and outdoor air
7. Indoor formaldehyde concentrations showed a trend in both seasons for increased formaldehyde concentrations at higher building levels, but this trend was not found to be significant ($p \leq 0.05$). However a seasonal effect for higher formaldehyde levels in summer

was significant for Level 4 and near-significant for Level 6. This effect could be related to the higher indoor temperature/humidity in summer c.f. winter since this factor is known to increase formaldehyde emissions from wood-based panels

8. **The occupant survey found that there were indoor environment complaints in CH1 from (in decreasing prevalence): air stuffiness, poor temperature control, dry air, lighting and noise (the latter two at ~1/2 the prevalence of the former)**
9. **Higher levels of building occupancy-related symptoms were observed on Level 6 than other levels, more so in summer, consistent with the higher formaldehyde concentration observed on this level.** The most prevalent daily/weekly symptoms on level 6 in summer were dry eyes (39%), lethargy/tiredness (36%), dry skin (20%), blocked nose/sore throat (16-19%), headache (12%) and chest tightness (8%).

General findings for thermal comfort, noise and lighting were:

1. Winter thermal comfort exhibited low dissatisfaction levels (range 5-15%) while Summer thermal comfort was more variable (dissatisfaction range 5-25%) especially for the early morning at the building perimeter. By comparison, occupant questionnaire responses showed high levels of complaint of *daily* temperature variability in both winter (38% complaint) and summer (27% complaint)
2. The background *office activity* noise was low for the open office areas, probably due to the highly sound absorbing environment which contributed to a more 'dead' than 'lively' acoustic quality. Work area measurements in an 'active environment' with people conversing were below 55 dBA c.f. a normal conversation level of 60-65 dBA. Generally, the target level of 40-45 dBA was exceeded, but AS2107-2000 specifies this for buildings that are operating but *unoccupied*. Reverberation times showed that there was little to no reverberation within the large open office environments.
3. Task illuminance exceeded 160 lux in all cases but the reading/writing/typing target of 320 lux was not achieved in ~30% of cases, with a bias to lower illuminances at lower Levels, probably due to lower daylight penetration. Occupant questionnaires showed a similar level of dissatisfaction with lighting for each Level with ~40% reporting dissatisfaction, but on a less than weekly frequency. Conversely, ~80% or more of occupants were satisfied with lighting on a weekly basis.

In overview, CH1 was considered to exhibit poor indoor air quality due to formaldehyde (especially on Level 6), though *all* other air quality metrics were acceptable. While thermal comfort, noise and lighting were of moderate to good quality, the occupants exhibited continuous, frequent complaints of stuffy air and temperature variability (both too hot and too cold). In both seasons, occupants reported high (20-30%) symptom prevalence related to building occupancy for dry eyes, lethargy/tiredness and headache, with greatest prevalence on Level 6. Notably, this Level had the highest formaldehyde levels, exceeding recommended criteria in summer.

3.2 Key observations in CH2

At present, only IAQ and occupant survey assessments have been fully evaluated and will be presented. General IAQ findings were:

1. There was a high level of consistency found for winter and summer assessments of IAQ and occupant perceptions
2. **All IAQ measures were within the recommended criteria, with the formaldehyde concentration being much lower than levels normally seen in office buildings (such as found in CH1) probably due to the low-emitting office furniture used**
3. CO₂ levels ranged from 500-690 ppm, much below the criterion 800 ppm, indicating that ventilation (based on 100% fresh air) was adequate to remove occupant odours
4. VOCs, formaldehyde, fungi/bacteria and fine particles (PM_{2.5}) were generally uniformly distributed in CH2, while ozone from office equipment and carbon monoxide were generally below detection. Indoor air concentrations of some VOCs exceeded those outdoors by

approximately 3-fold, showing there were indoor sources for these pollutants, though indoor TVOC levels were ~one-third the criteria level. VOCs measured by GC/MS were similar in both seasons, varying from a TVOC concentration of $<50 \mu\text{g}/\text{m}^3$ outdoors to $50\text{-}180 \mu\text{g}/\text{m}^3$ within CH2, this level of elevation normally being found in typical *established* buildings (Brown 2001), whereas *new* buildings (1-3 months old) can exhibit TVOC concentrations in thousands $\mu\text{g}/\text{m}^3$. TVOC concentrations above $500 \mu\text{g}/\text{m}^3$ are considered to indicate the need to remove strong VOC sources from buildings. CH2 was assessed at 5 and 10 months after construction and would not be classified as ‘new’

5. Fungi and $\text{PM}_{2.5}$ were much lower indoors than outdoors, by a factor of 10-fold or more, showing there were no indoor sources and significant cleaning of intake air due to filtration by the mechanical ventilation system
6. Similar indoor air VOC species were observed in both seasons and there was no specific and consistent trend in the VOC concentrations according to the location sampled or the season. Also, there was little difference between the dominant VOC species found in CH2 or CH1. This is considered to indicate that **VOC sources within CH2 were uniformly dispersed through the building and were from similar sources as in CH1**
7. **The occupant survey found that there were indoor environment problems in CH2 from (in decreasing prevalence): poor lighting, noise, poor temperature control and air stuffiness, with the poor lighting complaint persisting in winter after the lighting system had been modified**
8. **High incidences of occupant reported symptoms were observed for irritation/watering of eyes (approx. one-half of all occupants) in both seasons, possibly associated with poor lighting.** In winter, there were also high incidences of lethargy (34-44%) and headache (24-60%). Note that these are not consistent with the IAQ measurements, but may correlate to physical environment metrics.

In overview, CH1 exhibited poor indoor air quality due to formaldehyde (esp. on Level 6), though other air quality metrics were acceptable, and while thermal comfort, noise and lighting were of moderate to good quality, the occupants exhibited continuous, high incidence complaints of stuffy air and temperature variability (both too hot and too cold). Occupants reported high (20-30%) symptom prevalence related to building occupancy and for both seasons for dry eyes, lethargy/tiredness, and headache, with greatest prevalence on Level 6. CH2 was considered to exhibit high quality indoor air, though this was 5 months after construction and air quality conditions at initial occupancy are unknown. Occupants perceived the building to be poorly lit, noisy, variable in temperature and with stuffy air. Physical metrics are yet to be assessed for potential impacts on occupants.

4. CONCLUDING REMARKS

A range of physical and indoor air pollution metrics have been selected for measuring IEQ in office buildings relevant to their impacts on occupant well-being and comfort. These have been assessed in a target ‘traditional’ building and a new sustainable design building, both without significant problems or inconsistencies in applying the methodologies. Generally, IAQ metrics have been found to be well distributed through the buildings and to show high consistency across seasons. *This is considered to indicate that IAQ assessments can be made with fewer measurement locations and times than used here.* In the traditional building, high formaldehyde concentrations were found on one Level and occupant complaints of non-specific illnesses were highest on this Level. Physical measurements indicated good thermal comfort for this building, but approximately one-third of occupants considered temperature control to be poor. The sustainable-design building exhibited very low pollutant levels but the proportion of occupants reporting ‘stuffy’ air still exceeded 20%. Poor lighting was reported as a significant problem in this building by approximately one-half of occupants and this may have been a factor in the high symptom prevalence for irritation/watering of eyes, but physical measurements are yet to be assessed to confirm this.

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6. REFERENCES

- ASHRAE (1992). Thermal environmental conditions for human occupancy. ANSI/ASHRAE Standard 55-1992, Atlanta, Ga.
- ASHRAE (2004). Thermal environmental conditions for human occupancy. ANSI/ASHRAE Standard 55-2004, Atlanta, Ga.
- Australian Government (1995). Air-Conditioning and Thermal Comfort in Australian Public Service Offices - An information booklet for health and safety representatives. Canberra, Commonwealth of Australia.
- Standards Australia (1994). Interior lighting. Office and screen-based tasks. AS 1680.2.2, Homebush, NSW.
- Standards Australia (2000). Acoustics—Recommended design sound levels and reverberation times for building interiors. AS/NZS 2107:2000, Homebush, NSW.
- Brown, S.K. (1997). National State of the Environment Report - Indoor Air Quality. SoE Technical Report Series, Dept. Environment, Sports & Territories, Canberra, 88pp.
- Brown, S.K. (2002). Volatile organic pollutant concentrations in new and established buildings from Melbourne, Australia. *Indoor Air Journal*. Vol. 12, 55-63.
- Brown, S.K. (2005). Indoor Air Quality. In *Principles of Occupational Health and Hygiene. A Basic Guide for the Health & Safety Practitioner*, C. Tillman (Ed), Australian Institute of Occupational Hygiene, Tullamarine.
- Brown, S.K. (2006). High quality indoor environments for office buildings. CRC Construction Innovation Conference: Clients Driving Innovation. Gold Coast, March 12-14.
- International Organization for Standardization (1994). Moderate thermal environments - Determination of the PMV and PPD indices and specification of the conditions for thermal comfort, Switzerland.
- International Organization for Standardization (2005). Ergonomics of the thermal environment - Analytical determination and interpretation of thermal comfort using calculation of the PMV and PPD indices and local thermal comfort criteria, ISO 7730:2005, Switzerland.
- Olesen, B.W. (2004). International standards for the indoor environment. *Indoor Air* 14 (Suppl 7), 18-26.
- Raw, G.J. (1995). A questionnaire for studies of sick building syndrome. A report to the Royal Society of Health Advisory Group on sick building syndrome. BRE Report. RSH London.
- UK Government Sustainable Development Unit (1999), *Towards More Sustainable Construction: Green Guide for Managers on the Government Estate*. Sustainable Development Unit, June 1999
<http://www.sustainable-development.gov.uk/sdig/improving/partg/suscon/2.htm>.
- World Health Organisation (2000), *Guidelines for Air Quality*, 2nd Edition, WHO, Geneva.

IS WORK GETTING UNDER YOUR SKIN? LESSONS FROM THE OCCUPATIONAL DERMATOLOGY CLINIC, MELBOURNE

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Abstract

This presentation exemplifies the assessment of workers with suspected occupational dermatitis referred to the Occupational Dermatology Clinic in Melbourne.

Paper

The Occupational Dermatology Clinic started in 1993 and to end December 2004, 1743 patients have been referred for assessment and extensive patch testing. 1443 (82.8%) were diagnosed with a skin condition which was thought to be substantially or partially related to their work.

Dermatologists diagnose rashes on the basis of the history, appearance, degree of itching and time course. Sometimes a small biopsy of the skin may help in making the diagnosis. Itchy, red scaly rashes may be caused by eczema, which often occurs because of an inherited susceptibility, and is associated with asthma and hay fever. However, a similar appearing rash can be caused by contact dermatitis, which is related to an external substance contacting the skin. This often involves the areas of skin which come in contact with the causative factor, but sometimes can spread to other areas. The history of the rash is very important, including as to whether the rash has improved on holidays or time off work.

When assessing occupational contact dermatitis, it is first important to identify all the chemicals used in the workplace which can potentially contact the skin, and particular note is taken of those which can cause allergic contact dermatitis, known from experience or information provided on Safety Data Sheets. It is said that there are over 100,000 chemicals, and that approximately 3,600 have been reported to cause allergic contact dermatitis. In a workplace, it is necessary to understand the exact chemicals present and how they are handled, including the potential for actual skin contact, and whether these have been known to cause allergic reactions previously.

Workers are often exposed to many different substances, both at work and at home, and we have found it useful to develop a questionnaire to evaluate and assess different exposures, to assist our clinical assessment.

The skin rashes are then examined, to exclude numerous other causes of rashes, eg tinea, psoriasis, atopic eczema.

Contact dermatitis can be further sub-divided into irritant contact dermatitis and allergic contact dermatitis. 485/1443 (33.6%) were diagnosed with allergic contact dermatitis (ACD).

Allergic contact dermatitis is caused by specific allergic reactions to certain chemicals, which because of their molecular size and other properties, makes them potentially allergenic. Skin contact is required to initiate the process of developing an allergic reaction. Allergies may develop to a specific chemical after days, weeks or years of skin contact. The most common occupational allergens include chromate (in leather, cement), chemicals found in rubber, chemicals used in hairdressing, epoxy resins, other metals such as nickel, biocides or preservatives, and ingredients of some hand cleaners.

Case 1-4 exemplify allergic contact dermatitis to common occupational allergens.

Allergic contact dermatitis is specifically tested by patch testing. There is no diagnostic test for irritant contact dermatitis, but it is often diagnosed after the exclusion of allergic contact dermatitis. It is often impossible to differentiate irritant and allergic dermatitis on the basis of history and examination, so testing is usually required.

Irritant contact dermatitis is generally more common and was diagnosed in 608/1443 (42.1%) of those attending the clinic. Common skin irritants include water, especially the repetitive wetting and drying of the skin, heat, sweating, solvents, oils, soap, detergents, and dust.

People with a history of eczema and even asthma and hay fever, may be more prone to develop irritant contact dermatitis. Over the last few years, our group has developed a number of tools to prevent occupational dermatitis. The Skin@Work project was designed to inform careers' counsellors about high risk occupations for the skin. It was based on a previous study of careers' counsellors where we had found that careers' counsellors had poor recognition of the personal and occupational factors which contribute to dermatitis. Informative booklets about high risk careers were produced and distributed to schools throughout Australia.

Case 5 exemplifies irritant contact dermatitis. Internationally, there has been a focus on the role of wet work in contributing to occupational dermatitis. Some countries have published guidelines to reduce the amount of wet work performed by employees. Wet work is defined as exposure of the skin to liquid for longer than two hours per day or the use of occlusive gloves for longer than two hours per day or frequent hand cleaning. Workers in occupations involving wet work are especially prone to occupational irritant contact dermatitis. These include hairdressers, food handlers and healthcare workers. Together with the Office of the Australian Safety and Compensation Council, we have developed Guidelines for Wet Work (see www.ascc.gov.au)

Some workers may develop cumulative irritant contact dermatitis over many years, although this condition is poorly defined. Case 6 exemplifies this condition. This may take a long time to heal. Even after the skin appears normal, it is still more vulnerable to skin irritants.

Contact urticaria, an immediate hypersensitivity reaction was identified in 148 cases; over half were caused by latex allergy. We also see food handlers developing immediate allergic reactions of this type to certain foods, such as seafood. Other causes of this type of reaction include ammonium persulphate in hairdressers. Latex allergy should always be considered in those wearing rubber gloves and can be screened for with a blood test (RAST). Prick testing, which is different from patch testing, can also be used to diagnose this form of allergy. See Case 8 and Case 9.

Many workers have multiple contributing causes to their occupational dermatitis, such as ACD, ICD, latex allergy on a background of atopic eczema. This makes the clinical assessment quite complex, as in Case 10 and Case 11.

The prevention of occupational dermatitis relies on the principles of occupational hygiene, particularly eliminating exposure. However work environments are often complex and important causes of irritation include inappropriately irritating hand cleaners and incorrect use of gloves. Heat and sweating often contribute to dermatitis. A skincare plan for a workplace may include substitution of an irritant or allergen, engineering out exposure and/or use of appropriate protective gloves.

Gloves are often used incorrectly, and usually after the worker has developed dermatitis. Many workplaces rely on gloves to protect their workers, but we see many cases where gloves provide inadequate or inappropriate protection for workers, particularly for workers exposed to epoxy resins who often use rubber gloves which provide inadequate protection from epoxies and are often dissolved by concurrent use of solvents. Disposable vinyl gloves do not provide as much protection against microorganisms as latex or nitrile and thus are not always a good substitute for those with

latex allergy. Latex gloves are often used inappropriately, such as by hairdressers and food handlers, sometimes with disastrous consequences when people allergic to latex eat food handled with latex gloves.

Skin care also includes use of a less irritating skin cleanser or soap substitute and a greasy moisturising cream applied to the hands, preferably after work or before bed. While barrier creams are not always effective, they may make it easier to wash substances off the skin. Moisturising creams exist as lotions (runny and in a pump pack), creams (in a tube or a jar) and ointment (greasy like Vaseline). The greasier the moisturiser, the more effective it usually is. There is considerable international research interest in the repair of the skin barrier with moisturisers.

We have designed the RASH: Resources About Skin Health program as a package for workplace training for the prevention of occupational skin problems. It includes a training manual, CD, posters and was distributed to training organizations. It is available for companies to purchase.

Our group has also performed a follow up study of workers attending the clinic which has explored the different outcomes in occupational dermatitis. Preliminary results have suggested that some workers struggle with the complexity of the information concerning their diagnosis. We have tried to improve the explanations and advice given in the clinic, and when workers are diagnosed with occupational dermatitis, they are also reviewed after 4 months to assess their understanding of their skin condition. We have developed the concept of "Skin School": an educational program for workers who will attend for extra training in the understanding and management of occupational dermatitis which will start next year.

Unfortunately, some workers develop persistent post-occupational dermatitis, where their skin problem persists despite avoidance of causative factors, and even after stopping work, as exemplified in Case 10 and Case 11. Little is known about this debilitating condition, although some allergens such as chromate have always been associated with a poor prognosis. Hopefully our ongoing research will contribute to understanding its causes.

It is anticipated that these measures will contribute to the prevention and improved management of occupational dermatitis in Melbourne and hopefully Australia. Details of our work can be seen at www.occderm.asn.au. ODREC can also help with the investigation of outbreaks of dermatitis at work.

References available on request.

AN INVESTIGATION INTO THE HEALTH EFFECTS OF OCCUPATIONAL EXPOSURE TO BLACK SHALE AT MT TOM PRICE OPERATIONS, TOM PRICE WESTERN AUSTRALIA

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Abstract

Black shale (PBS) is extracted as a waste product of Iron Ore mining in the Pilbara region of Western Australia. The PBS located at Mount Tom Price Operations, administered by Pilbara Iron can contain high concentrations of pyrite (FeS₂), carbon, bitumens, poly-aromatic hydrocarbons (PAHs) and volatile organic carbons (VOCs) (Brocks et al. 2003 a). Limited research has occurred into the occupational exposures of mine workers in relation to dusts, fumes and mists associated with mining PBS.

Objectives

1. To determine the exact composition of PBS in the Tom Price region.
2. To quantify levels of exposure and potential health impacts of PBS on Pilbara Iron employees.
3. To assess and manage any potential risks associated with PBS exposure.

Methods

This longitudinal survey commenced during May 2007 and the sampling protocol is being repeated monthly for a period of 12 months, thus ensuring the capture of seasonal variations in exposure levels.

Samples of respirable dust, inhalable dust, vapors and gasses are being collected in terms of the appropriate methods. Hydrogen Sulphide, Sulphur Dioxide, Carbon Dioxide and Oxygen are being sampled with the aid of direct reading gas sampling instruments with data logging capacity. Workers potentially exposed to PBS have been stratified into similar exposure groups and on each sample run 6 personal samples from each SEG is being collected.

Data Analysis

Sample results are being compared to exposure standards and are being analysed with the aid of the IHSTAT statistical computer program.

Results

The results of the survey will be used to determine levels of risk and to develop appropriate interventions and guidelines for work in the PBS environment.

DANGEROUS GOODS RISK ASSESSMENT – QUALITATIVE VERSUS QUANTITATIVE

Richard Benbow

Benbow Environmental

ABSTRACT

Dangerous goods pose many risks to employees, the environment, the community and building spaces.

The change in legislation across Australia to a risk based assessment systems poses risks itself depending on the rigour of the risk assessment that is used.

A discussion using several examples and a proactive response from the attendees will be used to illustrate the outcome of qualitative versus quantitative analysis.

The opportunity will be taken to demonstrate the potential pitfalls of a risk assessment versus adhering to the relevant Australian Standards or an equivalent level of safety.

ASBESTOS MANAGEMENT

Asbestos management remains a serious concern for many property owners. The common use of asbestos containing materials within the building construction ceased over at least 24 years ago, but the remnants of its past use are still a cost to the community and exposing the unwitting to potential exposure to respirable asbestos fibres.

A strict regulatory regime is in place and the activities of the regulatory authorities, from the author's experience are armed with effective means of protecting employees where a hazardous material audit has been conducted by an experienced hygienist or equivalent professional. There are many instances where the asbestos containing materials are not in the building but are buried on the site or have been placed innocently or deliberately in the ground; less frequently now the asbestos cement may be in building rubble used as fill. There are many instances where in the past building rubble used as fill contained asbestos cement.

The author has prepared a number of case studies from the past 5 years, some as recent as the past two years.

Case Study One

A limited number of community sites were inspected for presence of hazardous materials, the discussion will focus on asbestos containing materials.

Where as competitive pricing for tenders for these audits means that the property owners are satisfied as long as they possess a consultant's report, the quality of the audits is very much in doubt. Our experience suggests that property owners are not being made aware of the risk of there being asbestos cement buried on site, often with little effort it can be found.

Tenders are not insisting that grounds be inspected resulting in a report of limited use. It is highly recommended that the grounds are inspected, where access to underneath building is available, this area is inspected and the verges of footpaths and nature strips are included.

Ignorance is the peril of those specifying the extent of the audit. For every site we audit we more often than not find asbestos containing materials either in unsealed areas of the site or the verges of footpaths.

Examples will be demonstrated during the presentation of this workshop paper.

Suburban and urban areas affected are not limited by the presence of asbestos containing materials in buildings. Similarly school sites are frequently finding asbestos cement in fill that is being “donated” by a well meaning person ignorant of contamination that is present.

The sources of the materials are relatively easy to understand and two messages need to go to property managers

Include the grounds and nature strips in the audit

Do not except fill from anyone without a signed certificate of clearance and letter of indemnity.

Action to be taken

Fragments of asbestos cement in soil converts a site into being contaminated by friable asbestos. Removal requires rigid compliance with Occupational Health & Safety laws. Transport and disposal of the soils require rigid adherence to protection of the environment and waste management legislation. There is a significant duty of care to the community, the property owner and future users of the contaminated land.

The basis steps are well understood and include:

Engage an experienced hygienist or equivalent person with minimum 10 years experience in asbestos management. Ensure this person actually oversees all site activities in person.

Prepare a health and safety plan in conjunction with a licensed asbestos contractor.

Prepare a remediation action plan with each step detailed. This plan needs to detail the procedures to ensure risk is extremely well managed. It is essential that adequate safeguards are in place. These centre on the fact that asbestos contractors are required and the attitude of those carrying out the tasks is a major factor in ensuring risks are minimised.

Rigorous checking by the hygienist of the quality of the work practices is needed. Benbow Environmental insists on remaining at site during a site clean up.

A Certificate of Clearance is required. This is a statement that the area is free of asbestos fibres and remnants of asbestos containing material. How is this guaranteed, as it is a critical final step?

There are several approaches applied depending on the circumstances.

For external clean-ups a triple inspection has been found from our experience to be needed. The first inspection is undertaken in the company of the asbestos contractor. A second inspection is undertaken by the hygienists and then the third final inspection when soil samples are obtained from many areas of the site to ensure an aggressive approach is taken. It is relatively easy to allow the pressure of the clean up to be successful to reduce the aggressiveness of the soil sampling.

For schools exposed to a clean-up, adopt a very tough approach in extensively sampling surface dusts for the presence of clumps of asbestos fibres. There is a policy of zero risk applied to the potential exposure of school children to asbestos fibres.

Case Study 2

This discussion follows from the previous example and involves a school that unwittingly accepted a truckload of clean fill removed from a residential site under redevelopment.

The truckloads, approximately 12 tonne, were spread over half a football field. Heavy rain “floated” the unknown pieces of asbestos cement across the edges of the field and onto access walkways, a cricket pitch and adjacent to basketball courts where it was discovered.

The standard method of removal would have been to remove a 300-mm depth of soil. A different approach was developed that proved very successful and was able to leave the surface of the field at the same surface level.

DISTANCE EDUCATION OF OCCUPATIONAL HYGIENE FOR UNDERGRADE STUDENTS.

Ryan Kift

Central Queensland University

Occupational hygiene education is an important consideration for anybody that is working in the area of Occupational Health and Safety (OHS). Currently Australian higher education institutions offer many different pathways to become educated in OHS. There are many different post graduate diplomas and degrees that offer education in what could be described as general OHS. There are a small number of Universities that offer undergraduate degrees in OHS. Several Universities incorporate some form of OHS into related fields (such as environmental science or engineering). There is debate about if Occupational Hygiene has a place within these generic programs or if it should be a specialist area only. Central Queensland University currently offers programs in both postgraduate and undergraduate study in OHS. It is the undergraduate program that this paper will concentrate on. Currently the Bachelor of OHS includes a course that intends to cover the principals of Occupational Hygiene.

This paper will discuss the merits of delivering Occupational Hygiene education within a general degree framework to undergraduate students. It will discuss if people training to become general OHS practitioner need Occupational hygiene education, and if so, in what form. The paper will then go on to discuss many of the positive and negative attributes that both students and employers can expect from a course that is delivered through a fully flexible (distance) mode. The way that the fully flexible delivery mode (all on-line) can be optimised within a hands-on discipline area such as occupational hygiene will also be discussed.

BOHS ASBESTOS QUALIFICATION – AN OVERVIEW

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Abstract

This paper will provide an overview of structure and contents of the BOHS system of qualifications for asbestos related subjects. The range of modules will be reviewed and the nature of the examination system discussed. The structure of the Faculty Examinations Committee will be discussed and input from stakeholders will be identified.

1. Introduction

The introduction of the UK Asbestos Regulations in the late 1960s saw the need for assessments of exposure to be carried out. The use of asbestos in the UK peaked around 1973 and by the early 1980s a significant increase in the interest in the effects of exposure to asbestos in the workplace was seen. During this period there was very little in the way of formal training in asbestos identification and measurement. McCrone Laboratories in London offered a short course on asbestos identification with Jean Prentice also sharing her knowledge of where to find asbestos with the students. The predecessor of the current Faculty Examinations Committee (FEC) British Examining Board in Occupational Hygiene (BEBOH) module on hazardous substances also contained a short section on fibrous hazards with course providers such as Portsmouth Polytechnic adding extra on identification and counting. The shortage of suitable training being available meant that asbestos surveys were almost exclusively carried out by occupational hygienists relying on their experience of dealing with other types of hazardous substance.

The significant increase in work in this area meant by the mid 1980s many occupational hygienists were finding that they were spending significant amounts of their time doing asbestos surveys and clearance tests.

The Examining Board recognised that this change in the work of many occupational hygienists was not being supported by the provision of a suitable and comprehensive training course. This recognition led to the development of the Asbestos and other Fibres Occupational Hygiene Module (S301).

As the asbestos clearance industry continued to grow asbestos surveying, analysis and clearance testing began to become more and more specialised with contractors having no occupational hygiene experience or training offering these services.

Concern over the competence of some of these specialist contractors led to calls for tighter control and for formal training to be required. BOHS responded to an approach by the UK HSE and produced the first of a series of specialised proficiency modules in asbestos.

2. Overview and structure of qualifications

The S301 and W506 courses are intended to provide a comprehensive knowledge base covering all aspects of the subject area and as such are primarily aimed at occupational hygienists and asbestos laboratory managers rather than specialists such as asbestos surveyors and analysts.

The proficiency modules (P401 – 406) are designed to give practical skills in defined areas of expertise and are designed to provide a level of proficiency in the relevant specialist area. The individual course aims are listed below. Detailed syllabi can be down-loaded directly form the BOHS website (www.bohs.org).

2.1 S301 Asbestos and Other Fibres

The aim of this module enhances the student's knowledge of occupational hygiene practice in relation to fibrous dusts. The module concentrates on asbestos, but other fibres, e.g. MMMF, Aramids, etc., which are increasingly finding uses in industry are also covered.

This module aims to be of benefit to those working in asbestos consultancy as well as main-stream occupational hygiene, giving an understanding of the health risks associated with asbestos and other fibres as well as the means of evaluation and control.

2.2 W506 Asbestos and Other Fibres (International)

This module is the international version of the S301 course and as such does not contain UK legislation and UK specific practice.

2.3 P401 Identification of Asbestos in Bulk Samples (PLM)

The aim of this module is to provide theoretical and practical knowledge in the techniques of asbestos sample identification using polarised light microscopy (PLM).

2.4 P402 Building Surveys and Bulk Sampling for Asbestos (Including Risk Assessment and Risk Management)

The aim of this module is to provide background and practical knowledge in the surveying of buildings for the presence of asbestos and any necessary bulk sampling that may be required. It also provides general guidance on the management procedures necessary to minimise exposure to any identified asbestos.

2.5 P403 Asbestos Fibre Counting (PCM)

This module provides theoretical and practical knowledge in the techniques of fibre counting of asbestos air samples using phase contrast microscopy (PCM).

This module, in conjunction with the Proficiency Module P404 – Air Sampling and Clearance Testing for Asbestos, covers the requirements of the '4-Stage Clearance procedure'.

2.6 P404 Air sampling and Clearance Testing of Asbestos

This module provides theoretical and practical knowledge in the techniques of air sampling and clearance testing of asbestos.

2.7 P405 Management of Asbestos in Buildings

The aim of this module is to provide the student with the practical knowledge and the skills to be able to manage asbestos in buildings and to provide a basic knowledge of asbestos removal procedures.

2.8 P406 Supervision and Management of the Safe Removal and Disposal of Asbestos

The aim of this module is to provide the training required for supervisors and those managing the removal of or working with asbestos.

The range of courses offered and the content of syllabi is continually reviewed and updated with the requirements of UK legislation and our partner organisations. Interest has recently been shown in an international version of P402 and BOHS is willing to consider developing international versions of any of the other proficiency modules if there is demand.

BOHS examinations do not provide certification. This is currently done through organisations such as ABICS (for asbestos surveyors) who require the candidate to hold the relevant proficiency certificate before applying.

3. Examination System

The BOHS examination system uses a combination of multiple choice question (MCQ) based examinations, essay questions, practical assessments, oral examinations and field reports as means of assessment.

MCQ examinations use questions drawn from subject specific question banks. Experts in the area write the questions and the question bank is regularly up-dated and reviewed by the MCQ Committee. Negative marking is used in this examination.

Practical assessments are practical examinations carried out by FEC registered Practical Assessors. The Practical Assessors follow a prescribed format for the examination and use FEC marking schedules. Practical Assessors work as independent consultants and are paid by the course provider. They are prohibited from teaching on the course being assessed or being directly employed by the course provider. Practical Assessors are required to attend a BOHS practical assessment training course before applying to join the register. Applications to join the Register are approved by the Chief Examiner. A FEC QA officer audits Practical Assessors on a regular basis and if their performance is not acceptable they are issued with formal warnings and can be removed from the Register.

The S301 qualification is the only asbestos course that has a part B examination that is an essay question and a formal oral examination. This is because this qualification forms part of the occupational hygiene module series rather than the proficiency module series.

Both P402 and S301 require the submission of reports of relevant work carried out in the field by the candidate. These reports are designed to show that the candidate is capable of carrying out the practical aspects of the work in "real life". Candidates are given guidance on what they should include in their reports. The reports are assessed by FEC report readers and classified as acceptable, needing clarification at oral (for S301) or required to be resubmitted.

Candidates that successfully pass all parts of the S301 examination are awarded a Certificate in Operational Competence (CoC) in Asbestos.

Candidates who hold all the proficiency certificates from P401 to P405 are classed as holding the CoC in asbestos.

4. Stakeholders

The Faculty Examinations Committee (FEC) runs the examination system on behalf of BOHS. This committee is chaired by the Chief Examiner and consists of the Quality Assurance Manager, the MCQ Committee Chairman, the Deputy Chief Examiner, The Asbestos Technical Advisor and the BOHS Administrator. As well as providing syllabi and examinations the committee appoints and manages the teams of examiners, report readers and practical assessors. FEC has two sub-committees, the MCQ committee and the International Qualifications Committee (IQC). The MCQ committee is responsible for maintaining the MCQ question banks. ICQ is a new committee that has been set up to manage BOHS international qualifications and has representatives from collaborating organisations such as AIOH and HKIOEH as members.

The UK Health and Safety Executive (HSE) are important partners in the development of qualifications in this area. Many of the existing qualifications have been developed at the specific request of the HSE and are referred to in official guidance. The P401 practical assessment uses AIMS samples and the P403 practical assessment used RICE slides provided by the HSE.

United Kingdom Accreditation Service (UKAS) accreditation is required for laboratories and organisations to carry out asbestos surveying, clearance testing, air sampling and similar work in the UK. UKAS require that people within these organisations have appropriate qualifications. As the sole provider of almost all of these qualifications FEC works closely with UKAS to ensure that they have input into changes and updating of these qualifications. UKAS would normally expect that the manager of an asbestos surveying service or testing laboratory to hold the S301 qualification.

The BOHS asbestos courses are delivered by approved course providers. To gain approval course providers are required to apply, in writing, to the Chief Examiner. As part of their application they must provide details of their organisation, their training facilities, their internal QA procedures and the CVs of their tutors. Approval is generally given for specific proficiency modules rather than a blanket approval to run all courses. Once the course provider is approved they are added to the list of BOHS approved course providers and they can then advertise the course. From early 2008 all course providers will be subject to regular quality audits and inspections by FEC quality assurance officers. Failure at audit may result in the course provider being removed from the approved list.

FEC maintains close contact with trade bodies such as the Asbestos Removal Contractors Association (ARCA) to keep them informed of changes and developments in the BOHS asbestos qualifications.

BOHS has recently launched a series of refresher courses and examinations designed to update asbestos practitioners on current developments and to meet their CPD requirements.

Regular meetings between FEC representatives and stakeholders allow dialogue and input into the direction and development of the qualifications.

5. Conclusion

BOHS has developed an integrated series of proficiency modules designed to meet the needs of professional practice and the requirements of UK Asbestos legislation. These modules apply best practice to the areas of analysis, fibre counting, clearance testing, building surveys, asbestos management and the management of asbestos removal.

It is the aim of BOHS to develop international versions of the qualifications in collaboration with our international partners. The comprehensive W506 Asbestos and other Fibres (International) syllabus has been approved and the examination will be available in March 2008. The development of course materials and a student handbook for this course are being funded by BP and it is

anticipated that this material will be available to approved course providers via the BOHS website early in 2008.

Finally, BOHS would welcome approaches from any organisation that might be interested in use or development of local versions of these modules.

AIOH AWARDS – BENEFITS & OUTCOMES

Gerard Tiernan¹, Kerrie Burton², Samantha Clarke³, Julia Norris⁴,

¹Simtars

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Abstract

The AIOH has a very impressive awards program that provides for travel to international conferences and site visits. Samantha Clarke and Kerrie Burton received the *Dräger Safety Pacific Development Award for Young Hygienists* in 2005 and 2006 respectively. Julia Norris and Gerard Tiernan received the *Airmet Professional Development Award* in 2005 and 2006 respectively. This paper will provide an overview of the site visits and conferences (British Occupational Hygiene Society annual conference and American Industrial Hygiene Conference and Exposition) attended by the award recipients. The site visits included a UK steel mill, the Institute of Occupational Medicine, the UK Health Safety Executive, Dräger in Lubeck, Germany, a carbon electrode plant in Italy, the National Mine Health and Safety Academy in Beckley, West Virginia, NIOSH in Pittsburgh, Morgantown, Cincinnati and Lake Lynn, Dayton University in Ohio and the SKC facility in Eight Four, Pennsylvania. This paper provides feedback to the AIOH community about the benefits and importance of these awards. It is indeed a credit to the AIOH that these awards are available and a testament to the work done by many past AIOH Councils and senior members. We, the members of the AIOH, are very fortunate to have access to such awards. They provide a unique opportunity for young and mid-career hygienists to see first hand and face to face international occupational hygiene experts and facilities. The unique opportunities provided to hygienists by these awards are of immeasurable benefit. The site visits and conferences have not only increased our skills and knowledge but have provided lifetime contacts for professional dialogue and information sharing. They have certainly enhanced our personal “Strive for Excellence”!

1. Introduction

The AIOH has an awards program that provides travel to international conferences and site visits - the *Dräger Safety Pacific Development Award for Young Hygienists* and the *Airmet Scientific Professional Development Award*. The Dräger award provides travel to and attendance at the British Occupational Hygiene Society annual conference and the AirMet award provides for attendance at the American Industrial Hygiene Conference and Exposition. Both awards also allow for associated site visits.

2. AirMet Scientific Professional Development Award

The AirMet Scientific Award is a premier, professional award of the Australian Institute of Occupational Hygienists. It is sponsored by AirMet Scientific on an annual basis. The award provides a selected applicant with the opportunity for professional development through attendance at the American Industrial Hygiene Conference and Exposition (AIHCE) and an invitation to travel to SKC Inc in Pennsylvania. Additional visits relevant to the applicant's professional development may be funded, subject to negotiation between the applicant, the sponsor and the AIOH Council. The award is essentially a mid-career award. It is not a reward for long service and will not normally be given to members who are approaching retirement. Perceived benefit to the recipient and perceived benefit to the profession in Australia are of prime importance. Full, Provisional and Fellow members of the AIOH are eligible to apply.

3. Dräger Safety Pacific Development Award for Young Hygienists

The Dräger Award is an award of the Australian Institute of Occupational Hygienists, sponsored on

an annual basis by Dräger Australia. The award provides a selected applicant with the opportunity for attendance at the British Occupational Hygiene Society annual conference supplemented with travel to Dräger, Luebeck, Germany and, subject to negotiation, additional visits relevant to the applicant's professional development. Any member of the AIOH who is under 35 on 31 December in the year of application is eligible to apply for the award.

4. Site Visits

The American Industrial Hygiene Conference and Exhibition was held in Philadelphia, Pennsylvania, and the BOHS Conference in Glasgow with a theme of "Promoting a Healthy Working Environment". Prior to the American conference there is a week-end of professional development courses (PDCs).

The additional site visits for both the Dräger Award and the Air Met Award are dependent on the award applicant to organize but past award winners and members of the Awards and Sponsorship Committee can be very helpful in setting up an itinerary.

The sites visited & PDCs were:

Gerard Tiernan

- The MSHA (*Mining Safety and Health Administration*) *National Mine Health and Safety Academy* in Beckley, West Virginia.
- NIOSH (National Institute for Occupational Safety and Health) Pittsburgh Research Laboratory (PRL).
- NIOSH Lake Lynn Laboratory, located 80 km southeast of Pittsburgh, an experimental underground limestone mine and surface quarry area.
- NIOSH Morgantown, West Virginia, *Division of Respiratory Disease Studies* (DRDS). The DRDS has a number of branches including *Field Studies* (conducts field investigations into exposure), *Surveillance* (collates survey data and runs health surveillance programs); and *Laboratory Research* (studies methods for exposure assessment and engineering controls). Morgantown also has the *Division of Safety Research* (DSR), the *Division of Respiratory Disease Studies* (DRDS) and the *Health Effects Laboratory Division* (HELD).
- PDC on Bayesian Statistics – run by AIHA *Exposure Assessment Strategies Committee*
- PDC on Risk Assessment run AIHA *Risk Assessment Committee*.

Julia Norris

- PDC on Developing a training package or EHS presentation worthy of an Academy Award Nomination!!
- PDC on Delivering an EHS presentation worthy of an Oscar.
- NIOSH Cincinnati, Ohio, *Division of Surveillance, Hazard Evaluations, and Field Studies* (DSHEFS) which conducts epidemiological studies and health hazard evaluations in industry. Cincinnati also is home to the *Division of Applied Research and Technology* (DART) which produces the *Manual of Analytical Methods* and also looks at controls and organisation of work or job stress; and the *Education and Information Division* (EID) which conducts risk assessments, and produces recommended exposure limits (RELs) and various NIOSH documents.

- NIOSH Morgantown- *Pathology and Physiology Research Branch* which is also associated with the University of West Virginia. Here there is a large research group focusing on studying the health effects of nanomaterials.
- Dayton University, Ohio - Dayton University Research Institute encompassing the Nanocomposite research centre.
- SKC facility at Eight Four, Pennsylvania.

NIOSH also has a centre in Spokane, Washington, which is the major NIOSH site for metal/non-metal mine research.

Kerrie Burton

- Bayesian Statistics workshop
- Corus Group Hygiene function. Corus is a multinational company which manufactures, processes and distributes steel products.
- Corus Group Scunthorpe Integrated Steel Plant.
- Institute of Occupational Medicine, Edinburgh.
- Health and Safety Executive in Bootle.
- Drager in Lubeck, Germany.

Samantha Clarke

- Bayesian Statistics workshop
- Institute of Occupational Medicine, Edinburgh.
- Health and Safety Executive in Bootle.
- Drager in Lubeck, Germany.
- SGL plant in Ascoli Piceno, Italy – a manufacturer of high and low density carbon electrodes predominantly used in steel-making furnaces

5. Areas of interest

AIHce

The American Industrial Hygiene conference and exposition is an incredible experience as well as an awesome feat of organisation. With over 7000 attendees, 300+ exhibitors, 14 concurrent sessions running at any one time and 80 continuing education seminars running over the weekend, it was hygiene on a grand scale. The conference provides an opportunity to experience the hygiene profession as it should be i.e. given national recognition, with dedicated government departments and funding and comprehensive membership including large industry, government, military, consultants and educators.

Areas of interest at the AIHce Conference were:

- The PDC on Bayesian Statistics presented by John Mulholland, Paul Hewett, Perry Logan and Gurusurthy Ramachandran.

- The Risk Assessment PDC presented by Mike Jayjock and Sharon Arnold.
- The AIHA *Exposure Assessment Strategies Committee* meeting (attended by Gerard Tiernan as an observer).
- The opening of the Conference with the trooping of the colours - three uniformed and armed soldiers march in carrying the US flag and then The Star Spangled Banner is sung.
- The opening speaker - Steve Uzzell, a photographer – who presented his incredibly moving and fascinating presentation “*Open Roads Open Minds: An Exploration of Creative Problem Solving*”.
- The expo – the American conference expo has to be seen to be believed with row after row of exhibits, lunch-time chat sessions, the poster display, the AIHA and ACGIH pavilions with all their publications and resources, and other special displays such as the Pandemic Planning display and the methamphetamine lab display that were on this year.
- The annual ACGIH meeting and AIHA meeting.
- The Job Fair at AIHce – you can look for jobs and book an interview on the spot.
- The American Industrial Hygiene Foundation Fun Run.
- The SKC International Luncheon.

The specific areas of interest for Gerard Tiernan’s site visits were:

- Discussions with William (“Rocky”) McKinney, *Instructor, Occupational Hygiene* at the MSHA *National Mine Health and Safety Academy*. Rocky explained the MSHA *Data Retrieval System* where it is possible to look up mine monitoring data on the web <http://www.msha.gov/drs/drshome.htm>.
- The hydrostatic testing of seals and mobile diesel engine emission testing platform at NIOSH’s Lake Lynn mine. The diesel research team is led by Alek Bugarski. <http://www.cdc.gov/niosh/nas/mining/researchproject29.htm> Alek explained that they were running a 100 kW diesel engine on a dynamometer and measuring various components of the exhaust emission. They were particularly interested in the nano-particles being emitted.
- At NIOSH Morgantown the *Coal Workers Health Surveillance Program*. This involves providing an X-ray, lung function and medical examination to miners. There were approximately 175 cases of coal worker’s pneumoconiosis (CWP) identified in the US last year. The Program has a *Mobile Occupational Safety and Health Unit*, which is run by Anita Wolfe and Dr Edward Petsonk. <http://www.cdc.gov/niosh/nas/mining/researchproject67.htm>
- Also at Morgantown discussions with Dr Martin Harper, *Chief, Exposure Assessment Branch, HELD*, regarding the ISO silica committee TC146 / SC 2 (of which he is currently Chair), the NIOSH Asbestos Roadmap <http://www.cdc.gov/niosh/review/public/099/>, a high flow respirable dust sampler from Germany which will allow analysis of silica down to the current ACGIH standard of 0.025mg/m³, a NIST standard reference material for on-filter silica analysis, thoracic size selective samplers, special gridded cover slips for asbestos counting so a counter can return to the same field of view, the effect of clay coatings on silica particles in terms of disease rates, and metal analysis of samples from plastic cassettes – results indicate that up to 50% of the sample can be on the walls of the cassette.

- At NIOSH Pittsburgh the research emphasis since the recent spate of fatalities in the US mining industry has been on communication, rescue stations, self-contained-self-rescuers, and seals. Discussions re these issues were held with Dr Güner Gürtunca, *Director, PRL* and Edward Thimons, *Chief, Respiratory Hazards Control Branch*.
- Also at Pittsburgh discussions with Jerry Joy, Jay Colionet, Steve Mischier and Jon Volkwein about dust monitoring and control and the diesel particulate control program. PRL has a dust laboratory with a functioning continuous miner face and longwall face.

The specific areas of interest for Julia Norris' site visits were:

- Discussions with Tabitha Maher, Certified Industrial Hygienist with Altair Nano, a nonomaterial producing company in Reno. Tabitha runs the hygiene program at Altair Nano, the basis of which is education, assessment and continuous improvement. The key to the successful program is complete transparency and honesty with comprehensive training for all new employees about what is in place and what they still don't know. Where possible the process has been automated and continuous assessment and improvement are encouraged. In addition the lack of guidelines and exposure standards for nanomaterials is not seen as an excuse to do nothing in regards to assessment and control, but rather as motivation to maintain exposures as low as reasonably achievable.
- At NIOSH Cincinnati the focus is on developing guidance material and establishing some exposure assessment criteria. Dr Chuck Geracci heads up the nanotechnology research centre and is the chief of the document control branch of NIOSH. Chuck's comment was "we don't know all the answers, but we know what questions to ask" and that even though there are a lot of unknowns when dealing with nanomaterials, we can still move forward in control and precaution. Also at NIOSH in Cincinnati, discussions were held with Dr Eileen Keumpal, whose focus is establishing exposure standards and guidelines. In doing so Eileen believes that we can learn from what we already know about the characteristics and behavior of superfines from welding and diesel exhaust studies to help expand our knowledge of both toxicology and sampling of nanomaterials.
- At NIOSH's Hamilton Laboratories in Cincinnati, there is a group dedicated to developing assessment and measurement strategies for nanomaterials. Doug Evans is a hygienist working for NIOSH who is undertaking workplace assessments as part of a collaborative program between industry and NIOSH. NIOSH provide free consultative services to nano industries in return for permission to use the data in its research. Doug also promoted that where there is a lack of guidelines and standards, apply basic risk management and hygiene principles. Some of the assessment strategies used were
 - Background -v- process (careful of forklifts, welding, generators when assessing background levels)
 - Particle number (Condensation particle counter, ELPI multi-stage impactor)
 - Surface area (diffusion charger >100nm)
 - Traditional pump and filter method
 - Indicator of source
 - Subsequent analysis for surface area or chemistry (SEM, TEM)
- At Dayton University research Institute discussions were held with Lynn Bowman, the

human factors associate at the institute. Lynn detailed some of the barriers to effective health and safety in a university environment with a large transient student population coming through the laboratories. A tour of the nanocomposite research facility where nanomaterials are used in the manufacture of plastic products was also undertaken.

- At NIOSH Morgantown discussions were held with Dr Vince Castranova, *Chief, Pathology and Physiology Research Branch*. Vince had organized several of his researchers to present their current projects which included pharyngeal aspiration studies of single walled and multi walled carbon nanotubes. There were some difficulties with inhalation studies because of the tendency for the nanotubes to agglomerate with some research going into developing a means to overcome this. The major findings so far included rapid and persistent fibrosis within 3 days of exposure in rats. Neurotoxicological effects were also seen at the higher end of the dose range. There were also concerns about continuous release of nanomaterial from the granulomas in the lungs given that simulated conditions similar to that in the lung had been found to promote dispersion of agglomerates.
- The visit to the SKC facility at Eight Four, Pennsylvania, provided a tour of the manufacturing area including the making of sampling tubes. Much of the manufacture, particularly of sample media tubes, is done manually.

BOHS Conference

The specific areas of interest at the BOHS Conference were:

- the Bayesian Statistics workshop;
- the discussions about REACH;
- crystalline silica from an EU perspective;
- sessions on noise, EMF and communication.

At the Health Safety Executive in Bootle the particular areas of interest were:

- COSSH and COSHH essentials. Paul Evans described COSHH Essentials and recent changes in legislation requiring companies to meet best practice as well as achieve compliance with OEL's. The thought being that if best practice controls are implemented and maintained, OEL compliance will result. It is anticipated that this will change the way hygienists (in particular, consultants) provide support to companies, i.e. reducing the emphasis on monitoring.
- Local exhaust ventilation. John McAlinden discussed the BOHS competency for LEV design, which considers exposures, COSHH and efficiency testing.
- Communication, in recognition of the fact that if you want to get a particular message out there you need to think about who you are trying to communicate to and consider the best tools to deliver the message effectively, usually more than one medium is needed.

At the Institute of Occupational Medicine discussions were held with:

- Rob Aitkin (Director of Strategic Consulting) who provided a history and overview of the facility, and discussed his interest in the field of nanotechnology.
- R Gravelly (Head of Human Sciences), whose interests include:

- Thermal stress – including the physiological effects of wearing PPE and attempting to correlate the radio pill (to measure core temperature) with cheaper alternatives;
 - Musculoskeletal disorders - manual handling and accumulative upper limb disorders; and
 - Worker psychology - work/life balance, the application of industrial controls (eg. Workers actually using LEV, wearing RPE then standing in the plume etc.) and the concept of improving OHS through awareness, attitude and behaviour.
- Keith Sinclair (Manager, PPE Testing), who looks after the:
 - Testing of worker suits and disposable overalls to meet British Standards; and
 - Respiratory fit testing to standard OC282/28. Interestingly they chose not to use the TSI N95 companion for disposable respirators, rather increasing the fit test criteria for passing.
 - Martie Van Tongeren (Head of Exposure Assessment), who has interests in:
 - Nanotechnology;
 - REACH (manganese industry);
 - EMF; and
 - Dermal and ingestion exposure (developing an ingestion model for dirty hand in mouth).

The Dräger visit was hosted by Oliver Schirk, Regional Focus Group Manager, and he provided:

- A history of the company including the corporate video, the interactive museum, which demonstrated the history of Dräger as well as the evolution of their many products, and a walk through the beautifully landscaped grounds with quaint buildings (more like a small village, than an industrial site).
- An overview of the manufacture of detector tubes, CMS chips, gas-tight suits and respirators. In a similar fashion to SKC much of the manufacture is done manually, particularly the detector tubes, with some automatic aspects as well.
- A tour of Lubeck, a beautiful city which we were able to appreciate both through a “walk through survey” and a river boat tour.

6. Benefits to yourself and the profession

The applications for both these awards ask the applicant to explain how attendance at the relevant international conference and any associated site visits will contribute to their professional development. This question is often difficult to answer when putting the application together and even after the experience it is difficult to describe, in meaningful words, the considerable benefits of all the opportunities the award has provided. These trips provide incredible opportunities for personal growth and development. There is nothing like meeting international experts face to face and viewing the way things are done in another part of the world. It broadens the mind beyond your own little patch. It lets you know that there are real people behind the books, articles and systems that you read and use every day - that these real people are passionate and excited about the work they are doing, and are keen to talk to you about their work – that you can now email someone you

have actually met and ask them a specific question. These interactions help to refocus your efforts and provide insight into concepts and ideas that you may not have seen before. We can all always learn more and these trips provide that learning and give a solid foundation for more learning. You get to share what is happening in Australia with international colleagues. And you find that in many areas Australia is doing very well. We believe that the benefits of these trips are not only limited to the here and now but will continue to grow and develop over the years ahead.

7. Thanks

We would sincerely like to thank Dräger, in particular Wolfgang May and Kate Leahy, and John Garnett from Air Met Scientific for providing us with this opportunity. We also acknowledge the hard work done by AIOH Awards and Sponsorship Committee and the AIOH Council.

PROPOSED GUIDELINE FOR WRITING OCCUPATIONAL HYGIENE REPORTS

Philip J Turner CIH, MSAFSC, MAIOH

WorkPlace Environment Consultants Pty Ltd

1. ABSTRACT

Occupational hygienists must clearly communicate their findings in written form in order to achieve improved health, safety and/or environmental performance. The challenge is to present pertinent and accurate technical information with sufficient detail in a form that can meet the demands of professional review but still be readily understood by a non-technical audience. Potential readers of any single occupational hygiene report include business owners, business managers, employees, trade union officials, government officials, other health professionals, engineering professionals and legal professionals. In order to promote effective communication by occupational hygienists the Victorian WorkCover Authority (VWA) issued an *Occupational Hygiene Reports* guideline in 1999, which was re-issued with little change by the Australian Institute of Occupational Hygienists (AIOH) in June 2006. This paper introduces a consultation draft of a revised and expanded document for comment by members. The draft includes ideas for good writing and suggested writing techniques in a range of formats. While encouraging clear and grammatical writing with a logical outline it emphasises the most important element of any report; the professional judgement of the writer.

2. INTRODUCTION

2.1 Occupational Hygiene Reports

Occupational hygienists must clearly communicate their findings in written form in order to achieve improved health, safety and/or environmental performance. The challenge is to present pertinent and accurate technical information with sufficient detail in a form that can meet the demands of professional review but still be readily understood by a non-technical audience. Potential readers of any single occupational hygiene report include business owners, business managers, employees, trade union officials, government officials, other health professionals, engineering professionals and legal professionals.

2.2 Victorian WorkCover Authority Guidance Note

In order to promote effective communication by occupational hygienists the Victorian WorkCover Authority (VWA) issued an *Occupational Hygiene Reports* guideline in 1999. The guidance note was limited to common contents that should be included in occupational hygiene reports, but did not attempt to prescribing a specific format.

2.3 Australian Institute of Occupational Hygienists (AIOH) Guideline

The VWA guideline was re-issued with little change by the Australian Institute of Occupational Hygienists (AIOH) in June 2006 as the *Guideline for Writing Occupational Hygiene Reports* (the guideline). In my view, the adoption of a government guideline without adequate consultation was not an appropriate course of action for the AIOH because the guideline has been prepared from the perspective of a government regulator and not from the perspectives of members of the institute.

3. METHODS

This paper presents the historical development of the current Australian Institute of Occupational Hygienists (AIOH) proposed *Guideline for Writing Occupational Hygiene Reports* and presents some suggested content.

4. RESULTS

4.1 Overview

Occupational hygiene results are often poorly reported. A document that helps members improve their writing skills can make a valuable contribution to the profession and be of assistance to employers, workers and other stakeholders.

4.2 Critique of Current Guidelines

The current guideline has some excellent advice, such as *reports should be written in plain English, answer the question(s) raised at the beginning and satisfy the technical reader that the work was properly conducted and appropriate conclusions drawn*. In addition, it suggests that reports should be dated, have a title that summarises the nature of the work, identify the company that commissioned the work, identify the person/s conducting the work, include an executive summary if long, explain why the report was needed, identify the date of any site visits, describe the process/es that are the subject of the report, include details of methodology, present results that are traceable to field notes, draw conclusions about exposure and make recommendations about control measures.

The reports envisaged by the current guideline have numerous unnecessary characteristics. It assumes that reports are commissioned from consultants by companies, must report more than results, need a title page, must be signed, are large if more than 7 pages, are based on a site visit, need information on control measures to comply with the scope and need to reference regulatory requirements. It conflates measurements with methods, results with discussion and conclusions with recommendations, which can lead to ambiguous reporting.

4.3 Omissions

The current guideline applies only to standard reports and provides little or no advice for many occupational hygiene communications such as field notes, result notices, verbal presentations, expert reports and conference papers. It provides little guidance on the process of writing or English usage, there are many good references available (Burchfield, 1996; Collins, 2003; Department of Finance and Administration, 2002). Although claiming not to prescribe a specific report format it effectively prescribes other content without sufficient consideration of the legal impact on members. Despite this, the organisation disclaims liability for itself (*the AIOH extends no warranties as to the suitability of the information for your specific circumstances*).

4.4 Report Outlines

Preparing an outline first is a powerful writing tool. The outline in the current guideline is a direct adaptation of the format of a scientific paper, but this does not acknowledge a different legal and commercial context for occupational hygiene reports. A set of expanded headings with different approaches depending on the purpose of the document is presented below. The final format of any report is a matter for the professional judgement of the author.

Suggested Report Outlines		
Current Guideline (All Reports)	Proposed Guideline (Standard Reports)	Proposed Guideline (Legal Reports)
Title Page Executive Summary Introduction Process Description Methods & Measurements Results & Discussion Conclusions & Recommendations	Executive Summary Introduction Work Authorisation Scope of Works Methodology Limitations Credentials of Writer/s Process Description Potential Health Effects Relevant Exposure Standards Relevant Legislation Relevant Codes & Guidelines Control Measures Results Discussion of Results Conclusion/s Bibliography Signature & Date	Work Authorisation Assumptions Documents Provided Acknowledge Code of Conduct Credentials of Writer Methodology Limitations Defendant & Plaintiff Work History Description of Incident/Illness Relevant Statutory Duties Relevant Codes & Guidelines Details of Exposure Causation Foreseeability Preventability Conclusions References Signature & Date

5. DISCUSSION

5.1 AIOH Action

The AIOH Council has authorised a revised guideline for occupational hygiene reports and a consultation draft has been issued for comment. It encourages clear and grammatical writing with a logical outline and emphasises the most important element of any report, the professional judgement of the author. Members are encouraged to review the document, pass on their views and/or make use of the advice.

5.2 Professional Action

Most occupational and industrial hygiene texts (including the one sponsored by the AIOH) focus on technical issues to the detriment of communication issues. Some small exceptions include *The Occupational Environment-Its Evaluation and Control* by the AIHA (McDonald, 1997) and *Patty's Industrial Hygiene and Toxicology* (Halley & Wolkonsky, 1978). The same can be said about AIOH conferences. More professional emphasis on communication can only help the profession, as this step is often the weakest link in the important activity of risk assessment.

5.3 Government Action

The Victorian WorkCover Authority (VWA) deserves credit for the issue of the original document, but a guideline alone is not sufficient. The VWA and other authorities would do well to undertake activities aimed at improving the quality of professional advice.

6. CONCLUSIONS

Occupational hygienists must clearly communicate their findings in written form in order to achieve improved health, safety and/or environmental performance. A consultation draft of a communication guideline has been issued for comment.

7. REFERENCES

AIOH (2007) *Guideline for Writing Occupational Hygiene Reports* Australian Institute of Occupational Hygienists, Tullamarine, Victoria

Burchfield RW (1996) *Fowler's Modern English Usage*, Revised Third Edition [Oxford University Press; Oxford]

Collins (2003) *Australian Thesaurus*, Second Edition, HarperCollins Publishers, Pymble, New South Wales

Department of Finance and Administration (2002) *Style Manual for Authors, Editors and Publishers*, Sixth Edition, John Wiley & Sons Australia, Brisbane Queensland

Halley PD & Wolkonsky P (1978) *Industrial Hygiene Records and Reports* in Clayton GD and Clayton FE *Patty's Industrial Hygiene and Toxicology*, Third Edition, Wiley, New York

McDonald SM (1997) *Report Writing* in DiNardi SR (editor) *The Occupational Environment-Its Evaluation and Control*, AIHA Press, Fairfax Virginia

Victorian WorkCover Authority *Occupational Hygiene Reports* October 1999

INVESTIGATION OF A 'BACKYARD' RESPIRATOR FILTER MANUFACTURER

Susan McGurty, M.SC.(A), MAIOH, COH

WorkSafe victoria

Abstract

WorkSafe Victoria became aware of a Victorian manufacturer of aftermarket respirator filters through a field intervention program in the spray painting industry. Inspectors came across these filters being used with competitor's respirator face pieces. The markings on the filters did not indicate the face piece they were to be used with, nor did they have any Australian Standard markings. The Occupational Hygiene Unit carried out prevention activity seeking the manufacturers' compliance with the Occupational Health and Safety Act 1985/2004, with reference to the Australian Standard AS/NZS 1716:2003 Respiratory protective devices. Compliance was not achieved and the manufacturer was successfully prosecuted with a resulting fine of \$27,000.

Introduction

Programmed activity focussing on particular industries or hazards is routinely carried out by the WorkSafe Victoria Health and Safety Inspectorate. As part of the Spray Painting Program, Panel Shops were targeted for inspection. An inspection checklist was prepared by the Occupational Hygiene Unit and briefings were provided on the more technical components of the program. One of the points in the checklist was in relation to ensuring that appropriate respiratory protection is provided to employees (meeting the Australian Standard AS/NZS 1716 Respiratory protective devices).

Inspectors came across the aftermarket filters (without Australian Standard markings) being worn with a range of competitors face pieces. Compliance tools were used to ensure that the employers provided appropriate respiratory protection while carrying out spray painting activities. The aftermarket filter manufacturer subsequently wrote to WorkSafe complaining that his product was being slandered and if WorkSafe was to continue with this activity he was going to sue. The matter was referred to the Occupational Hygiene Unit for further action.

Discussion

The Occupational Hygienist met with the aftermarket filter manufacturer and determined that the filters did not have any markings to indicate which face piece they were designed to be used with. Furthermore, there was no evidence that the filters had been tested to AS/NZS 1716 with the designated face pieces. The filters were marked as Class A1P2 filters (suitable for protection against certain organic vapours with a boiling point greater than 65°C and for thermally generated particulates) and it was claimed that the filters have been tested to AS/NZS 1716. The manufacturer stated that the company had previously been issued with a Supreme Court injunction preventing it from promoting the filters with the competitors face pieces.

At about the same time, a complaint was received by WorkSafe Victoria from the competitor who instigated the Supreme Court proceedings regarding the aftermarket filters. The complaint included a test report indicating that the aftermarket filters dramatically failed the performance criteria as a Class A1P2 respirator filter. The test report showed that organic vapour breakthrough of the filter occurred after 2 minutes, with the required standard being 70 minutes.

WorkSafe Victoria subsequently arranged for the aftermarket filters to be tested by TestSafe Australia to AS/NZS1716. The filters were selected from workplaces that previously used or sold the filters. The resultant tests indicated that the filters passed as Class A1P1 filters (providing protection against certain organic vapours with a boiling point greater than 65°C and mechanically generated particulates), not the Class A1P2 rating as claimed. Furthermore, there were deficiencies in the labelling and packaging markings, and the accompanying use instruction sheet.

In December 2004, three Improvement Notices were issued to the manufacturer of the aftermarket filters under the Occupational Health and Safety Act 1985. The Improvement Notices required the manufacturer to:

- provide written information detailing the brand, type and model number of the designated face pieces that the aftermarket filters were to be used with, to all its Victorian customers;
- provide adequate information on the label, packaging and use instruction sheet about the safe use of the aftermarket filters with the designated face pieces (with reference to section 12 of AS/NZS1716:2003);
- have the aftermarket filters tested with designated face pieces to AS/NZS1716.

The aftermarket filter manufacturer failed to comply with the first two improvement notices after numerous attempts. The fundamental difficulty being that the filter manufacturer did not have an arrangement with the manufacturers of the face pieces to allow them to be informed of any changes to the face piece that may affect the performance of the respiratory protective device system. Furthermore, there were many marking and instruction deficiencies that were not able to be resolved due to the fact that the aftermarket manufacturer had a limited knowledge and understanding of the requirements of the Australian Standard and the importance of ensuring the health, safety and welfare of the consumers.

Two months after the compliance due date, the manufacturer completed the testing of the aftermarket filters with the competitors face pieces (to comply with the third improvement notice). The test report indicated that the filters (selected by the aftermarket filter manufacturer) and the two face pieces from different manufacturers passed the performance requirements as a Class A1P1 respiratory protective device system.

In April 2005, WorkSafe Victoria issued an Alert detailing the requirement for sufficient information to be provided on the safe use of respiratory protective device systems including the need to use the same brand of face piece and filter (not to mix and match brands).

A further improvement notice was issued to the aftermarket filter manufacturer to inform all users of the filters of the change in the filter rating from a Class A1P2 to a Class A1P1. This improvement notice was complied with. The drop in rating from a P2 to a P1 filter was not explained by the filter manufacturer.

In June 2005, another improvement notice was issued on the filter manufacturer to develop and implement a quality assurance system to ensure ongoing product integrity to AS/NZS1716. The filter manufacturer engaged a product certification company with the view to obtaining product certification for the manufacturer of the aftermarket filters and compliance with the improvement notice. The filter manufacturer did not obtain product certification but provided falsified documents to WorkSafe Victoria in an attempt to demonstrate that certification had been obtained. This improvement notice was not complied with.

Conclusion

WorkSafe Victoria issued proceedings against the company manufacturing the filters for failure to comply with the four improvement notices. A further charge was issued against the director of the company who provided the Inspector with false information in regard to compliance with the quality assurance improvement notice.

The matters were successfully prosecuted. The manufacturer pleaded guilty to the four charges of failing to comply with an Improvement Notice, resulting in the company being convicted and fined \$19,500 plus costs. The director was found guilty of providing false information to the Inspector, and was convicted and fined \$7,500 plus costs.

INCIDENT AND INJURY FREE (IIF) PROGRAM A MIND GAME PROGRAM TO ACHIEVE EXCELLENCE

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ABSTRACT

One of the biggest challenges facing any Occupational Hygienist is to ensure worker's compliance with the control requirements in order to minimize exposure to various physical, biological and chemical hazards. Sadly, often their hard work does not pay off, due to psychology of the workplace and workers, including over confident workers or the workers that choose to do things, their own way and the way they have done it for many years. This is why Psychological hazard is regarded as the fourth hazard on any worksite and IIF program was chosen as the preferred tool to control this hazard.

The project in which the IIF Program is being implemented is a joint venture between Shell, ConocoPhillips and Qatar Petroleum. This joint venture is to build two of the largest and state of the art LNG procession trains in the world. Currently there are in excess of 14,000 workers on site and it is anticipated that the man power picks to above 20,000. Taking into account the geographic environment of the Gulf, where temperature during the summer months can soar to near 50 degree C and humidity in excess of 85%, one can only imagine the size of the challenges facing the occupational health and safety professionals. Combine this with the risks associated with management of change for a very dynamic work environment and multicultural workforce from 15 different nations and languages, with totally different attitude toward health and safety and one would begin to understand the gigantic scale of the challenge.

It was, however the decision of the senior management that an Incident and Injury Free culture was needed to support the slogan of "every body goes home safe everyday!".

To initiate the program it was imperative to create a common denominator and a thinking platform for all workers irrespective of their background, education, experience, rank or education. To shape this and with the support of professional consultants, three tier educational platforms were formulated to provide training and expand on the abilities of the employees in the three layers of workforce, namely: workers, supervisors and management. This was complemented with two layers of Train the Trainer programs for different groups of workers.

Trainings are being delivered in many different languages, the content of the trainings were selectively chosen and tailor made to the audience. Whilst the project as a whole is well integrated, the material content for each level of training was designed to give the participants the skills needed to demonstrate "care and concern" for one's self and others in the work environment. One can never count the incidents that were prevented or measure the exposures that did not take place or the lives that were not affected, but one thing is sure, IIF has had a profound impact in the way workers interact with their work and home environment. Based on the structured formal and informal feedbacks from the workers, IIF is a technique that no project can afford to be without.

This paper will discuss the structure, details, content and challenges of this monumental undertaking and hopes to raise the awareness that; it is only after creating the right mindset, that other efforts of the occupational health and safety professionals will bear fruit.

SAFER WORKPLACES WITH IMPLEMENTED DRUG- AND ALCOHOL POLICIES IN THE GENERAL INDUSTRY

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Abstract

Besides the possibility of being exposed to toxic chemicals in the workplace environment there are quite a number of other, personal parameters, which may have an impact on the safety of a worker. This can be the influence of drugs and/or alcohol or simply fatigue. Implemented drug- and alcohol policies with random tests of the employees would help to create and maintain discipline in order to reduce or eliminate impairment by drugs and alcohol and thus to improve workplace safety. Test equipment should be easy to use, reliable and convenient as well as cost effective. They should have the possibility for documentation of the results.

Introduction

Safety in the workplace is no longer limited to the classical risk assessment regarding chemicals, noise, and heat stress, personal parameters like alcohol or drug consumption are getting more and more focus and importance, not only in industrialised but also developing countries. Companies are planning and implementing their drug- and alcohol policies, which usually imposes random tests of the employees, in many cases the chance for each individual to undergo at least one test per year is very likely. Consequences after positive results will vary of course from company to company, but they must be clear for the employees.

In order to achieve good acceptance the test equipment should provide direct reading onsite with some degree of documentation or evidence. Needless to say that test results should be available for both drugs and alcohol without using any invasive methods which would dramatically increase cost.

Measurement of alcohol

Many instruments and technologies are available for the measurement of alcohol based on breath tests, different types of sensors allow for fast and specific measurements in the field. However, for the professional application with certain consequences after positive results semiconductor based sensors should be avoided due to cross sensitivities to other substances which would lead to high readings. One of the most common cross sensitivities with a too high reading is carbon monoxide, which is very common among smokers. A cost effective breath alcohol tester would be a handheld unit with LC-display and electrochemical or fuel cell sensor. The sensor itself is fairly specific to ethanol, other substances which could be expected in the breath of human beings usually are not indicated, even not acetone, which can be found in low concentrations as a consequence of diabetes. Calibration of the instrument is recommended every six months, an activity which is user friendly with so-called dry gas in a small cylinder or wet-gas with a simulator in an aqueous solution.



Fig. 1: Alcotest instrument for checking breath samples

The units are usually available with a built-in data logger to store the measurement results internally and also have features for printing and downloading to a computer. For downloading the data a special software is required from the manufacturer of the alcohol tester, raw data can be exported to e.g. Microsoft Excel, printed, individualised, and stored.

Date	Day	Time	Personnel Number	Test Result	BAC	Device Model	Serial Number:	Test No.
07.08.2007	Tuesday	23:33:29	1234567890	High	1,54	A6810	ARYH-0006	37
07.08.2007	Tuesday	23:30:20		High	2,51	A6810	ARYH-0006	36
07.08.2007	Tuesday	23:29:26		Alert	0,42	A6810	ARYH-0006	35
07.08.2007	Tuesday	23:02:39		Alcohol	2,31	A6810	ARYH-0006	34
07.08.2007	Tuesday	22:56:46		Alert	0,48	A6810	ARYH-0006	33
07.08.2007	Tuesday	22:13:30		Fail	0,56	A6810	ARYH-0006	32

Fig. 2: Test results obtained from the breath alcohol tester

The table above shows an example of a series of data from measurements, which should be organised on a confidential level, as we are talking about sensitive data. The personnel number above is just a random example.

Measurement of drugs of abuse

Similar to the measurement of alcohol we would like to have convenient methods for testing drugs after consumption, however, from a workplace safety point of view it is not the objective to determine that an individual has consumed drugs at any time in the past, but to avoid that somebody goes to work impaired by drugs immediately after consumption. Unlike alcohol there is no way to measure drugs in the breath of human beings as the vapour pressure of drugs is by far too low to be determined in the gaseous phase. Therefore we need different human media for drug testing and also a different detection technology. The most common method so far is based on immunoassays and urine as media.

Unfortunately there are some unavoidable disadvantages in conjunction with urine tests, we need at least a toilet and a person as a witness to proof that the urine sample comes from the right individual, which is intrusive of course. A good alternative would be saliva or oral fluid as a human sample medium. It reacts even faster to drugs of abuse and is significantly more convenient than getting urine samples. Tests with direct reading are based on immunoassays and are available in two different versions, one with subsequent visual evaluation, the other one goes into a reader, where the results are visible in a display and can also be documented as a print out or evidence.



Fig. 3: Saliva test as a six drug panel for amphetamine, opiate, cocaine, methamphetamine, PCP, and cannabis

Each test card is individually packaged and allows for testing up to 6 drugs at the same time. As a further safety feature it contains a control layer to make sure that only valid tests are taken into consideration for evaluation. Pictures can be taken from the used card as the proof of evidence.

Additionally each saliva sample can be stored in a deep freezer for up to 12 months for any subsequent laboratory analysis with a GC/MS without taking a second sample at a different point in time.

Cut-off concentrations for the drugs in saliva are as follows:

Drug	Cut-off concentration ng/mL
Cocaine, COC	20
Opiate, OPI	40
Amphetamine, AMP	50
Methamphetamine, MET	50
Phencyclidine, PCP	10
Cannabis, THC	50

A more advanced method for the measurement of drugs would be a system based on saliva as well, but with subsequent application of a reader for storing the data, displaying the results and printing the data.



Fig. 4: DrugTest instrument for displaying the results and for print out

Drugtests with direct readings utilizing saliva samples are regarded as drug screeners, results are obtained as a go/no go result around the cut-off concentration without any further differentiation of individual concentrations. Main application is to determine impaired people, so that subsequent laboratory tests are only required in case of a positive result, which is a substantial cost-saving approach. High specificity of immunoassays of saliva tests are again cost saving, as they show a significantly improved specificity compared with urine tests minimizing the most critical false positive results.

Summary

In addition to the exposure to toxic or explosive chemicals we put more and more effort into minimizing certain personal parameters like drug- or alcohol consumption prior to work in order to further enhance the overall safety at the workplace. Drug- and alcohol policies in the general industry lead to a stable discipline in order to avoid having people being impaired at their job. Direct reading measurement devices, which are convenient to use and cost-effective play a major role in maintaining this discipline among the employees.

DO I BELIEVE WHAT I TEACH?

Dr David Goddard

Department of Epidemiology & Preventive Medicine at Monash University, Melbourne

David Goddard is a University teacher and occupational physician with special interests in occupational hygiene and toxicology. These interests developed from his earlier work as a government medical inspector of workplaces.

He has taught medical undergraduates and post-graduate students in public health and occupational health at Monash University since 1990, something he loves to do. In 2001, he was successfully nominated by his undergraduate students for the Vice-Chancellor's Award for Distinguished Teaching.

My purpose in being here

I am a medically-trained educator. I love this role because the subject matter is important to my students and our community, because it is ever interesting, and because to teach effectively is such an ongoing challenge.

Part of *your* role as occupational hygienists is likely to be to influence others to prevent harm to the health of people at work. Perhaps there are things that I have found in my 18 years as a full-time educator that could assist you in that role. That is my hope in offering this.

Information is abundant and accessible, so a prime role of education is to assist students to sift what is likely to be true from what is fanciful. Yet the 'gauge of the sieve' is determined by the person holding it and –

- there are many uncertainties, particularly about cause and effect;
- knowledge about prevention is always seen through the prism of sectional interest yet, without those sectional interests, little gets done;
- description of exposure deals in numbers, and many, *many* people, when presented with numbers, experience feelings that extend from mild discomfiture up to sheer dread. Anxiety impedes learning, sometimes totally.

In this presentation, I use examples drawn from my experience to invite you to reflect on how this affects learning and teaching about occupational health.

Teaching, why, what?

I teach to bring about some intended or targeted change in a student's capabilities. However, what is *taught* (as distinct from what is taken in by students from other sources) forms just a part of what is learnt – and sometimes quite a small part. Using analogy is always risky, but I liken a learning environment to an electric field – a space where charged particles accelerate in a particular direction. Those entering will be charged with expectations. The field created to assist learning co-exists with other fields that are created by, for example, the charges of other entrants, and fields generated by moral imperatives, sports or other special interests. These can all affect the pace and direction of learning and the component of any accomplishment that is directed toward a teacher's ends.

A good teacher may act like an electrical conductor – to facilitate and target the effects of the field. The allure of an episode of teaching springs from relevant, well-designed learning materials and a

teacher who, with good humour, offers emphasis, meaning, and apt links to other relevant areas of knowledge.

What is belief (and what are some of mine)?

Belief can be *yes* or *no*, but belief in a social movement or a system of thinking is nearly always a matter of degree. So-called belief in the operation of a law may amount to little more than hope; so-called belief in the harmful effects of, say, alternating magnetic fields, may simply be acceptance – a working hypothesis – without serious commitment to the idea of cause and effect. [One may *accept* that a lottery ticket will not win without *believing* it will not.]

As a teacher, belief comes into the extent to which I select material for emphasis, and the energy with which I strive to persuade a student to my view. I want my students to make decisions that serve our community well, and when those students later join my profession, I want to respect their integrity, even though I may not fully share their views.

Some examples of my beliefs about substances at work are:

- Our understanding of the effects of substances is assisted by seeing similarities and patterns when we compare substances, and by knowing the particular susceptibilities of target cells, organs and body systems.
- Clear talk about specific health *risks* from substances requires reference to level of exposure. Statements without reference to quantity can be useful to classify or compare. However, given a situation where a substance is in use, then talking about the likely effects of that substance without telling the corresponding level of exposure can be seriously misleading. Avoid letting *hazard* masquerade as *risk*.
- When referring to risks that are attenuated rather than removed, the term *safe* means of ‘acceptable risk’. Acceptability is like beauty – in the eyes of the beholder. Not all eyes will be in accord.
- If people are exposed to a substance in a context of fear, then any symptoms may relate either to the substance or to the fear.
- The media are like a digital hearing aid – they selectively amplify.
- The operation of cause-based compensation schemes can be reliable and fair only where we can determine cause. That is hard to achieve when the alleged harmful exposures happened long ago. Court outcomes are then less certain.

What sort of questions tax my belief in what I teach?

- When is an algorithm good enough? When should I urge students to go further than a simple recitation of today’s practice and demand that they lend energy to asking “Why?” and “What if ...?”
- When a sincere and earnest student speaks more from the heart than the mind, when do I remain silent – effectively an endorsement – and when do I put hard and time-consuming thought into airing his or her fallacies kindly.
- How do I assist my students to draw coherent priorities in prevention from the scatter of outcomes flowing from common law action?
- At what point does the homage paid to celebrity hazards, such as asbestos, start to incur excessive opportunity costs in other health areas?

Where am I *most* uncertain?

Two areas where I face the greatest uncertainties are:

- When does a gathering of symptoms become a diagnostic entity?
- What extent of evidence do we require before we fairly accuse some substance or ray of being a cause of a chronic health effect such as cancer?

I shall take these in turn, then mention a few other issues that enter my classrooms.

What constitutes a diagnostic entity?

Once we give a something a name, we tend to think it exists as an entity – but does it? For a medical practitioner, the central reason for making a diagnosis is to initiate and sustain an apt therapeutic intervention (if required). For the person affected, the acceptance of this diagnosis situates them on a path with a map to guide their short-term future and perhaps their destination. Commonly, too, if the condition is severe or the period of care is likely to be prolonged, the person will obtain an ‘events guide’ that addresses social and financial needs such as contact with fellow-sufferers, alternative modes of treatment, and avenues of financial support including compensation.

The form of a diagnosis is most useful when it addresses the part(s) of the body affected and the nature of the pathological process that is occurring there. But sometimes this level of precision cannot be achieved – the process is somewhat obscure. The term *syndrome* is commonly applied to a disease entity whose mechanism of development is veiled to our present knowledge.¹

Whereas past medical practice has ascribed the term *syndrome* to a cluster of signs and symptoms arising from a common – albeit indistinct – pathology, there is now a trend to use the term *syndrome* to describe a social situation with a mix of pathologies but an alleged common cause, e.g. *sick building syndrome*. Such a designation provides the ‘events guide’ without the map; there are things to do but no well-defined way to go. Nominating a causally-defined syndrome as an explanation of a person’s ills affords that person membership of a particular social group – a group that commonly acts to take preventive, legal or political action.

Multiple chemical sensitivity – a predicament for both patient and doctor

Some people claim that a chemical exposure (or exposures) at work has triggered a lasting effect on their well-being and diminished their ability to tolerate other common chemical exposures in their work and home life. As a result, they appear to experience symptoms with exposure to multiple common chemicals – almost a feeling of ‘abuse’ by the constituents of the air they breathe, or the water or food they consume. Sometimes, guided by a health adviser, they will flee urban life to live in isolation. Commonly, they have:

- a spray of symptoms without clear pathogenesis that are provoked by exposure to chemical accompaniments to urban life;
- concomitant phobia or other anxiety-related disorder that clouds attribution of cause.

These unfortunate people often have strong views, but also feel curiously vulnerable because few of their health attendants appear to understand (or want to understand) what ails them. They sense scorn or impatience from practitioners of conventional medicine and can feel hopeless or hostile.

¹ It may be that in the past the pathological mechanism was unknown, although now it is clearer, e.g. Down syndrome. However, the term *syndrome* is retained because it is inappropriate to refer to a lifelong condition of altered health as a disease.

There is no doubt that most of these people are suffering – their symptoms clearly dominate much of their lives. However, for these people, the shorthand language that we in the medical profession call “a diagnosis” seriously lacks an ability to encompass the situation. If a person has earlier sought health advice from many sources, and especially if this has drawn him or her to change lifestyle, then what he or she has is not a diagnosis but a *way of living* to which there are some medical aspects. A doctor or other health practitioner attending such a patient risks being, as in the Indian parable, like the blind men with the elephant. Parts may be characterised rather than the enormous whole – some will say the main issue is immunological, others toxicological, others psychological – all maybe part-truths [1].

So, *multiple chemical sensitivity* (or, if you prefer, *idiopathic environmental intolerance*) enters my teaching, but do I believe it is a diagnostic entity?

I think that essentially the term offers greater social purpose than medical illumination. If multiple chemical sensitivity were to be more generally recognised as an entity, there would be a wider swag of doctors willing to offer treatment, together with access to compensation to fund long periods of treatment and time away from work. I simply encourage medical students and doctors who venture here to bear in mind the ethic of non-maleficence: “But first, do no harm” – and to always act in what they believe to be the best interests of their patients.

What extent of evidence do we require before we fairly accuse some substance or ray of being a cause of a chronic health effect?

I raise this not to answer it, but to talk through some of the challenges to my beliefs that come from teaching these things. A good example is occupational cancer.

Occupational cancer

There are two issues here:

- What is *occupational* cancer?
- By how much must exposure be attenuated to make the risk so low that it can be, for practical purposes, ignored?

Reliably establishing the causes of any one cancer-type is very difficult because cancer proceeds from a combination of events. These events occur over a period of years or decades, and causal factors seldom lay their fingerprint on the cancer histology. So there is dispute about what cancers are occupational and what are not. Protagonists bring their individual values to address this uncertainty; and key decision-makers, such as courts or standards-setting authorities, have different perspectives depending whether their main preoccupation is humanitarian care for the afflicted or practical prevention of injury and disease.

Further, there is no comprehensive information on the type and intensity of occupational *exposures* to known and suspected human carcinogens in Australia. Some overseas countries do better, e.g. Finland. However, constant and rapid technological changes to workplaces make nigh impossible the task of finding out in broad-scale who is exposed to what. This lack of exposure data adds uncertainty to the tally of cancers characterised as occupational. And, one area of enduring dispute is the degree to which exposure to a carcinogen must be lowered before the risk of exposure becomes too small to be of concern [2].

In may 2007, various Australian media suggested that a cluster of breast cancers in a radio studio in Brisbane may have been due to non-ionising radiation; but it is usually very difficult to find the true cause of disease clusters – as it has been so far there.

Apart from the ultraviolet rays of the sun on the skin of outdoor workers, it is likely that *asbestos* is the most significant occupational carcinogen, and mesothelioma is its most spoken-of legacy. Yet *how low* must exposure be before the added risk of mesothelioma becomes negligible. Compensation has been received by workers with mesothelioma whose past asbestos exposure has appeared to be minor. Of course, it is impossible to tell in retrospect whether the minor asbestos exposure was the culprit and equally impossible to prove that it wasn't. The courts, faced with this evidential vacuum, tend toward humanitarian expedient; the reality is we simply don't know.

Epidemiology can never clarify the lower limits of risk because, in trying to tell the difference between health outcomes with *minimal* exposure and health outcomes with *no* exposure, the epidemiologist always faces an unforgiving mathematical operation – dividing by the square root of n . Where n , the number of cases is small, as inevitably it is when exposures are low, it becomes impossible to show that minimal exposure differs in any health-related way from no exposure.²

Asbestos is a 'celebrity' hazard. Years of adverse publicity has given it horror symbolism, so that many frightened people consider that disaster starts with one fibre – as if having a tiny bit of asbestos is like having a tiny bit of mesothelioma. Because we have no good evidence for what happens at low levels of exposure, asbestos tends to be treated in a qualitative way, i.e. "better out than in", rather than the more quantitative approach of lowering the exposure level. Measurements of asbestos in air are done but results are essentially treated on a dichotomous basis – below the level of detection or above it. The trend is to eliminate asbestos from as many places as possible.

So, do I believe what I teach about asbestos?

Part of respecting my students' rights and intellects is to encourage them to look at a problem from several viewpoints. Yet few people have muted views about asbestos; statements such as "One fibre can kill you" are made stridently, indignantly, and with strong moral overtones. In the face of those sentiments, to argue for the reasonable cost of a necessary intervention – that more health benefits could be gained by spending preventive dollars elsewhere than on removing the last skerrick of asbestos can be about as popular as speaking on the virtues of paedophilia!

Such attitudes can be selective. I have heard an occupational health worker in the petroleum industry argue (essentially) that one fibre can kill. He was far less emphatic when he was asked whether one molecule of benzene could do the same.

So in a morally-charged environment, is it possible to define a middle ground – before striving becomes obsession?

What is normal? What is mad?

Six or seven years ago, when my daughter was in her mid-teens, she asked accusingly: "Why can't we be a *normal* family?" At the time, our family was made up of two parents, Ruth and I, who loved each other and our two children, a boy and a girl. We were all in good health and we lived in middle-class Melbourne. Our children went to the local high school where they were encouraged in learning, sport and social activities. On many weekends we would all go orienteering. Up until then, no-one had ever claimed that our lives were other than normal, but then we came to realise that *normal people* are those you don't know very well.

² The problem is that P-values or confidence intervals all come from fractions that have in their denominator the square root of n . There are two ways to make the magnitude of a fraction bigger – either increase the size of the numerator or decrease the size of the denominator. As n (in the denominator) gets smaller, so the P-value gets bigger or the confidence interval gets wider. This makes it hard to show that an observed difference is due to anything more than chance. Thus, epidemiology loses its potency in this region of low-level exposures.

The *lunatic fringe* is a fearful or dismissive term I sometimes hear applied to those who hold extreme views on a particular set of issues, e.g. environmental issues, by those who consider themselves to have mainstream or middle-of-the-road views. The extremeness is fuelled by a passion that mainstream commentators consider to be out of proportion to the evidence of risk.

There was a time when the term *lunatic* was applied to the mentally ill, i.e. to those whose aberrant behaviour was involuntary. Nowadays, the epithet refers to those whose behaviour is *voluntarily* aberrant to the extent of being dangerous [e.g. lunatic drivers who cross double white lines on a curve or the crest of a hill]. So, in the context of ‘lunatic fringe’, the adjective ‘lunatic’ underlines that a very radical change to society is, to some degree, a leap in the dark, a risky irrational step. Reference to ‘fringe’ suggests that people on the extremes (right or left) have more in common with one another than with those in the middle, i.e. that the full continuum of social actions on an issue is represented better by, say, a steep parabola than by a straight line, left to right. Leaders on the ‘fringe’ often strive to have their ideas accepted as mainstream, sometimes with success.

So, what do I teach? The realm of normalcy possesses some inertia but, across the years, the location of that realm is influenced – sometimes markedly – by action at the margins.

When hazard masquerades as risk

When they speak about hazardous substances in a workplace, it is common to hear reporters on current affairs programs argue or imply the following:

This workplace (or product) contains Substance X.
Substance X can cause a fatal health effect.
Therefore this workplace or product is harmful.

So what do I teach? I ask my students to substitute the term *dihydrogen oxide* – which indeed can drown, scald or freeze people – for *Substance X*! The problem with a hazard statement is that it is vacant – it needs to be tenanted by particulars such as the form and extent of exposure.

Propaganda

Propaganda is written to further a cause. Some hallmarks of propaganda about exposure and disease include:

- Reference to “goodies” and “baddies”, where the adversary is stripped of humanity by implying that they universally lack compassion and sensitivity, thus legitimising doing them harm;
- When something devastating, e.g. a high risk of cancer or birth defects, is proffered as a possibility yet with little attempt to circumscribe or quantify the risk – there is just enough information to unsettle people but not enough to let them see the limits of the problem or to feel reasonably in control;
- Gratuitous put-downs to heighten infamy, e.g. with substances – mixtures are called concoctions, storages are called dumps, and the adjective *toxic* is added simply for emphasis – like a condiment [3].

The message is part truth, part fantasy. Why is that a problem? After all, in occupational health and environmental health circles you will meet well-heeled, often well-regarded people who peddle speculation and fantasy with earnestness, or at least with a straight face. Some direct their interventions toward new (or trendy) syndromes; others favour chronic predicaments. They sell hope.

I teach about propaganda, so what do I believe? Whilst speculation and fantasy may be a *starting point* for the development of worthy ideas, it is just that – a starting point. If applied to current health policy it is very likely to make preventive efforts less focussed, less efficient. Preventive dollars are ever scarce and my ethical stance is that we must use them to reduce as much harm to health as we can. Even when we successfully fight to gain more dollars for prevention, we must still use the money efficiently lest we harm the reputation of health and safety which, in turn, stands to impede future money flow.

When relevance declines

Obviously, no teacher can tell his or her students everything that is relevant to a topic, so inevitably the information presented is selective. Part of a teacher's role is to emphasise some things and give mere passing reference to others.

Ideas or ways of writing have periods of vogue. Personalities have their time in the sun. At age 63, I can sense the afternoon shadows lengthening over my day in the occupational health marketplace. *You* will have colleagues that once you admired but whose decline now occasions impatience or embarrassment when rarely you meet.

Twice in the last decade, I have been given large manuscripts by elderly colleagues – one on lead, one on cochlear physics. They have in each case been presented to me as a high reach of their understanding, drawn from decades in the field. I have read and helped them edit these works but, sadly, they have almost no audience. Their carefully crafted work takes effort to follow – it's not a few easy bytes – and I have not urged my post-graduate students to read it. In this, I feel poignant betrayal.

Conclusion

So, do I believe what I teach?

I expect that some of my students will go on to lead opinion. For their judgment to be respected they must find ways to decide whether they will accept or reject new information. Acceptance means they must change some of their ways, sometimes at great cost; rejection necessitates defending that view against others' urgings.

I try to tell my students the truth. What I mean by truth is the way that facts are organised by those I respect within discipline areas, and by events that I have observed whose interpretation is corroborated by those I respect. I acknowledge that every objective fact must be consistent with all other objective facts and that every objective fact has some explanation. I, too, acknowledge that no-one is an expert on *subjective* matters.

Sometimes, the facts presented to us are too few or disparate to form a pattern (e.g. the evidence for whether a substance causes cancer). Sometimes we have too little basic knowledge even to recognise when a pattern exists. In situations where the truth cannot be pinpointed, it is valuable always to at least determine the approximate realm or domain of the truth. The application of common sense can be particularly valuable when things are uncertain or vague. The springboard of common sense is a knowledge of the limits of the normal behaviour of people and things. A sensible person may not know the actual place of her pet guinea pig in a schema of the animal kingdom, but she would know that it is more like a rodent than a feline and that it certainly isn't a type of snake.

A consistent seeking to define the limits of the realm of the truth will minimise the risk of my students making gaffes. With exposure to substance hazards, their tools in this task include the

fundamental principles taught in secondary school chemistry and physics – things such as the gas laws, vapour pressure, concentration, nomenclature, and the periodic table [4].

And, I hope that I show my students by example that good teaching is *not* wholly a commodity. Part of it is about what the teacher does and how he or she cares for the learning of his or her students.

References

[1] Harrison RJ. Ch 43 Multiple chemical sensitivity. In LaDou J ed. Current occupational and environmental medicine, 4th ed. New York: McGraw Hill Medical, 2007: 749 – 756.

[2] Benke G, Goddard D. Letter. Estimation of occupational cancer in Australia still needs local exposure data. Aust NZ J Pub Health 2006; 30(5): 485 – 486.

[3] Goddard D. MPH2022: Assessment and control of workplace hazards – unit guide. Churchill, Vic: Monash University, 2007: p. 8.26 to 8.28.

[4] *ibid.* p. 8.24 – 8.25.

MESOTHELIOMAS AND ASBESTOS EXPOSURE - HISTORICAL ASPECTS AND FUTURE PROJECTIONS

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Abstract

In this presentation the relationship between the incidence of mesothelioma and asbestos type is summarised. Then the relationship between the incidence and time since exposure is considered with an emphasis on the importance of including a term to take account of the gradual elimination of amphibole asbestos fibres from the lungs over time. These aspects are explored concentrating on the contributions of some of the researchers, including Drs John Gilson, Chris Wagner, Molly Newhouse and Stephen Jones, with whom the first author has been privileged to work. It is shown that making allowance for elimination explains the apparent discrepancy between animal experiments, in which chrysotile and amphibole produce similar numbers of mesotheliomas, and epidemiology where the incidence is much higher after exposure to amphibole asbestos than chrysotile. When making predictions of future mesothelioma numbers it is important to take into account the use of amphibole asbestos in the population, and the effect of elimination many decades after exposure.

Introduction

There are two possible reasons for undertaking projections of the number of future mesotheliomas – first to estimate the requirement for medical services, and second to estimate future liability for compensation. The mesothelioma rate after exposure to asbestos depends on the amount of exposure, the type of asbestos, and the time since exposure. In this paper we will concentrate on asbestos type and time since exposure, both of which are critical.

It is beyond the scope of this presentation to give a full review and we will concentrate on the contributions of some of those with whom the first author (GB) has been privileged to work.

Dr J. C. (Chris) Wagner (1923-2000) is well known for the identification of the link between crocidolite asbestos and mesothelioma in South Africa (Wagner, Sleggs and Marchand 1960). He described this as a “lucky break” but he had to have great insight to convert his luck into a major discovery. It may seem surprising that a diagnosis of mesothelioma was not recognised by the leading pathologists of the day. Indeed because of this Chris Wagner had difficulty in finding a journal willing to publish the paper – distinguished pathological referees were not impressed with this new tumour “mesothelioma”. So the first important contribution was to obtain recognition of “mesothelioma” as a distinct pathological entity, and secondly the discovery of the link with blue or crocidolite asbestos. Chris Wagner was always interested in the differences between fibres types. His father was a distinguished geologist and so he was well aware of the geological differences between asbestos types, and therefore saw no reason to assume that their health effects would necessarily be similar.

Dr John Gilson (1912-1989), who was the Director of the Medical Research Council’s Pneumoconiosis Research Unit in Penarth, Wales, developed objective ways of reading chest radiographs and developing international classification schemes, such as the UICC classification. He also had astute insights into epidemiology and the ‘big picture’, and he was one of the first to suggest that, of the three major commercial types of asbestos, crocidolite resulted in the highest rate of mesothelioma, with amosite next, and chrysotile much lower (Gilson 1966). Twenty years later this was generally accepted although even then it was sometimes ignored in important sources (US

Department of Labor 1983). Today it is doubtful if anyone would seriously challenge this conclusion.

As well as conducting their own research programs both John Gilson and Chris Wagner were very active in encouraging others to become involved and through their efforts many studies that have become well known were initiated. These include Dr Newhouse's studies of the Barking and Ferodo factories in England, and Stephen Jones's study of the women war-time gas-mask workers in Nottingham, England. They were also influential in encouraging the study of Canadian chrysotile miners which was carried out over many years by Corbett McDonald and many well known colleagues (see later).

Dr Muriel L. Newhouse (1912-2000), invariably known as Molly, has been described as a "Doyenne of Occupational Medicine" in a biographical article (Salerno and Feitshans 2004). She did not start her research career until she was in her early fifties. She had served in the Royal Army Medical Corps during the Second World War going into Europe soon after D-Day and working close to the combat zone, and later served in India and Singapore where she treated rescued prisoners of war. She was promoted to full Colonel, the highest rank then available to a female medical officer.

In what, according to Salerno, was her first published paper she described the occurrence of mesotheliomas after environmental exposure to asbestos – women who had laundered their husband's work clothes and those living close to a factory (Newhouse and Thompson 1965). This was an extremely important paper, as was recognised by its reprinting in 1993.

Asbestos type

Various studies have been carried out over the years providing evidence of mesotheliomas after exposure to each of the three commercial asbestos types. For crocidolite these include the Wittenoom workers and residents studied in Western Australia for the last 30 years or so, and the Nottingham gas mask workers studied by Stephen Jones. Both of these studies provide important information on the influence of time since exposure on mesothelioma rate which we will discuss later.

Many populations exposed to asbestos have been exposed to a mixture of types, amphibole and chrysotile. Most of these can provide no information to compare types, but there are exceptions including the Ferodo study of Molly Newhouse. At this factory friction products were made and chrysotile was almost the only asbestos used. But there was one contract that specified the use of crocidolite and this was carried out over two periods of time with the work done in one part of one of the workshops. There were 10 mesotheliomas and it was established that 8 of these had worked on the crocidolite contract. Forty controls, matched on year started work in the factory, were selected and only 3 of these 40 had worked on the crocidolite contract (Berry and Newhouse 1983). This provides strong evidence that crocidolite is more potent than chrysotile in producing mesothelioma.

Hodgson and Darnton (2000) carried out a quantitative meta analysis of mesothelioma and asbestos type (Table I). Crocidolite was estimated as 5 times more potent than amosite with chrysotile much lower, and not surprisingly mixed exposure intermediate.

Table I: Mesothelioma mortality by type of asbestos

Asbestos type	Total expected mortality (%) per f/ml-year*
Crocidolite	0.51
Amosite	0.10
Chrysotile	0.0010
Mixed (amphibole and chrysotile)	0.021

* adjusted for age at first exposure – derived from Table 1 of Hodgson & Darnton (2000)

Animal experiments

Chris Wagner carried out a number of animal experiments at the Pneumoconiosis Research Unit. One of the first showed similar mesothelioma rates for crocidolite and chrysotile (Wagner and Berry 1969). At first sight this seems in conflict with the epidemiology and at the time some argued that as animal experiments were in controlled conditions, whereas there are many uncontrolled factors in epidemiological studies, then the experimental results justified regarding the different fibre types as similar in effect.

The late Merle Stanton was an influential figure in animal experimentation and he set out the hypothesis of a “durable” fibrous shape as the key to effect (Stanton and Wrench 1972). So how do we take durability into account? Durability exerts its effect over time so we will now turn to the question of the relationship of mesothelioma incidence to time since exposure.

Elimination and time since exposure

The early classical model was that incidence increases with time to a power of 3 or 4, based on the multistage model of Armitage and Doll (1954). This model certainly fitted data from a number of studies covering a follow-up of 30 to 40 years after exposure (Doll and Peto 1985). The model is based on a model for continuous exposure and this was considered reasonable since once inhaled asbestos fibres remain in the lungs for many years or decades. So the lung is exposed continuously even though the actual exposure in the workplace may have finished many years ago. But if durability is relevant then it has to be brought into the model and this can be done by introducing an additional term representing exponential decay, corresponding to elimination of fibres from the lungs over time. The incidence at time T after the start of exposure is then given by

$$I(T) = c T^k e^{-L}$$

approximately, where k is a power, c is proportional to cumulative exposure, and L is the rate of elimination (Berry, 1991, 1999). The exposure in the body remains continuous but at a declining intensity over time. How large is the rate of elimination? There is now reasonably good evidence that it is of the order of 10% per year for crocidolite.

The effect of elimination is particularly important after about 35 years from exposure. The relationships, with and without elimination, are fairly similar over the first 35 years, but then diverge markedly. The model without elimination continues to increase but that with elimination flattens off and starts to decline. Whilst 10% seems a low rate of elimination its effect over many years is large, and when using a model for prediction into the future we will often be predicting for 40 or more years after exposure, and so the choice of model, that is the choice of elimination rate, is critical.

It seems fairly evident that durability will be more important in human studies than in animal experiments. This is because durability exerts its effects over long periods of time, 30 and 40 years plus following human exposure, compared with only 2 or 3 years for rats.

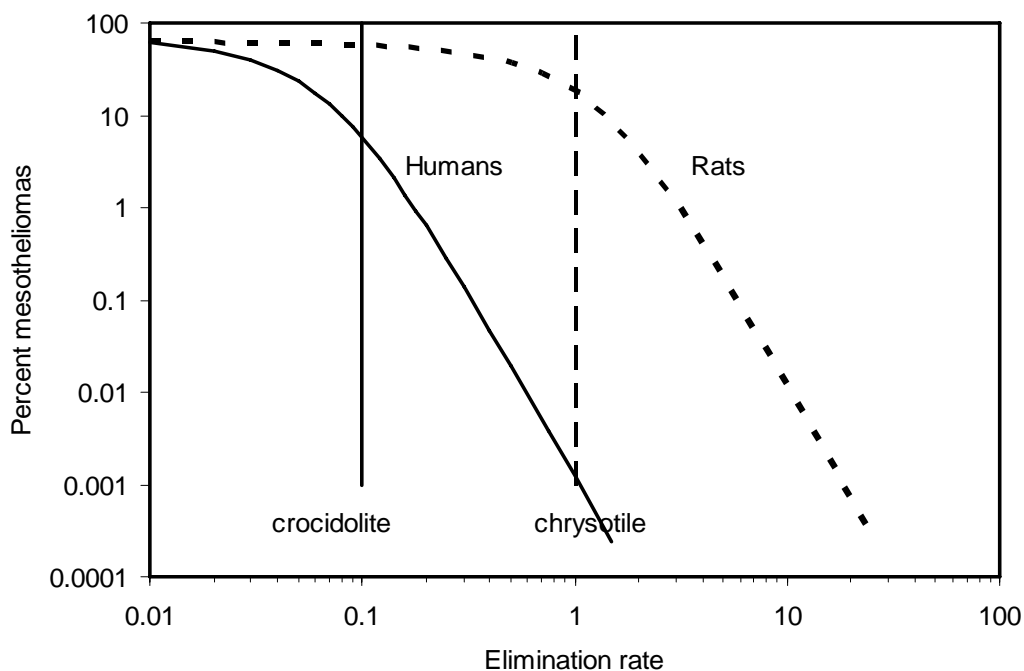


Figure 1: Mesothelioma rate by elimination rate for rats and humans

This is shown in Figure 1. The top dotted curve for rats shows a high fitted rate of mesothelioma up to an elimination rate of 1, which perhaps corresponds to chrysotile, and this rate is only slightly less than that for crocidolite with an elimination rate of 0.1 (that is 10% per year). In contrast after human exposure in the lower curve, the fitted mesothelioma rate is much lower for chrysotile than for crocidolite. Whilst the model is a simple one, and reality is undoubtedly more complicated, this does show that there is no conflict between the animal and epidemiological results, provided that durability is taken into account.

There are epidemiological results to support the elimination model, providing evidence that the incidence does not increase indefinitely as the third or fourth power of time after exposure. Dr Stephen Jones (1929-2000) collected cases of mesothelioma in Nottingham over thirty years and identified that many were in women who during the Second World War had worked in a factory that had been requisitioned to make gas masks using crocidolite asbestos (Jones et al. 1980). Shortly before his death he handed over the records to Corbett McDonald and this facilitated some further epidemiological study of this rich data source. There are now 65 mesotheliomas. The first occurred in 1963 and the most recent in 1994. The number of person-years at risk, taking into account the deaths that have occurred over the years, noting that the youngest member of the group must now be approaching 80 years of age, was established for each year of follow-up, and so the mesothelioma rate calculated for each calendar year. It was found that the mesothelioma rate was fairly stable between 30 and 50 years after exposure (McDonald et al. 2006). This provides strong evidence that the classical model requires the inclusion of an elimination term.

The second example comes from the Wittenoom miners and millers, but first we will interpose some general remarks on predictions.

Predicting future numbers of mesotheliomas

Predictions have been made in two situations. First of known workforces where one is dealing with a closed group of people, with information on exposure by date, duration and perhaps intensity. A model can be fitted to the existing data and the model then extrapolated into the future.

Secondly predictions are made of regional populations. Here the population is known only by size and age, not as individuals. There is no exposure information, not even what percentage of the population have been exposed to an appreciable degree. But there is information on the use of asbestos in the region over the years.

An example of the first situation is Wittenoom. Predictions were made using the data up to 1986 and a range of estimates made of the future numbers up to 2020 (de Klerk et al. 1989; Berry 1991). From a later follow-up it was possible to see how these predictions accorded with the data collected for the period 1987 to 2000 (Berry et al. 2004). A range of models with different rates of elimination were used from 1987, and gave markedly different predictions by 2020. By 2000 the predictions were sufficiently different to allow some discrimination, and the observed numbers for 1987-2000 corresponded quite closely with the predictions based on an elimination rate of 15% per year, and were much less than the predicted number from a non-elimination model.

Regional projections have been made in many situations (Britain: Peto et al. 1995, Hodgson et al. 2005; Europe: Peto et al. 1999; Netherlands: Segura et al. 2003; US: Price & Ware 2003; Australia: Leigh and Driscoll 2003). As noted earlier exposure information is unavailable and the method used is essentially to estimate relative exposures from the data available and then use these to project forward. What is known about the use of asbestos in the region is important. In Australia it is known that there was no new use of crocidolite later than the early 1970s and of amosite about 10 years later. There was limited new use of chrysotile until 2003. Failing to take the known use of amphibole asbestos into account can lead to errors in predictions. Some of the predictions made in Europe have later been adjusted downwards when the known use was taken better into account.

An example in Australia was the prediction made for estimating the future liability of James Hardie (see Haigh 2006). Here there was under estimation and the reason for much of this underestimation was that the results from Wittenoom had been used to form a model; however Wittenoom closed in 1966 whereas amphibole (amosite) was used in Australia for another 15 or so years. In effect the peak incidence was taken as several years too early, and this had a large effect on the number of predicted mesotheliomas in later years (Clements et al. 2007a).

Initially the models usually used were age/birth cohort models but it can be shown that these are likely to break down in the situation where asbestos use is changing over time. An important step forward was made by Hodgson et al. (2005). Their model was based on exposure being dependent first on calendar year, where the average exposure over the population would be related to the amount of asbestos processed in each year, and second on age, where exposure would depend on the age distribution of those who were actually working with the asbestos. The exposure was then integrated over time for a particular age in a particular year. The second author (MC) has developed methods to improve fitting of this sophisticated model and applied the methods to mesotheliomas in New South Wales (Clements et al. 2007b).

For NSW the estimated exposure component by age gave an increasing effect from 20 to 40 and then a decline to a low level by age 60. This, certainly the latter part, fits in with what we know of workforce participation. The estimated calendar year effect shows a rapid rise through the 1940s and 1950s, a peak in the 1960s, followed by a decline to a low level by 2000. This fits in reasonably well, although not exactly, with the known use of amphibole asbestos in Australia.

For modeling of mesothelioma incidence among Australian males (Figure 2) using case data for the period 1983-2003, this model predicts a peak in 2017 (95% CI: 2014, 2022), with the total number of cases for 2006-2060 being 21200 cases (95% CI: 16800, 27000).

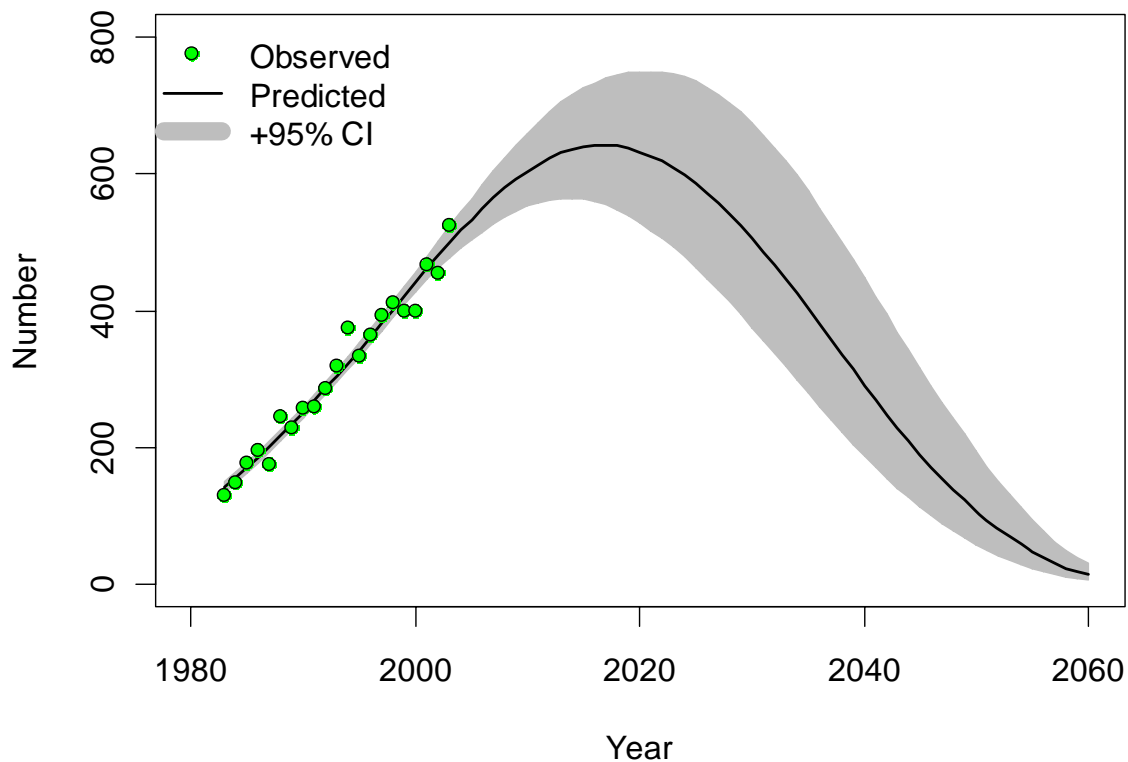


Figure 2: Predicted number of incident mesothelioma cases, Clements et al (2007b) model, Australian males

Conclusions

- 1) The suggestion by Gilson that the different asbestos fibre types pose different mesothelioma risks, with crocidolite giving the highest risk, amosite next, and chrysotile a much lower risk, has been confirmed by subsequent research.
- 2) There is increasing evidence that the increase in mesothelioma rate with time since exposure is moderated by an “elimination” type effect.
- 3) Predictions of future mesothelioma numbers should take into account information on the use of amphibole asbestos, and the effect of elimination over long periods.

But even taking everything known into account as well as current knowledge allows, the future may not be kind to our predictions. The next twenty years will likely reveal new insights. Mark Twain (also attributed to Neils Bohr) is without doubt correct in saying that “Prediction is difficult, especially of the future”.

Acknowledgements

During 40 years in the area the first author has been fortunate to work, and is still working to a limited extent, with many excellent researchers who have made important contributions.

References

Armitage P, Doll R. (1954). The age distribution of cancer and a multi-stage theory of carcinogenesis. *Br J Cancer* 8, 1-12.

- Berry G. (1991). Prediction of mesothelioma, lung cancer, and asbestosis in former Wittenoom asbestos workers. *Br J Ind Med* 48, 793-802.
- Berry G. (1999). Models for mesothelioma incidence following exposure to fibers in terms of timing and duration of exposure and the biopersistence of the fibers. *Inhal Toxicol* 11, 111-130.
- Berry G, Newhouse ML (1983). Mortality of workers manufacturing friction materials using asbestos. *Br J Ind Med* 40, 1-7.
- Berry G, De Klerk NH, Reid A, Ambrosini GL, Fritschi L, Olsen NJ, Merler E, Musk AW. (2004). Malignant pleural and peritoneal mesotheliomas in former miners and millers of crocidolite at Wittenoom, Western Australia. *Occup Environ Med* 61, e14, 1-3. (<http://www.occenvmed.com/cgi/content/full/61/4/e14>).
- Clements M, Berry G, Shi J (2007a). Actuarial projections for mesothelioma: an epidemiological perspective. *Institute of Actuaries of Australia XIth Accident Compensation Seminar 1-4 April 2007, Melbourne*, 1-17. Institute of Actuaries of Australia, Sydney.
- Clements M, Berry G, Shi J, Ware S, Yates D, Johnson A (2007b). Projected mesothelioma incidence in men in New South Wales. *Occup Environ Med* 64, 747-752.
- de Klerk NH, Armstrong BK, Musk AW, et al. (1989). Predictions of future cases of asbestos-related disease among former miners and millers of crocidolite in Western Australia. *Med J Aust* 151, 616-20.
- Doll R, Peto J. (1985). *Asbestos: Effects on Health of Exposure to Asbestos*, London: Health and Safety Commission, HMSO.
- Gilson JC. (1966). Health hazards of asbestos: recent studies on its biological effects (Wyers Memorial Lecture 1965). *Trans Soc Occup Med* 16, 62-74.
- Haigh G. (2006). *Asbestos House*. Carlton North, Victoria: Scribe Publications.
- Hodgson JT, Darnton A. (2000). The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann Occup Hyg* 44, 565-601.
- Hodgson JT, McElvenny DM, Darnton AJ, et al. (2005). The expected burden of mesothelioma mortality in Great Britain from 2002 to 2050. *Br J Cancer* 92, 587-593.
- Jones JSP, Smith PG, Pooley FD, Berry G, Sawle GW, Wignall BK, Madeley RJ, Aggarwal A. (1980). The consequences of exposure to asbestos dust in a wartime gas-mask factory. In: *Biological Effects of Mineral Fibres*, ed. J.C. Wagner, 637-53. IARC Scientific Publications No. 30, Lyon.
- Leigh J, Driscoll T. (2003). Malignant mesothelioma in Australia, 1945-2002. *Int J Occup Environ Hlth* 9, 206-217.
- McDonald JC, Harris JM, Berry G. (2006). Sixty years on: the price of assembling military gas masks in 1940. *Occup Environ Med* 63, 852-855.
- Newhouse ML, Thompson H. (1965). Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br J Ind Med* 22, 261-269. (reprinted 1993, 50, 769-778)
- Peto J, Hodgson JT, Matthews FE, et al. (1995). Continuing increase in mesothelioma mortality in Britain. *Lancet* 345, 535-539.
- Peto J, Decarli A, La Vecchia C, et al. (1999). The European mesothelioma epidemic. *Br J Cancer* 79, 666-672.
- Price B, Ware A. (2003). Mesothelioma trends in the United States; an update based on surveillance, epidemiology, and end results program data for 1973 through 2003. *Am J Epidemiol* 159: 107-112.
- Salerno DF, Feitshans IL. (2004). Muriel (Molly) Lina Newhouse, MD: British doyenne of occupational medicine. *J Epid Commun Hlth* 58, 17- .
- Segura O, Burdorf A, Looman C. (2003). Update of predictions of mortality from pleural mesothelioma in the Netherlands. *Occup Environ Med* 60, 50-55.

Stanton MF, Wrench C (1972). Mechanisms of mesothelioma induction with asbestos and fibrous glass. *J Natl Cancer Inst* 48, 797-821.

US Department of Labor, Occupational Safety and Health Administration. (1983). Occupational exposure to asbestos; emergency temporary standard. *Federal Register* 48 (215), 51085-51140.

Wagner JC, Sleggs CA, Marchand P (1960). Diffuse pleural mesothelioma and asbestos exposure in the Northwestern Cape Province. *Br J Ind Med* 17, 260-271.

Wagner JC, Berry G. (1969). Mesotheliomas in rats following inoculation with asbestos. *Br J Cancer* 23, 567-581.

DO SMOKING BANS IN ENCLOSED PUBLIC PLACES IMPROVE AIR QUALITY?

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Abstract

Environmental tobacco smoke (ETS) is a complex mixture of particulate and vapour phase compounds, which have been classified as a toxic air contaminants by the Californian Environmental Protection Agency (CEPA, 2005). ETS exposure in NSW hospitality venues has been estimated to cause over 73 worker deaths per year amongst hospitality workers. On 2nd July 2007, NSW introduced a smoking ban in all enclosed public places. A study is currently underway to observe the changes in a number of indoor air quality parameters associated with the ban, in particular nicotine, PM_{2.5}, PM_{1.0}, respirable particulates, carbon monoxide and particulate polycyclic aromatic hydrocarbons. Monitoring was undertaken using a range of real-time instruments and air sampling techniques, and has shown a significant improvement in indoor air quality since the introduction of the smoking ban.

Acknowledgments: This project was supported by the University of Western Sydney and ACT Health

Introduction

Environmental tobacco smoke (ETS) is a complex mixture of particulate and vapour phase compounds. ETS is classified as a toxic air contaminant by the Californian Environmental Protection Agency (CEPA, 2005). ETS exposure in NSW hospitality venues has been estimated to cause over 73 worker deaths per year amongst hospitality workers (Repace, 2004). As of the 2nd July 2007, NSW banned smoking in all enclosed public places.

Aims & Objectives

The aim of this study is to observe the changes in indoor air quality parameters before and after the smoking ban.

The objectives of the data presented are to:

- Compare data collected before and after the ban;
- Observe for any trends immediately following the ban.

Methods

Monitoring of air quality was undertaken before and after the NSW smoking ban. Pre-ban monitoring was conducted in a single venue over 4 consecutive days. Post-ban monitoring commenced 2 weeks after the ban, and continued one day per month thereafter.

The monitoring equipment was located centrally in the gaming area of a licensed club where smoking was permitted. Each of the following parameters was monitored for 4 hours:

- **Respirable Particulates** were measured by AS2985 using SKC aluminium cyclones fitted with 25mm 5µm PVC filters at a flow rate of 2.5 L min⁻¹.
- **PM_{2.5}** was monitored with a TSI DustTrak fitted with the PM_{2.5} inlet and calibrated at 1.7 L min⁻¹.
- **PM_{1.0}** was monitored with a TSI AM510 fitted with the PM_{1.0} inlet and calibrated at 1.7 L min⁻¹.
- **Particulate Polycyclic Aromatic Hydrocarbons (PPAH)** were monitored using an EcoChem Photoelectric Aerosol Sensor 2000CE (PAS2000CE).

- **Nicotine** was monitored according to NIOSH 2551 and analysed at WorkCover NSW Laboratories.

Other indoor air quality parameters were measured and will be reported on at a later date.

Significant Findings

Figure 1 indicates that ETS had a significant impact on particulate levels in the gaming area. Before the smoking ban PM1.0 particulates contributed to a significant portion of PM2.5 (1:1 ratio). However, the difference between PM2.5 and PM1.0 levels increased after the ban, indicating a reduction in exposure to particulates <1.0µm in the absence of indoor smoking.

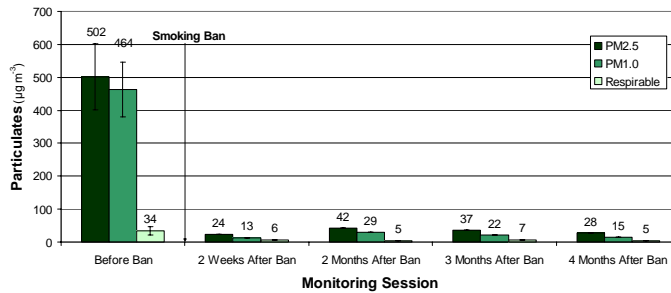


Figure 1: Comparison of Pre and Post Smoking Ban Levels of PM_{2.5}, PM_{1.0} and Respirable Particulates

Pre smoking ban PM_{2.5} levels exceeded recommended public health exposure limits of 100 µg m⁻³ (maximum 1 hour average) in residential premises (Health Canada, 1995), and 25 µg m⁻³ (24 hour average) in ambient air in Australia (NEPC, 2003). Respirable particulates were below the recommended occupational environment threshold limit value (TLV) of 3 mg m⁻³ for particulates not otherwise classified (ACGIH, 2007).

Figure 2 compares the mean pre ban PPAH level before the ban (n=4) with levels measured up to 4 months after the ban.

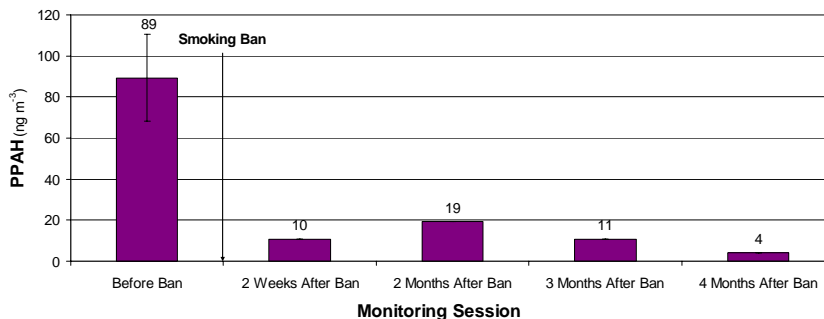


Figure 2: Comparison of Pre and Post Smoking Ban PPAH Levels

Figure 2 identifies that PPAH were also significantly reduced in the absence of indoor smoking. Post-smoking ban fluctuations in PPAH levels mirror particulates in Figure 1. This indicates that current particulate levels may be influenced by combustion by-products such as cooking exhaust, wood smoke or diesel exhaust, previously masked by the presence of ETS.

Figure 3 compares nicotine levels before and after the July 2 smoking ban.

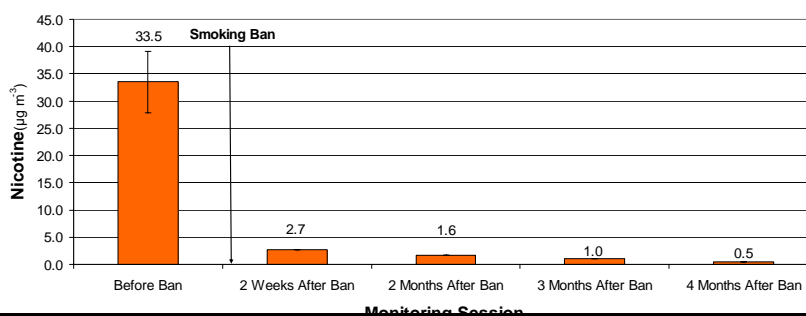


Figure 3: Comparison of Pre and Post Smoking Ban Nicotine Levels

In Figure 3, the nicotine levels were seen to drop significantly in the 2 weeks following the smoking ban. Nicotine levels continued to lower with each monitoring session over the 4 months (Figure 3). Carpets were cleaned after the smoking ban. However, nicotine adsorbs to other reservoirs like stainless steel and wallboard, and low desorption coefficients may mean nicotine could persist for months or even years after emissions ceases (Van Loy et al, 2001). Nicotine levels initially exceeded the World Health Organisation's recognised level of 1-10 µg m⁻³ (as an indicator of ETS) associated with increased morbidity and mortality from other serious health effects. However, nicotine levels are currently below 1 µg m⁻³ and more likely to relate to nicotine off gassing, than as an indicator of ETS exposure.

Conclusions

The smoking ban significantly reduced particulate, PPAH and nicotine levels in the gaming area. The impact of other indoor air contaminants on particulates and PPAH levels is now visible in the absence of ETS with fluctuations occurring between monthly monitoring sessions. Nicotine levels have continued to steadily decline after the smoking ban.

References

- ACGIH (2007). *2007 TLVs and BEIs*, American Conference of Governmental Industrial Hygienists; Cincinnati
- CEPA (2005). *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant*, California; California Environmental Protection Agency.
- Health Canada (1995). *Exposure Guidelines for Residential Indoor Air Quality*; Revised Edn, Health Canada; Ontario.
- NEPC (2003). *Variation to the National Environment Protection (Ambient Air Quality) Measure*, National Environment Protection Council, Canberra
- Repace, J. L. (2004). *Estimated Mortality from Secondhand Smoke among Club, Pub, Tavern, and Bar Workers in New South Wales; Australia*, Sydney; The Cancer Council New South Wales.
- Van Loy, M. D., Riley, W. J., Daisey, J. M. & Nazaroff, W. W. (2001). 'Dynamic behaviour of semivolatile organic compounds in indoor air. 2 nicotine and phenanthrene with carpet and wallboard', *Environmental Science and Technology*, Vol.35 pp 560-567.
- WHO (2002). *Air quality guidelines for Europe*, 2nd Edn, world Health Organisation: Copenhagen

VENTILATION AND INFECTION

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ABSTRACT

The dense contact networks that characterize urban cities form an ideal basis for rapid and uncontrolled disease propagation, especially by the airborne route of spread. People in developed countries/regions spend more than 90% of their time indoors in homes, offices, schools, vehicles, and airplanes etc. Using a new social contact model integrated with epidemiological models, we investigated the roles of ventilation and “indoor contact” in a connected indoor environment (offices, homes, hospitals, schools, restaurants, public buses etc). A recent multi-disciplinary systematic review of literature shows that there is strong and sufficient evidence for the association between ventilation, the control of airflow direction in buildings, and the transmission and spread of infectious diseases.

Our study into the three large SARS outbreaks in Hong Kong and Beijing revealed the new research challenges in the field of ventilation, e.g. determining the minimum ventilation requirements in hospitals, schools and offices in relation to the spread of airborne infectious diseases, understanding the evaporation and dispersion of respiratory droplets, air distribution for ventilation removal of both gaseous pollutants and large droplets (particles), interaction between respiratory flows and ventilation, the need of satisfying ventilation requirements in hospitals and isolation rooms in resources-limited countries etc and finally the need of an multi-disciplinary research approach.

INTRODUCTION

Ventilation of indoor environment is the process of supplying external air into a space and distributing it within it. Ventilation of buildings is important for health [Seppanen et al 1999, Berglund et al 1992, Sundel, 2004], thermal comfort [Kaczmarczyk et al 2004, Fanger 2006] and productivity [Seppanen and Fisk 2004]. To provide healthy air for breathing of occupants in a building is the primary purpose of ventilation.

Airborne transmission refers to the passage of micro-organisms from a source to a person through aerosols, resulting in infection and/or disease of the person with or without consequent disease [Li et al 2007]. The roles of building ventilation in transmission of respiratory diseases have probably been underestimated and in many investigations of disease outbreaks, there is a lack of the ventilation scientists and engineers in the team [Li et al 2007], even for outbreaks of diseases that are known to be airborne, such as tuberculosis, measles and chickenpox. The current concerns about an influenza pandemic and the growing threat of deliberately released agents such as anthrax serve as timely reminders that airborne infectious diseases remain a serious threat to human health. Does the ventilation community have a role to play? How important is this role relative to other public health measures?

The dense contact (both space and social) networks in the modern large cities form an ideal basis for rapid and uncontrolled disease propagation, especially by the airborne route of spread. As in 2000, there were globally 17 megacities with more than 10 million population and 22 large metropolitan areas with 5 to 10 million population. In public health measures, the most investigated is quarantine, isolation of cases, and use of anti-virals, and seldom building ventilation. Is ventilation an effective public health intervention measure? The answer may be simple, as many claim that there has not been any solid evidence on the association between building ventilation and the transmission of infectious diseases.

Indoor environments—offices, homes, hospital wards, classrooms, restaurants and public transport—are “connected” in terms of airborne disease transmission between people. People are constantly in contact as they move from one indoor environment to another, making it very easy for an airborne disease to be transmitted from an infected to a susceptible individual and ultimately to others. A study of the roles of building ventilation on disease transmission needs integration of the knowledge of social contact pattern, epidemiology dynamics of infectious diseases and building ventilation.

In this paper, we will review our study into the roles of ventilation in the three large SARS outbreaks in Hong Kong and Beijing, to summarize the findings of the recent multi-disciplinary systematic review of literature on the association between ventilation and infection, and to present some preliminary results in investigating the roles of ventilation and “indoor contact” in a connected indoor environment. New research challenges in the field of ventilation are then discussed.

METHODS

Methods used in outbreak investigations

The three large SARS outbreaks in Hong Kong and Beijing that we investigated included the Amoy Garden outbreak [Yu et al 2004, Li et al 2005, Li et al 2006], the Prince Wales of Hospital Ward 8A outbreak [Wong et al 2004, Yu et al 2005, Li et al 2005] and the Beijing Hospital T outbreak [Qian, 2007]. The general methods used in investigating the three outbreaks include epidemiological analysis, field ventilation measurement, computational fluid dynamics and multi-zone airflow modeling.

Epidemiological analysis was carried out by our collaborating epidemiologists who collected and analyzed data including the index patient, the number of infected people, the date of infection and the date of symptoms etc. Important risk factors were studied using the multivariate Cox proportional hazards regression. An important point to notice about the environmental engineering methods is that the airborne evidence of infection disappeared rapidly once the infectious period is over. Investigations of the environmental conditions such as air flows at the time of the outbreak are very difficult and quite often impossible. Thus, the purpose of field ventilation measurement was to collect essential ventilation data for further computer modeling studies. Field measurement provided data for validating computer simulations for the situation at the time of measurement, so that confidence in the computer simulation results for ventilation at the time of infection may be established.

Methods used in the literature review

We searched the major literature databases between 1960 and 2005, and then screened titles and abstracts, and finally selected 40 original studies based on a set of criteria. We established a review panel comprising of medical and engineering experts covering areas of microbiology, medicine, epidemiology, indoor air quality, building ventilation etc. Most of the panel members had experience with research on recent SARS epidemic. This panel systematically assessed the 40 original studies through both individual assessment and a two-day face-to-face consensus meeting.

Methods used in the connected indoor environment study

We divided the total population into several sub-populations such that every individual in a sub-population can be assumed to have the same hourly schedule. All locations, i.e. indoor environments are divided into sub-groups, such as homes, classrooms, offices, restaurants, shops, public places and public transport etc. Infectious probabilities for all pair of individuals in the

network combine another network, i.e. the indoor transmission network of airborne diseases. The SEIR model [Anderson and May 1992] is employed to study the epidemiology of the airborne disease over the indoor transmission network. Three parameters are crucial in the indoor transmission network, i.e. the visiting probability of a location by an individual, the effective quanta generation rate of the infector and the ventilation rate of the location. We divide an urban indoor environment network into basic types of indoor environments, such as household, school, office, restaurant, shops, transportation vehicles, and other public spaces. We collect statistical data of the population in Hong Kong about the number of spaces for each type of indoor environment, the number and probability of people visiting each type of indoor environment, typical ventilation rate for each type of indoor environment, etc. The developed social contact model allows us to estimate the probability of an individual meeting another in one indoor environment. The probability of an individual infecting another with an airborne disease in one indoor environment can thus be estimated by integrating with the Wells-Riley equation [Riley et al 1978]. The results for the connected indoor environment study are still at the preliminary stage.

RESULTS

Three SARS outbreaks

The first SARS cases occurred in mid-November in Guangdong, China. The disease spread to the rest of the world by a Guangdong infected medical doctor visiting Hong Kong in February 2004. He stayed in a hotel M and infected at least 14 hotel guests and visitors from various countries including Hong Kong, Canada, Vietnam and Singapore. Most of these infected individuals sparked large outbreaks in hospitals after they returned home.

The Ward 8A outbreak - In Hong Kong, a 26-year-old Hong Kong resident visited Hotel M in late February during the Guangdong doctor's stay, and contracted the infection. He was treated in Ward 8A at a hospital PWH, which subsequently led to a large SARS outbreak from 11 to 25 March 2003, with 138 probable cases. The patients included 69 health-care workers, 16 medical students who were attending clinical teaching or examinations in the ward, and 53 patients/visitors who were either in the same ward or had visited their relatives there.

The Amoy Garden outbreak - On 14 and 19 March, a discharged patient from PWH visited Block E, Amoy Garden, a high-rise housing estate. The Amoy Gardens outbreak between 21 March and mid-April 2003 was the largest community cluster during the 2003 SARS epidemic outside mainland China, with a total of 321 infected cases and 42 deaths.

The Hotel T outbreak - A 27-year-old business woman in Taiyuan, Shanxi traveled to Guangdong in mid February 2003 and developed fever on 22 February. She was treated in a Beijing Hospital T on 2 March, and stayed with her sick mother in Ward 4 of the 7th floor Respiratory Department between 3 and 5 March, until they were transferred to an infectious disease hospital on March 5. In Hospital T, 8 patients and 24 health-care workers developed SARS between 8 March and 18 April.

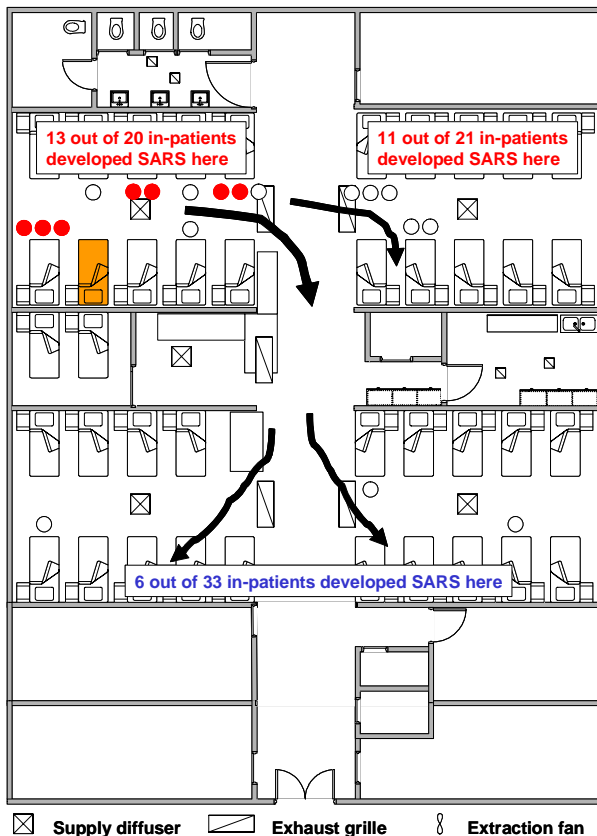


Figure 1. Floor plan of Ward 8A during the time of outbreak in March 2003. There were four large cubicles, each with 10 beds. The bed where the index patient stayed is marked in dark color. The location of the 19 (out of 20) medical students who attended the 40-minute bedside clinical assessments on 6 and 7 March are shown by bullets (developed SARS) and circles (did not develop SARS). The numbers of in-patients who developed SARS in each cubicle are also marked. The thick arrows indicate the overall airflow direction at the breathing level. Adapted from Li et al (2005).

In all these three large outbreaks, we identified the very probable roles played by building ventilation and airflow on the disease transmission.

The Ward 8A outbreak – Our predicted bio-aerosol concentration was the highest in the index patient’s cubicle at the time of exposure, followed by the adjacent cubicle, and the two distant cubicles. This bio-aerosol dispersion pattern seemed to be associated with the spatial infection pattern in Ward 8A. The attack rate was the highest in the index patient’s cubicle, followed closely by the adjacent cubicle. The attack rates in the two distant cubicles were low. The association between the predicted bio-aerosol concentration and the spatial infection pattern suggested a probable airborne transmission route in the Ward 8A outbreak, in addition to the commonly accepted large droplet and close personal contact transmission.

The Amoy Garden outbreak - Our epidemiological analysis, experimental studies and airflow simulations all provided support to the probable airborne spread of SARS in the Amoy Gardens outbreak. Virus-laden aerosols generated in the vertical soil stack of a Flat (wing) in Block E returned to the bathroom through dried-up floor drain traps and then entered the re-entrance, probably by the suction of an exhaust fan. The aerosols moved upwards in the re-entrance and could enter flats bordering the re-entrance in the upper floors due to the negative pressure created by exhaust fans or the action of wind flows around the building. The horizontal spread to other flats of Block E was by air movements between flats. After the plume reached the top of the re-entrance in Block E, 3-dimensional spread to some flats at certain heights in Blocks B, C and D followed and was due to the actions of the predominant northeasterly wind. SARS virus infection in the Amoy

Gardens seems to be of an unusual, perhaps even novel faeco-respiratory airborne route [Li et al 2006].

The Hotel T outbreak – The distribution of infected wards was not random and there was clearly a vertically spatial pattern. 70% (7 of 10) inpatient SARS cases located vertically at the floors above the index patients. Staying in the upper conduit-associated wards above the ward where the index patient stayed having the highest risk. Both air flow measurement and multi-zone airflow modelling indicated that the virus generated on the 7th or 8th floors can be effectively transported upwards through the Wards 4-5 service conduit, with the concentration of virus-laden aerosols highest on the 13th and 14th floor, followed by moderate on the 7-or 8-12th floors, and zero on the 5-6th floors. The spatial distribution of SARS cases in the hospital and the airflow measurement and analysis all supported a hypothesis of a probable airborne transmission for at least 2 of the 8 patient infections, while close contact and droplet transmission cannot be ruled out in all other infections.

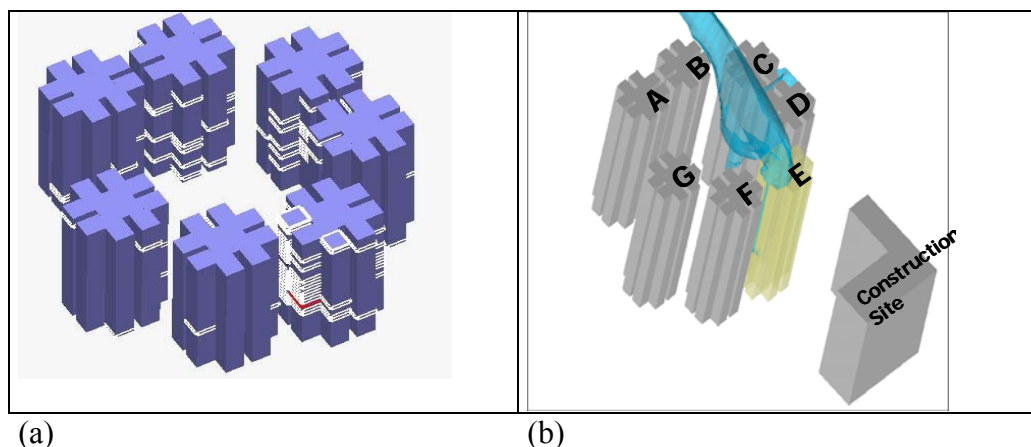


Figure 2. (a) A map of the three-dimensional spatial distribution of the infected flats in the Amoy Gardens between 21 March and 3 April. Two Flats (wings) in Block E were mostly infected. Four Flats in Block E were moderately infected, while two others had fewer infections. Blocks B, C and D were infected in some concentrated areas. Most other Flats (wings) were not infected [Li et al 2006]. (b) The buoyant plume rising from a re-entrance in Block E was carried downstream by a northeasterly wind, reaching middle levels of Blocks C and D. The L-shaped building was a nearby construction site. The construction site blocked the wind flow to the lower part of Block E, and Blocks C and D. The wake flow of the construction site created a negative pressure region in the open space between Blocks E, C and D. This negative pressure region caused the plume to bend downwards, towards Blocks C and D [Yu et al 2004].

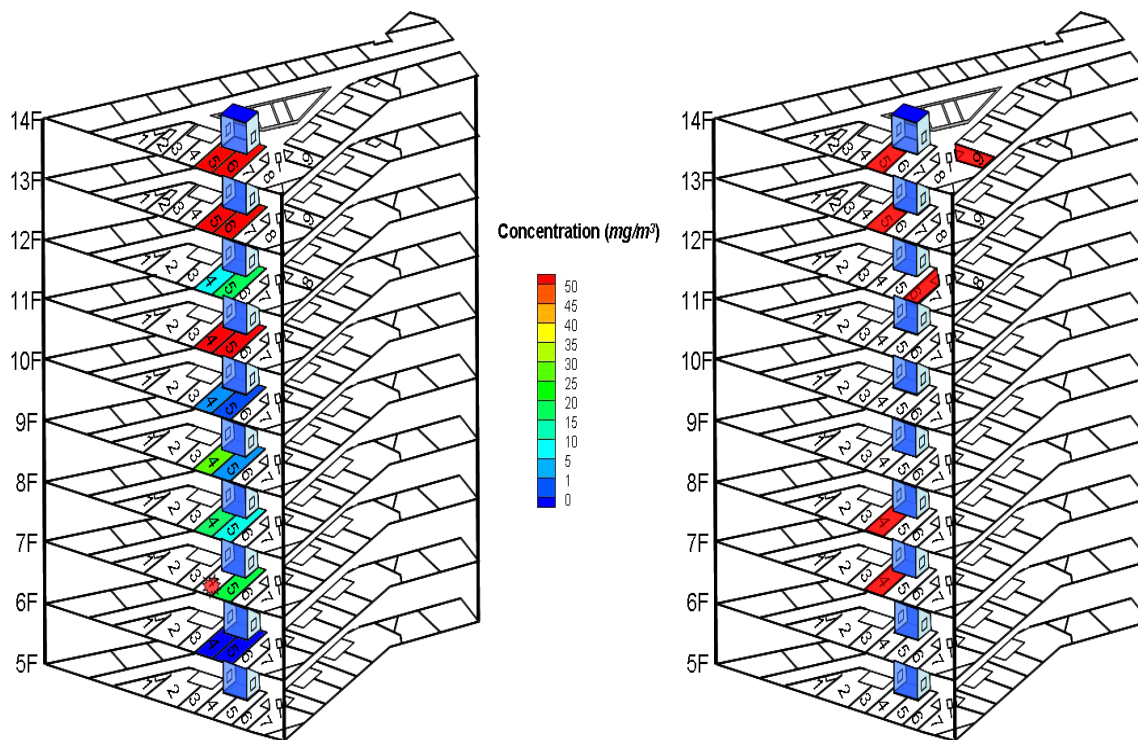


Figure 3. Illustration of the service conduit serving Wards 4 and 5 of 5-12 Floor and Wards 5 and 6 of the 13-14th Floor in Hospital T in Beijing. The measured concentrations of tracer gas in toilets when it was released in the toilet of Ward 4, Floor 7 are shown in the left figure, while the wards where infection occurred are shown in red (or dark) color in the right figure.

The literature review

Of the 40 studies that was assessed, 18 were considered as non-conclusive for supporting a direct contributory role of ventilation rate/airflow pattern to the airborne spread of infectious agents, 12 were considered partly conclusive, and 10 were deemed clearly conclusive as supporting a direct contribution.

The panel concluded that there was strong and sufficient evidence in the current literature to demonstrate a definite association between ventilation and airflow patterns in the indoor environment and the transmission of infectious diseases. Responsible agents cited include measles, TB, chickenpox, influenza, smallpox and SARS.

Among the 10 studies considered to be conclusive, five specifically examined the role of ventilation rates, i.e. by Menzies et al. (2000), Hoge et al. (1994), Moser et al. (1979), Riley et al. (1962), and Schulman and Kilbourne (1962). Menzies et al. (2000) presented one of the rare detailed population-based studies of the role of ventilation in hospitals. It showed a higher TB infection risk for health-care workers working in non-isolation rooms with ventilation rates of less than two air changes per hour (ACH). Five showed an association between airflow patterns and the spread of diseases, i.e. by Wehrle et al. (1970), Bloch et al. (1985), Gustafson et al. (1982), Hutton et al. (1990), and the study of a SARS outbreak by Li et al. (2005), Wong et al. (2004)] and Yu et al. (2005).

In many studies, the difficulties in ruling out other transmission routes such as direct contact and large droplets are obvious. Most studies involve the description of a single index case with secondary cases possibly arising from the index case. On the airflow side, there is some attempt to connect the index and the secondary cases using some kind of experimental or mathematical airflow studies such as tracer gas techniques, smoke visualization or computational fluid dynamics (CFD) simulations. Usually, the ventilation and airflow component of each study is quite poor and hence

there are many partly or non-conclusive studies. Of the 40 studies, 18 involve TB, of which 11 are non-conclusive, 3 are partly conclusive, and only 3 are conclusive. It is interesting to note that in many studies only descriptive information on ventilation is given. We observed a tendency that most of the studies that are partly conclusive were conducted by investigators from a single discipline (usually engineering or medicine), whereas multidisciplinary investigative teams using a more comprehensive and sophisticated set of techniques often scored higher on this assessment criterion (i.e. conclusive). In most of these studies, a floor/section plan of the buildings where the outbreak occurred and a sketch of the ventilation system design would be very useful to demonstrate the impact of the air environment.

There was, however, insufficient data to specify and quantify the minimum ventilation requirements in any setting, including in nosocomial environments such as hospitals and even more so in schools, offices and other buildings, in relation to the spread of infectious diseases via the airborne route. There is a knowledge gap.

Connected indoor environment

Based on preliminary data collection, estimates and assumptions for Hong Kong in 2005, there were 7 million people including 3.45 million employed individuals, and 3 million indoor environments (spaces, with a possibility of underestimation) including 2.3 million homes, 41,000 classrooms, 600,000 offices, 12,000 restaurants, nearly 47,000 shops, 200 individual movie theatres, and 8700 public vehicles. Our preliminary indoor contact network combines the data of individuals, locations and the visiting probability of each individual visiting each location. One novelty of our model is that it will take into account daily habits. It will realistically consider the favorite locations that an individual may visit, and assigns to each individual a number of favorite restaurants, bus routes and classrooms. By using the statistical information for all sub-populations and location groups, we can build the indoor contact network. As a preliminary attempt and due to the space limitation, it is not possible to list all the detailed input parameters as well as how we obtained them, including the average size of each indoor space and ventilation rates. It should be mentioned that the results presented here have not been validated. Our main purpose is to present a possible approach for estimating the effect of building ventilation on disease modeling at the population level.

Figure 4 shows the predicted reduction in the daily proportion of the infected in a susceptible population for two different ventilation rates in all indoor environments that we considered, after exposing with an index patient on Day 0. When the ventilation rate is 8L/s, the peak daily infection occurs on day 100 with 6% of the population infected. The occurrence of the peak daily infection incidence delayed and the value reduces as the ventilation rate increases. When outdoor ventilation rate is 16L/s, the disease would not spread in the social network. These would have significant public health implications. Most importantly, such an integrated approach by combining a social contact model, the wells-Riley equation and the SEIR model allows us to study the infection rate in each type of indoor environments in a population or even closure of one indoor environment type, e.g. Figure 5. In the intervention scenario, all students and public place workers are considered as household individuals and all other individuals have a zero possibility of visiting a public place. Closure of all classrooms and public places has a much less impact as compared with increasing ventilation rate (Figure 4).

This may allow us to examine the relative importance of ventilation requirements for different indoor environments at the whole population level. Our interests are in the relative importance of increasing ventilation in indoor environments in the next influenza pandemic as compared to other public health interventions, such as contact tracing, quarantine as well as anti-viral.

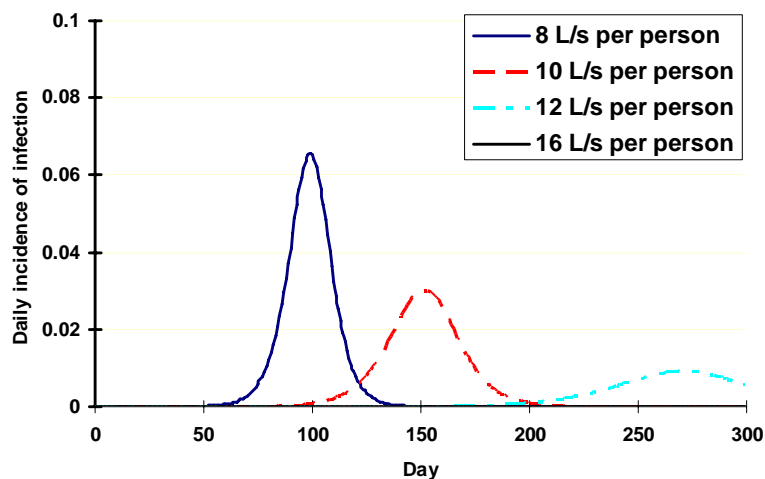


Figure 4. The predicted daily infection incidence (i.e. infection percentage of the total population) in Hong Kong after the first index person on Day 0 as outdoor air ventilation rate increases from 8, 10, 12 to 16 L/s per person for all indoor environments (spaces).

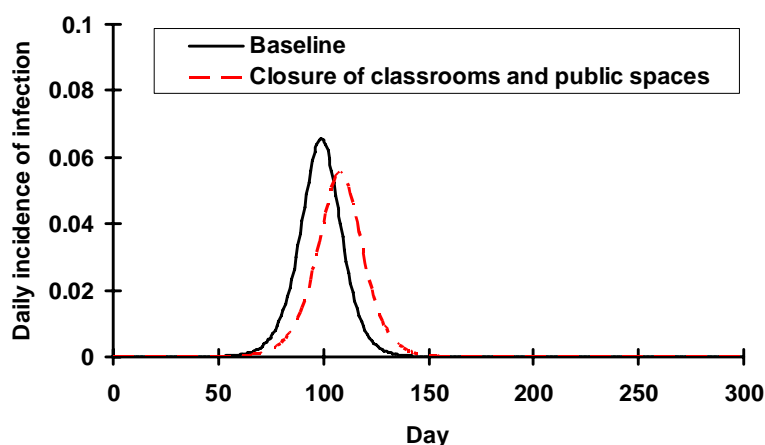


Figure 5. Baseline (ventilation rate = 8 L/s per person and all indoor environments are open) and one intervention scenario (closure of classrooms and public spaces) for the predicted daily infection incidence (i.e. infection percentage of the total population) in Hong Kong after the first index person on Day 0.

DISCUSSION

New research challenges in the field of ventilation

Our findings revealed the new research challenges in the field of ventilation, e.g. understanding the evaporation and dispersion of respiratory droplets [Xie et al 2007], air distribution for ventilation removal of both gaseous pollutants and large droplets (particles), interaction between inhalation/exhalation flows and ventilation [Qian et al 2006, Bjorn and Nielsen 2002], and the need of satisfying ventilation requirements in hospitals and isolation rooms in resources-limited countries etc [CDC, 1994]. Due to space limitation, the details will be covered in a workshop to be conducted in this conference.

The need of a paradigm shift in ventilation study

If a building is built with high ceiling, narrow depth, open windows or solar chimneys, natural forces such as wind and buoyancy can drive ventilation, i.e. natural ventilation. If a building is built without envelope openings, mechanical ventilation with fans can be used. In clinical settings, the

term of mechanical ventilation has a slightly different meaning, and it is to assist people with breathing disorders to move enough air in and out of their lungs. Similarly, mechanically ventilated buildings may have “breathing disorders”, i.e. the inability of using natural ventilation.

Many modern buildings are built and operated with “breathing disorders”. They were first designed to have “breathing disorders” as mechanical fans are available. However, both the ductwork cost and operating cost prevents us from providing very high ventilation flow rates in our buildings, but a minimum one, far less than those available in well designed and operated buildings with natural ventilation. To me, the recent revitalisation of natural ventilation has not meant a return to unreliable conditions. With the aide of new computer design tools and the integration of modern manufacturing and control technologies, natural ventilation can be as reliable as mechanical ventilation when properly designed and operated, in particular if the concept of hybrid ventilation is used.

Natural ventilation either alone or in a hybrid mode, offers an opportunity of providing very high ventilation rate, say 20-30 ACH. Transient high ventilation requirements need controllable large openings. There are no regulations to specify the transient high ventilation requirement. Achieving a transient high ventilation rate is one of the most important benefits of natural ventilation. The transient high ventilation rate also might be needed when there are renovation activities in the building, which generate very high amounts of pollutants in the air, or in isolation rooms when a suspected infection source is identified.

Although ventilation of buildings is important for health, thermal comfort and productivity, but providing healthy air for breathing of occupants and keeping their health should be the primary purpose of building ventilation. We note that while many papers have been published to reveal how we can improve air distribution in buildings using sophisticated computer modeling and experimental methods, but there is insufficient information/data to specify and/or quantify the minimum ventilation requirements in hospital and non-hospital environments in relation to the spread of airborne infection.

The need of a multidisciplinary study

In our review, we noted a few factors that might have contributed to the relatively rare conclusive scientific evidence of the roles of ventilation and airflow in the transmission of airborne diseases [Li et al 2007]. The most inherent limitation in almost all existing investigations is due to the rapid disappearance of airborne evidence of infection, once the infectious period is over. It becomes very difficult to then, retrospectively, reproduce the same ventilation and airflow conditions that existed at the time of the outbreak. Most of the time, outbreaks are only noticed when a significant number of people have been infected, so by this inherent nature, it is difficult to estimate, accurately, the exact time of onset of infection in the index case, as well as in any secondary cases. The presence of airborne infectious pathogens can ‘disappear’ rapidly after the source has been removed.

It was also interesting to note that the available advanced measurement methods for ventilation and air distribution at the time of investigation were not used by the investigators in many of the studies that we reviewed. These readily available techniques [Etheridge and Sandberg 1996] include various flow visualization methods for airflows in buildings, passive and active tracer gas methods for ventilation flow rates, CFD simulations for predicting airflow patterns, multi-zone airflow modeling for global airflow patterns, and airflow rates in multiple buildings. Most of these ventilation and airflow methods are often not available to most epidemiologists and microbiologists. The fact that many existing epidemiological studies, including those in the review, do not include adequate airflow studies reflects the lack of participation of engineers in outbreak investigations, or the lack of access to available engineering measurement methods by the investigation team.

We strongly suggest the need for a multidisciplinary approach to investigating both cross-sectional survey and population-based study [Menzies et al 2000] and outbreak situations [Tang et al 2005]. There is a need to develop a multidisciplinary research culture, not only for investigating a particular outbreak, but for general research challenges related to the control of disease spread in buildings. A multidisciplinary team of infectious disease and engineering specialists needs to be rapidly assembled as soon as an airborne infectious disease outbreak is identified, in order to accurately and comprehensively measure and document all relevant parameters (relating to both the people and the environment) at the earliest possible time. This will allow a detailed causal analysis to be performed, once the outbreak is over, with contemporaneous data collected during the outbreak. Obviously, the measurement of such parameters must take place in a manner that does not interfere with the health-care-related aspects of the outbreak.

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REFERENCES

- Anderson, R M and May, R M. 1992. *Infectious diseases of humans: dynamics and control*. Oxford University Press, Oxford.
- Berglund, B, Brunekreef, B, Knoppel, H, et al. 1992. Effects of indoor air pollution on human health. *Indoor Air*. Vol 2, pp 2–25.
- Bjorn, E, Nielsen, P V. 2002. Dispersal of exhaled air and personal exposure in displacement ventilated rooms. *Indoor Air*. Vol 12(3), pp 147-64.
- Bloch, A B, Orenstein, W A, Ewing, W M, et al. 1985. Measles outbreak in a pediatric practice: airborne transmission in an office setting. *Pediatrics*. Vol 75, pp 676–683.
- CDC (Center for Disease Control and Prevention). 1994. Guidelines for preventing transmission of *Mycobacterium tuberculosis* in health-care settings. *Morbidity Mortality Weekly Report*. Vol 43, RR–13.
- Etheridge, D and Sandberg, M. 1996. *Building Ventilation – Theory and Measurement*. John Wiley & Sons, Chichester, UK.
- Fanger, P O. 2006. What is IAQ? *Indoor Air*. Vol 16(5), pp 328-34.
- Gustafson, T L , Lavelly, G B , Brawner, E R Jr et al. 1982. An outbreak of airborne nosocomial varicella. *Pediatrics*. Vol 70, pp 550–556.
- Hoge, C W, Reichler, M R, Dominguez, E A, et al. 1994. An epidemic of pneumococcal disease in an overcrowded, inadequately ventilated jail. *New England Journal of Medicine*. Vol 331, pp 643–648.
- Hutton, M D, Stead, W W, Cauthen, G M, et al. 1990. Nosocomial transmission of tuberculosis associated with a draining abscess. *Journal of Infectious Diseases*. Vol 161, pp 286–295.
- Kaczmarczyk, J, Melikov, A, Fanger, P O. 2004. Human response to personalized ventilation and mixing ventilation. *Indoor Air*. Vol 14 Suppl 8, pp 17-29.
- Li, Y, Duan, S, Yu, I T S, et al. 2005. Multi-zone modeling of probable SARS virus transmission by airflow between flats in Block E, Amoy Gardens. *Indoor Air*. Vol 15, pp 96–111.
- Li, Y, Huang, X, Yu, I T S. et al. 2005. Role of air distribution in SARS transmission during the largest nosocomial outbreak in Hong Kong. *Indoor Air*. Vol 15, pp 83–95.
- Li, Y, Leung, G M, Tang, J W, et al. 2007. Role of ventilation in airborne transmission of infectious agents in the built environment - a multidisciplinary systematic review. *Indoor Air*. Vol 17(1), pp 2-18.

- Li, Y, Qian, H, Yu, I T S and Wong, T W. 2006. Probable roles of bio-aerosol dispersion in the SARS outbreak in Amoy Garden, Hong Kong. In *Population Dynamics and Infectious Diseases in Asia*, edited by A C Sleight, C H Leng, B S A Yeoh et al., World Scientific, New Jersey, pp. 305-327.
- Menzies, D, Fanning, A, Yuan, L, et al. 2000. Hospital ventilation and risk for tuberculous infection in Canadian health care workers. *Annals of International Medicine*. Vol 133, pp 779–789.
- Moser, M R, Bender, T R, Margolis, H S, et al. 1979. An outbreak of influenza aboard a commercial airliner. *American Journal of Epidemiology*. Vol 110, pp 1–6.
- Qian, H, Li, Y, Nielsen, PV, et al. 2006. Dispersion of exhaled droplet nuclei in a two-bed hospital ward with three different ventilation systems. *Indoor Air*. Vol 16, pp 111-128.
- Qian, H. 2007. Ventilation for controlling airborne infection in hospital environments. PhD thesis, the University of Hong Kong.
- Riley, E C, Murphy, G and Riley, R L. 1978. Airborne spread of measles in a suburban elementary school. *American Journal of Epidemiology*. Vol 107, pp 421–432.
- Riley, R L and O’Grady, F. 1961. *Airborne Infection – Transmission and Control*, The MacMillan Company, New York, NY.
- Riley, R L, Mills, C C, O’Grady, F, et al. 1962. Infectiousness of air from a tuberculosis ward: ultraviolet irradiation of infected air: comparative infectiousness of different patients. *American Review of Respiratory Diseases*. Vol 85, pp 511–525.
- Schulman, J L and Kilbourne, E D. 1962. Airborne transmission of influenza virus infection in mice. *Nature*. Vol 195, pp 1129–1130.
- Seppanen, O A, Fisk, W J and Mendell, M J. 1999. Association of ventilation rates and CO₂ concentrations with health and other responses in commercial and institutional buildings. *Indoor Air*. Vol 9, pp 226–252.
- Seppanen, OA and Fisk, WJ. 2004. Summary of human responses to ventilation. *Indoor Air*. Vol 14 Suppl 7, pp 102-18.
- Sundell, J. 2004. On the history of indoor air quality and health. *Indoor Air*. Vol 14(Sup. 7), pp 51–58.
- Tang, J W, Eames, I., Li, Y, et al. 2005. Door-opening motion can potentially lead to a transient breakdown in negative-pressure isolation conditions: the importance of vorticity and buoyancy airflows. *Journal of Hospital Infection*. Vol 61, pp 283-286.
- Wehrle, P.F., Posch, J., Richter, K.H. et al. (1970) An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe, *Bulletin of the World Health Organization*, 43, 669–679.
- Wells, W F. 1955. *Airborne Contagion and Air Hygiene: an Ecological Study of Droplet Infection*, Harvard University Press, Cambridge, MA.
- Wong, T W, Li, C K, Tam, W, et al. 2004. Cluster of SARS among medical students exposed to single patient, Hong Kong. *Emerging Infectious Diseases*. Vol 10, pp 269–276.
- Xie, X, Li, Y, Chwang, A T Y, et al. 2007. How far droplets can move in indoor environments – Revisiting Wells evaporation-falling curve of droplets. To appear in *Indoor Air*, 2007. (Online published in 2006)
- Yu, I T S, Li, Y, Wong, T W, et al. 2004. Evidence of airborne transmission of the severe acute respiratory syndrome virus. *New England Journal of Medicine*. Vol 350, pp 1731–1739.
- Yu, I T S, Wong, T W, Chiu, Y L, et al. 2005. Temporal-spatial analysis of severe acute respiratory syndrome among hospital inpatients. *Clinical Infectious Diseases*. Vol 40, pp 1237–1243.

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