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...linking the science and practice of Environmental Health

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

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

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

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Aims

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

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The Journal is seeking papers for publication.

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Reports and Reviews

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

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CONTENTS ENVIRONMENTAL HEALTH, VOLUME SIX, NUMBER TWO, 2006

Contents Environmental Health, Volume Six, Number One, 2006

Environmental Health, Volume 6, Number 2, draws our attention to a variety of issues from air and water pollutants, to housing and homelessness. Hansen adds to a growing body of literature regarding the association between maternal exposure to ambient air pollution during pregnancy and adverse birth outcomes. His research in Brisbane focuses on exposure during the first three months of pregnancy. Evidence from this study suggests that exposure to particulate matter during the second month of pregnancy is associated with an increased risk of delivering a neonate with a congenital anomaly. Hansen's study brings to our attention the need for further research in this area.

Atmospheric pollutants are also explored by Lawson and Wang with their three part series on diesel exhaust particles (DEP). Part one reviews the epidemiological evidence of correlations between DEP and adverse health effects. Part two concentrates on the pathophysiological effects of DEP, while Part three focuses on the biochemical effects of nanoparticles, and their chemical composition. Lawson and Wang have revealed a strong case for an increased research focus on the harmful effects of nanomaterials, rather than coarse or fine DEP sizes.

Bailie & Wayte explore a continuous Quality Improvement Approach to Indigenous Housing and Health. This paper proposes a long term ecological and quality improvement approach to maximise the impact of housing programs on Indigenous Health, and discusses how some current key initiatives could be developed to support such an approach. Bailie & Wayte also identify important gaps in information from State and Commonwealth data systems on Indigenous housing. This paper proposes that an ecological approach with concurrent targeting of infrastructure factors, behavioural factors and factors in the policy environment will improve the effectiveness of housing-related interventions.

Holmes explores a tangential theme through her qualitative investigation into the lived experience of homelessness and the role of trauma in the lives of homeless people in Darwin. Holmes emphasises the need for the *National Environmental Health Strategy* and environmental health practitioners to recognise homelessness as a discrete area for action. The study also presents discussions on ways in which environmental health practitioners could incorporate the complexities of homelessness into their environmental health services.

Nanbakhsh presents an interesting experiment to assess stormwater detention systems for treatment efficiencies. The experimental stormwater detention systems in his investigation received high volumes of concentrated runoff treated by filtration with different aggregates. These aggregates, such as gravel, sand, Ecosoil, block paving and turf, were tested to ascertain their influence on water quality. Nanbakhsh found that the use of additional aggregates with high adsorption capacities in the primary treatment stage does not make a significant impact on water quality.

I hope you enjoy our current issue of *Environmental Health*. If you have any comments on articles published in this issue of the Journal, or on a particular environmental health issue, don't keep them to yourself! Send a Letter to the Editor, and let your views make a difference. We should love to hear from you!

For those of you preparing a presentation for the upcoming AIEH National Conference in November, now is the time to start polishing your notes for publication in the Journal. Having your research and reports published in a peer reviewed journal not only reaches a wide audience of environmental health peers and practitioners, but also assists in adding authority to your presentation in November. To ensure publication before your presentation, papers must be submitted by the end of August. For more information on preparing articles for the Journal, contact Jaclyn Huntley, Assistant Editor at journal@aieh.org.au.

Jim Smith
Editor

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Ambient Air Pollution and Congenital Anomalies in Brisbane, Australia: Should We be Concerned?

Craig Hansen

Faculty of Science, Health and Education, University of the Sunshine Coast, Queensland & School of Medicine, University of Queensland

There is a growing body of literature suggesting that maternal exposure to ambient air pollution during pregnancy is associated with adverse birth outcomes. The aim of the research was to assess the relationship between ambient air pollution during the first three months of pregnancy and the risk of a congenital anomaly among children born in Brisbane. A retrospective cohort design was employed where a citywide average of daily PM_{10} (particulate matter with an aerodynamic diameter <10 μm), bsp (light scattering particles), ozone (O_3), and nitrogen dioxide (NO_2) concentrations were linked to the days of gestation for term singleton live births in the Brisbane local government area for the period of 1st July 2000 to 30th June 2003. Maternal exposure estimates for each pollutant were calculated as the average of the daily concentrations within each of the first three months of pregnancy. An inter-quartile range increase in PM_{10} during the second month of pregnancy was associated with an 8% (OR = 1.08, 95% CI 1.01 - 1.15) increased risk of delivering a neonate with a reported congenital anomaly. Further research is warranted where specific congenital anomalies are examined.

Key words: *Air Pollution; Pregnancy; Congenital Defects*

There is a growing body of literature suggesting that maternal exposure to ambient air pollution during pregnancy is associated with adverse birth outcomes such as preterm birth, low birth weight, and intrauterine growth restriction (Glinianaia et al. 2004; Lacasana et al. 2005; Maisonet et al. 2004; Sram et al. 2005). In addition, recent studies in Southern California (Ritz et al. 2002) and Texas (Gilboa et al. 2005) found that ambient air pollution during the second month of pregnancy is associated with birth defects.

The majority of air pollution-birth outcome research has been conducted in cities throughout the world where ambient air pollution levels are relatively high, however, a recent study in Sydney, Australia (Mannes et al. 2005) found that lower concentrations of air pollutants were associated with sub-optimal fetal growth. Ambient air pollution concentrations in Sydney are similar to those in Brisbane and this provided the rationale to carry out an air pollution-birth outcome study in Brisbane. Many birth outcomes were investigated and this brief report focuses only on the results pertaining to a reported congenital anomaly (non-categorised).

Methods

Birth data for all singleton live births in Brisbane for the period of 1st July 2000 to 30th June 2003 were collected from the Queensland Health Perinatal Data Collection Unit. The main source of air pollution in Brisbane is vehicle emissions (Queensland Environmental Protection Agency 2001) and therefore data pertaining only to neonates born to mothers who had resided within the Brisbane local government area (approximately a 20 km radius around the city) during pregnancy

were obtained. The variables obtained from the database were date and method of delivery, birth weight, neonate gender, gestational age (weeks), a reported congenital anomaly (non-categorised), age of mother, number of previous pregnancies, number of previous abortions/miscarriages, marital status, indigenous status, number of antenatal visits, pre-pregnancy medical conditions, and an index of relative socio-economic disadvantage associated with the suburb of residency during pregnancy (index of SES).

The date of the mother's last menstrual period (LMP) was not provided and therefore was estimated by calculating back the number of weeks of gestation from the date of delivery. Neonates with a gestational age of <37 weeks and who were born to mothers with pre-pregnancy medical conditions such as diabetes, respiratory problems (e.g. asthma), epilepsy, cardiac problems, hypertension, and a previous stillbirth, and who had missing data on variables were excluded from the final sample.

To estimate the concentrations of ambient air pollution in Brisbane, hourly readings for PM_{10} (particulate matter with an aerodynamic diameter <10 μm), bsp (light scattering particles), ozone (O_3), and nitrogen dioxide (NO_2) were obtained from the Air Services Unit, Queensland Environmental Protection Agency. These data were recorded at 5 monitoring stations across Brisbane for PM_{10} while data for bsp, NO_2 and O_3 were recorded at 4 monitoring stations. A daily average (24-hr average) was calculated from the original hourly data for PM_{10}, bsp, and NO_2, and an 8-hour moving average was calculated for O_3. To estimate the level of ambient air pollution in Brisbane a citywide average of the daily values was then calculated across the available monitoring stations for each pollutant.

The dates of gestation for each mother/neonate pair were matched to the dates of the daily air pollution time-series. Monthly-specific exposure estimates for each mother/neonate pair were calculated as the mean of the daily pollutant values within each of the first three months (three 30-day periods) of pregnancy starting from the date of the LMP. The main focus was on the first three months of pregnancy because this is the period most vulnerable to teratogenic insult (Finnell et al. 2002).

Multiple logistic regression was employed to calculate odds ratios (OR) and 95% confidence intervals (CI) for a reported congenital anomaly in relation to an inter-quartile range (IQR) increase in the maternal exposure estimates for each pollutant. An IQR increase was chosen in order to compare results across different pollutants. The analyses controlled for neonate gender, gestational age, age of mother, parity, number of previous abortions/miscarriages (nil, 1, 2 or more) marital status (married, single, other), indigenous status (yes/no), number of antenatal visits (8 or more, 5-7, <4), an index of SES, and season of birth.

Results

The air pollution concentrations for Brisbane during the study period are presented in Table 1. There were 31,307 births in the Brisbane local government area during the study period and of these there were 28,200 births eligible for inclusion into the study. Of the 28,200 eligible births there were 26,617 term births, in which 1,209 (4.5%) were born with a reported congenital anomaly.

As shown in Table 2, an IQR increase in PM_{10} during the second month of pregnancy was associated with an 8% increased risk of being born with a congenital anomaly. A similar effect can be observed for bsp during month two, however, these results failed to reach statistical significance.

Plots of the odds ratios for a reported congenital anomaly associated with monthly-specific exposures for each of the nine months of pregnancy were also examined and a steady pattern of non-significant odds ratios for the remaining months of PM_{10} exposure was observed

Table 1: Air pollutant concentrations for Brisbane, September 1999 to June 2003

Pollutant				Percentiles			
	Mean	SD	Min	25th	50th	75th	Max
bsp (Mm^{-1})	17.1	14.1	1.1	8.8	12.9	20.7	168.9
PM_{10} ($\mu g/m^3$)	19.6	9.4	4.9	14.6	18.1	22.7	171.7
O_3 (ppb)	26.7	7.8	6.7	21.0	25.9	31.5	61.1
NO_2 (ppb)	8.8	4.1	1.0	5.5	7.8	11.4	24.2

Notes:
ppb = parts per billion
SD = standard deviation, Min = minimum, Max = Maximum
bsp, PM_{10}, NO_2 = 24-hr; O_3 = 8-hr.

Table 2: Odds ratios (and 95% CI) for a reported congenital anomaly associated with an inter-quartile range increase in monthly-specific maternal exposure to ambient air pollutants during the first three months of pregnancy

	Month 1	Month 2	Month3
	OR (95% CI)	OR (95% CI)	OR (95% CI)
PM_{10}			
Crude effect	1.02 (0.96 - 1.08)	1.08 (1.02 - 1.14)**	0.97 (0.91 - 1.03)
Adjusted [1]	0.98 (0.91 - 1.05)	1.08 (1.01 - 1.15)*	0.93 (0.86 - 1.01)
bsp			
Crude effect	1.01 (0.95 - 1.07)	1.06 (0.99 - 1.14)	0.97 (0.90 - 1.04)
Adjusted [1]	0.96 (0.89 - 1.04)	1.06 (0.97 - 1.16)	0.93 (0.84 - 1.02)
O_3			
Crude effect	1.08 (0.99 - 1.18)	1.06 (0.97 - 1.15)	0.99 (0.90 - 1.09)
Adjusted [1]	1.08 (0.96 - 1.21)	1.01 (0.89 - 1.15)	0.86 (0.74 - 0.99)
NO_2			
Crude effect	0.94 (0.85 - 1.04)	0.93 (0.84 - 1.03)	0.84 (0.76 - 0.94)**
Adjusted [1]	0.70 (0.57 - 0.85)***	0.78 (0.65 - 0.94)*	0.66 (0.55 - 0.80)***

Notes:
** p value < 0.05. ** p value < 0.01. *** p value < 0.001.*
1. Adjusted for - gestational age, neonate gender, mother's age, parity, indigenous status, number of antenatal visits, marital status, previous abortions/miscarriages, index of SES, season of birth.
Inter-quartile ranges in the maternal exposure estimate -
PM_{10} ($\mu g/m^3$) Month one = 4.2, Month two = 5.0, Month three = 5.5; bsp (Mm^{-1}) Month one = 8.7 Month two = 10.0, Month three = 10.2
O_3 (ppb) Month one = 7.2, Month two = 7.3, Month three = 8.1; NO_2 (ppb) Month one = 5.5, Month two = 5.5, Month three = 5.4

(results not shown). This suggests that the increased risk associated with second-month PM_{10} did not occur because it was part of an erratic pattern across the nine months. A similar pattern was observed for bsp exposures across the nine months. Both NO_2 and O_3 were not adversely associated with a reported congenital anomaly.

Discussion

The main result showed that particulate matter during month two of pregnancy was associated with an increased risk of being born with a congenital anomaly. As reported earlier, only two other studies have investigated the effect of temporal variations in ambient air pollution on birth defects and despite inconsistency across different pollutants, both studies reported an association during a similar window of exposure to the current study. In Southern California, ambient carbon monoxide (CO) during month two was associated with cardiac ventricular septal defects while O_3 during month two was associated with aortic artery and valve defects, pulmonary artery

and valve anomalies, and conotruncal defects. No effect was found for PM_{10} and NO_2 exposures in Southern California (Ritz et al. 2002). In Texas, ambient CO during weeks 3 to 8 of pregnancy was associated with multiple conotruncal defects and teratology of fallot while PM_{10} was associated with isolated atrial septal defects, and sulfur dioxide (SO_2) was associated with isolated ventricular septal defects (Gilboa et al. 2005).

Despite the reported PM_{10} concentrations being highest in Southern California, there was no effect found with PM_{10}, yet there was an effect with PM_{10} in Texas and in the current study. As suggested by the authors of the Texas study (Gilboa et al. 2005), this inconsistency may be due to heterogeneity in the source of particles and the fetotoxic chemicals that adhere to their surface. This may provide an explanation as to why an effect was found in Brisbane, despite the low PM_{10} concentrations.

The biological mechanisms whereby air pollution may cause birth defects remain to be explained. Based on blood samples collected from mothers, placentas, and umbilical cords there is a growing body of evidence that shows maternal exposure to air pollution is associated with DNA damage (Perera et al. 1998; Perera et al. 2002; Perera et al. 2004; Topinka et al. 1997; Whyatt et al. 1998). The majority of studies that have investigated damaged DNA and air pollution have focused on particulate matter as the link may be in the chemical composition of particles. Polycyclic aromatic hydrocarbons, which are highly biologically active compounds, make up part of the composition of particles and are important to mutagenicity (McDonald et al. 2004), especially with regard to damaged DNA during pregnancy (Perera et al. 1999). In fact, various chemical properties of vehicle emissions may be independently associated with mutagenicity and lung toxicity (McDonald et al. 2004).

It is important to note that NO_2 during month three was a strong protective factor for being born with a congenital anomaly. However, season of birth had a strong affect on the NO_2 exposure estimates, as NO_2 is a seasonal pollutant that peaks during winter and troughs during summer. Also, a possible explanation for this result may be the negative correlation between month two PM_{10} and month three NO_2, yet the correlation was very weak (r = -0.15, p = <0.001). While there is no biological plausibility that supports NO_2 being a protective factor for congenital anomalies, it highlights the importance of interpreting these results with caution and due to the multiple comparisons performed in the overall Brisbane birth outcome study there is the increased possibility that these results occurred by chance.

The current study has several limitations. First, due to ethical reasons information regarding the type of congenital anomaly reported was unavailable and as there are a wide variety of congenital anomalies that are not associated with environmental exposures, further research examining specific anomalies is required.

Second, direct exposure to air pollution could not be assessed and based on a citywide average of available data, surrogate measures of ambient air pollution were used. Although this enhances the possibility of exposure misclassification, it is most likely random and the likely consequence is that the pollutant effect is actually underestimated. This limitation is well acknowledged in air pollution-birth outcome research (Bobak 2000; Liu et al. 2003; Sagiv et al. 2005). In addition, data for CO and SO_2 were limited for the study period and could not be examined and these pollutants were associated with birth defects in the American studies (Gilboa et al. 2005; Ritz et al. 2002).

Third, it could be argued that these results are confounded by various maternal lifestyle factors such as smoking (direct and passive) and alcohol consumption during pregnancy. However, due to the seasonal patterns in

ambient air pollution, when the maternal air pollution exposure estimates are derived from temporal variations these factors are not confounders because they are constant over time and are therefore most likely not associated with the exposure estimate. Similar unpublished air pollution-birth outcome research by the author on a smaller cohort of births in Brisbane (from the Mater-University of Queensland Study of Pregnancy), where detailed information on various lifestyle variables were examined, showed that maternal lifestyle factors such as smoking during pregnancy were not associated with the exposure estimate and the air pollution effect estimate for the examined birth outcome did not change when these factors were controlled for in the analyses.

In conclusion, there was evidence suggesting that particulate matter in Brisbane during the second month of pregnancy is associated with an increased risk of delivering a neonate with a congenital anomaly. Despite the limitations of the current study and the fact that these results may have occurred by chance, it needs to be reiterated that the exposure period associated with these results is consistent with the two American studies (Gilboa et al. 2005, Ritz et al. 2002). The aim of this brief report is to call attention to the need for further research focusing on specific congenital anomalies in relation to maternal exposure to ambient air pollution during pregnancy in Brisbane.

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Public Health Impact of Diesel Exhaust: Toxicity of Nano-sized Diesel Exhaust Particles - Part I

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Diesel exhaust particles (DEP) can exist in a range of particle sizes, such as PM_{10}, $PM_{2.5}$, and nanoparticles. It is also suspected that nano- or ultra fine particles make up a large part of particulate components in DEP. These particles can also make up a large component of ambient air pollution. Epidemiological and human studies have demonstrated an association between air pollution and various adverse health outcomes, including morbidity such as asthma, and mortality such as cancer, although the underlying biological mechanism is not well understood. There is currently no occupational exposure level for diesel fumes in the United Kingdom, however, Western Australia has a provisional standard and BHP and some other mines have adopted the recommended standard of 0.2 mg/m^3 for sub micron DPM or 0.1 mg/m^3 for elemental carbon. It is known that breathing high concentrations of diesel exhaust induces pulmonary inflammation, broncho-constriction, and oxidative stress. Current research practice mainly focuses on two particle sizes namely: coarse (2.5-10μ) and fine (< 2.5μ). It is suggested that more research should be undertaken on smaller nanoparticles (<100 nm) because it is hypothesised that they could cause more harm.

Key words: *Diesel Exhaust Particles (DEP); Nanoparticles; Air Pollution; PM_{10}; $PM_{2.5}$; Epidemiology*

Following a review of air quality standards in the United States in 1997 the US Environmental Protection Agency (US EPA) decided to retain the 1987 PM_{10} standard and proposed and adopted a new standard for fine particles, $PM_{2.5}$. Earlier, in 1995 the Expert Panel on Air Quality Standards of the United Kingdom (UK) had reviewed the emerging evidence and recommended the adoption of a PM_{10} standard (Greenbaum 2003).

So at the present time and for the past 10 years we have had researchers and government authorities focusing on environmental particles of these two sizes of particulate matter (PM). However, in current research practice there seems to be three particle sizes investigated, namely: coarse (2.5-10μ), fine (<2.5μ), and ultrafine (nanoparticles <0.1μ) (Li et al. 1996; Oberdorster 2001). It might be prudent to review the current exposure standards especially in light of new research on the issue of nanomaterials, and their effects on public and occupational health.

Nanoparticles are particles with diameters between 1 and 100 nm (1nm=1/1000 μm). Nanoparticles might be suspended in a gas (as a nanoaerosol), suspended in a liquid (as a colloid or nano-hydrosol), or embedded in a matrix (as a nanocomposite). The precise definition of 'particle diameter' depends on particle shape as well as how the diameter is measured (NIOSH 2005).

Particles of this size take on novel properties and functions that differ markedly from those seen in the bulk material and allow them to perform differently in terms of such attributes as conductivity, reactivity, and optical sensitivity. Nanoparticles are able to interact with complex biological functions operating at the scale of biomolecules, and therefore have the

potential to generate toxicity (Cheng 2004; McNeil 2005; Nel et al. 2006).

Diesel engines are known to emit high levels of nanoparticles (diameter less than 50 nm), but the physical and chemical mechanisms by which they form are not well understood. This information is lacking because of the small size of these particles and low mass, which makes nanoparticles difficult to analyse (Tobias et al. 2001). Diesel particulate nanoparticles can be classified as combustion-derived nanoparticles (CDNP). CDNPs have a large surface area and they contain metals and organics; these have the potential to produce oxidative stress and might exert genotoxic effects. CDNP and their components once in the body, have the ability to translocate to the brain and other organs via the blood, and therefore can cause a range of adverse reactions (Donaldson et al. 2005).

It is known that diesel particles can exist in a wide range of particle sizes, such as PM_{10}, $PM_{2.5}$ and as varying sized nanoparticles. Diesel particles form a large component of the PM_{10} in urban air in the UK (Murphy et al. 1998) and they also might make up a large part of ultrafine particulate air pollution in urban areas (Steerenberg et al. 1998). Dynamometer studies have shown that diesel engines are significant sources of ultrafine particles less than 0.1μ in diameter (Gertler et al. 2002). Kim et al. (2001) found that PM from a turbocharged diesel engine contains a higher percentage of particles smaller than 100 nm, than that obtained from an air-aspirated diesel engine, and Vogt et al. (2003) found soot particles to have a mean diameter of approximately 50 nm in the exhaust plume of a diesel car.

Murr, Esquivel & Bang (2004) used transmission electron microscopy (TEM) to investigate several hundred individual particulates including aggregates of carbonaceous/diesel PM. They found that most airborne particulates were aggregates ranging in aerodynamic diameter from a few nanometres to a few microns, and that homogenous aggregates of diesel exhaust had spherule diameters between 10 and 30 nm. Kwon et al. (2003) also found that nanoparticles were in a diameter range of 30-70 nm in diesel exhaust emissions. In a review Ono-Ogasawara and Smith (2004) found that the number of ultrafine particles, less than around 100 nm in diameter in DEP, is dominant in contrast to mass size distribution.

Epidemiological studies have demonstrated an association between air pollution and various health outcomes including mortality, and asthma, although the underlying biological mechanisms are unclear. Of the motor vehicle generated air pollutants, DEP account for a highly significant percentage of the particles emitted in many towns and cities (Sydbom et al. 2001). There is extensive epidemiological evidence that increased levels of the inhalable particulate fraction of air pollution (PM_{10}) are associated with increased morbidity and mortality. To obtain an idea of the sizes of particles retained in human lung parenchyma, Churg and Brauer (1977) used analytical electron microscopy to count, size and identify particles in the upper lobe apical segment parenchyma of autopsy lung tissue from 10 non-smokers. The overall geometric mean particle size was 0.38μ. Ultrafine particles (those with a diameter < 0.1μ) made up less than 5% of the total and most of these were metals. It was also found that 96% of the particles had diameters less than 2.5μ. Probably, the nano-sized particles were absorbed and distributed to other parts of the body rather than staying in the sites as the larger ones.

Data from epidemiological studies have shown that allergic conditions have increased over the last 30-40 years, despite a decrease in the severity of grass pollen episodes. Other epidemiological investigations have indicated an interaction between allergic diseases and traffic pollution, and laboratory findings have shown that DEP enhances sensitivity to

allergens. Epidemiological studies in Japan have shown that atopic subjects living in urban areas are more likely to suffer from the effects of air pollution, with increased coughing, sputum production, wheezing and throat irritation. The incidence of allergic rhinitis and asthma have been shown to be greater in areas where there is heavy traffic and therefore high levels of motor vehicle exhaust emissions (Miyamoto 1997). Studies investigating cellular and subcellular mechanisms suggest that pollutants are likely to influence the actions and interactions of a variety of cells, and lead to the synthesis of proinflammatory chemicals that modulate the activity and functions of inflammatory cells (Davies, Rusznak & Devalia 1998). DEP appear to have greater immunological effects in the presence of environmental allergens than they do alone (Pandya et al. 2002). It is not known whether nanoparticles can contribute to the development of allergic reactions.

Diesel exhaust particles can act as non-specific airway irritants at relatively high concentration levels. At lower concentrations, DEP promotes the release of specific cytokines, chemokines, immunoglobulin, and oxidants in the upper and lower airways. Breathing high concentrations of diesel exhaust (DE) induces pulmonary inflammation, bronchoconstriction, increased airway reactivity, and oxidative stress (Mudway et al. 2004). DE is composed of gaseous and particulate matter and nanoparticles are an important component. Data on 5-aminolevulinic acid (ALA) synthesis and heme formation in lymphocytes from groups of 50 miners exposed to DE and 50 unexposed surface workers were obtained by Muzyka et al. (2004). The levels of benzene, carbon monoxide, and nitric oxide in air as well as concentrations of 1-nitropyrene and elemental carbon in PM were used as markers of exposure to DE in the mine. Significant differences in the activity of ALA synthesis and heme formation between exposed miners and surface workers were found, showing the potential harmful effects of exposure to DE.

Behndig et al. (2006) investigated whether antioxidants were up-regulated following a low dose DE exposure and examined how these responses related to the development of airway inflammation at different levels of the respiratory tract where particle dose varies significantly. A total of 15 volunteers were exposed to DE airborne PM with a diameter <10µ for 2 hours. Following DE exposure, it was observed that there was an increase in bronchial mucosa neutrophil and mast cell numbers, as well as an increase in the number of neutrophils, interleukin-8 (IL-8) and myeloperoxide concentrations in bronchial lavage. No inflammatory responses were seen in the alveolar compartments, but both glutathione and urate concentrations were increased. The contribution of nanoparticles within DE to the harmful effect is not well understood.

In comparing worldwide epidemiological studies, there seems to be a cohesive and consistent relationship between increases of particle concentration and the increase of morbidity and mortality. An increase in daily average PM_{10} (particles <10µ) is correlated with an increase in mortality (Brandli 1996). Epidemiological studies have also indicated that particles less than 10µ in diameter, are involved in the development of asthma and chronic obstructive pulmonary disease (COPD) although the mechanism is unknown (Churg & Brauer 1977). Diesel exhaust is a public health concern and a contributor to both ambient and occupational air pollution. There is currently no occupational exposure level for diesel fumes in the UK (Wheatley & Sadhara 2004), however, Western Australia has a provisional standard and BHP and some other mines have adopted the recommended standard of 0.2 mg/m^3 for sub micron DPM or 0.1 mg/m^3 for elemental carbon (Irvin 2005). It is important to understand the harmful effects of nanoparticles within DE for setting such safe exposure standards.

While much research work in this field has been carried out, researchers need to give more consideration to the toxic effects that might be caused by nano-sized particulate matter. Investigating only particulate matter of PM_{10}, and $PM_{2.5}$ might not be sufficient to protect the public from the harm of nanoparticles, especially with the increasing production of nanomaterials in industry (Cheng 2004; Donaldson et al. 2005).

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Public Health Impact of Diesel Exhaust: Toxicity of Nano-sized Diesel Exhaust Particles-Part II

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Diesel exhaust (DE) is a public health concern and a contributor both to ambient and occupational air pollution. There is currently no occupational exposure level for diesel fumes in the United Kingdom or Australia. Current research practice focuses on mainly three particle sizes, namely: PM_{10}, $PM_{2.5}$, and ultrafine (<0.1μ). It is 12 years since Oberdorster and Utell introduced their ultrafine particle hypothesis stating that ambient ultrafine particles might cause adverse health effects. It is suggested that more research should now be undertaken on the smaller particles (nanoparticles) less than 50 nm in diameter, because it is hypothesised that more harm could be caused by these particles. The effect of diesel exhaust and diesel exhaust particles (DEP) on various in vivo animal models is discussed.

Key words: *Diesel Exhaust Particles (DEP); Diesel Exhaust (DE); Nanoparticles; Air Pollution; Inflammation; Toxicity*

There is evidence that particulate air pollutants such as diesel exhaust particles (DEP) can enhance the effects of chronic inflammatory processes as well as acute symptom responses in the respiratory tract. Diesel exhaust (DE) is recognised as a public health concern and a contributor to both ambient and occupational air pollution (Churg & Brauer 1997; Li et al. 1996). DE is composed of gaseous and particulate matter and nanoparticles are an important component. In current research practice there seems to be mainly three particle sizes investigated, namely: PM_{10} (2.5-10μ), $PM_{2.5}$ (<2.5μ) and ultrafine (<0.1μ) (Oberdorster 2001; Li et al. 1996). In 1994 Oberdorster and Utell announced their ultrafine particle hypothesis that these sized particles could cause harm (Oberdorster & Utell 2002). We think that it might be time to renew the research focus on the smaller particles, in particular on the effects of nanoparticles on public and occupational health.

Diesel engines are known to emit high concentrations of particles with a diameter less than 50 nm, but the biological properties of these small particles are not well understood (Tobias et al. 2001). Diesel particles can exist in a wide range of particle sizes, such as fine (<2.5μ), ultrafine (nanoparticles) (<0.1μ), and as varying sized nanoparticles. They also form a large component of the fine particle fraction in urban air and may be an important part of ultrafine particulate air pollution in urban areas (Murphy et al. 1998; Steerenberg et al. 1998).

Nanoparticles are particles with diameters between 1 and 100 nm (1 nm= 1/1000μm). Particles of this size take on novel properties and functions that differ significantly from those seen in the bulk material and allow them to perform differently. The novel nature of nanoparticles offers the ability to interact with complex biological functions in new ways, operating at the scale of biomolecules, for example proteins and DNA, and therefore has the potential to generate toxicity (Cheng 2004; McNeil 2005; Nel et al. 2006). In the size range <100 nm, the number of surface molecules is

inversely related to particle size. For example, in a particle of 30 nm size, about 10% of its molecules are expressed on the surface, whereas at 10 and 3 nm, the ratio increases to 20% and 50% respectively. Because the number of atoms or molecules on the surface of the particles may determine the material reactivity, it is thought that this may be the key to defining their chemical and biological properties (Oberdorster et al. 2005).

Particle exposure has traditionally been monitored as mass concentration of PM_{10} and as $PM_{2.5}$. The mass concentration is strongly influenced by the large particles. Therefore particle mass is not a good measure for characterising the amount of the small, possibly more biologically toxic particles. Nygaard et al. (2004) used polystyrene particles (PSP) ranging in diameter from 0.0588 to 11.14µ, carbon black (CB) and diesel exhaust particles (DEP) to study the effect of particles on the immune response to the allergen ovalbumin (OVA) in BALB/cA mice. At a given mass dose, the small particles (0.0588 and 0.202µPSP, CB and DEP) increased the allergen-specific immunoglobulin E (IgE) serum levels to a significantly higher degree than the large particles (1.053, 4.64, and 11.14µ). The fine particles (0.202µ) with OVA increased cell numbers, expression of surface markers, production of interleukin-4 (IL-4) and IL-10 whereas the largest (11.14µ) did not. The authors found that the fine particles exerted stronger effects on allergic responses than larger particles at equal mass doses, and suggest that the dose described as total particle surface area or particle number better predicts the effect of particles than particle mass.

Dong et al. (2005) carried out a study in which Brown Norway rats were first sensitised to ovalbumin (OVA) for 30 min and then exposed to either filtered air or DEP (22.7 mg/m^3) for 4h/day for 2 days and then challenged with OVA. Ovalbumin sensitisation and challenge resulted in a significant infiltration of neutrophils, lymphocytes and eosinophils into the lung. Diesel exhaust particles pre-exposure augmented OVA-induced production of allergen-specific immunoglobulin E (IgE) and IgC and pulmonary inflammation characterised by significant increases in T-lymphocytes and infiltration of eosinophils after OVA challenge whereas DEP alone did not produce these effects. Combined DEP and OVA exposure produced significant airway hyper-responsiveness in this animal model. The authors suggest these responses show that DEP pre-exposure aggravates the allergic responses to the subsequent challenge with OVA and might, in part, be attributed to the generation of reactive oxygen species (ROS) in alveolar macrophages and alveolar type II cells.

To elucidate the mutagenicity of DE, Sato et al. (2000) estimated mutant frequency and determined the mutation spectra in rat lung after 4 weeks exposure to 1 or 6 mg/m^3 DE, which contained suspended PM using male Big Blue rats. After exposure to 6 mg/m^3 DE, the mutation frequency (MF) in the lung was 4.8 fold higher than control rats, but no increase in MF was observed in rats exposed to 1 mg/m^3 DE. Sixty-nine mutants were identified after exposure to 6 mg/m^3. The authors also found that the relative adduct level and amount of 8-deoxy-hydroxyguanosine (8-OHdG) were significantly increased in the lungs of the rats exposed to DE, and lesions in genomic DNA was observed. The mutagenicity of DEP was studied by Song and Ye (1995) using the Salmonella assay in vitro and mice micronucleus in vivo test. The results of both assays showed that DEP had mutagenic activity.

Han et al. (2001) provided the first direct evidence of hydroxyl radical OH generation in the lungs by living mice after tracheal instillation of DEP, using non-invasive L-band ESR spectroscopy. They found evidence that the intratracheal exposure to DEP in these mice produced hydroxyl radicals in the lungs through an iron-catalysed reaction of superoxide or hydrogen peroxide.

In a study carried out by Tokiwa et al. (1999) diesel particle extracts (particle size 1.47-1.05μ) were found to show mutagenicity for YG3003 mice, a sensitive strain to some oxidative mutagens. Formation of 8-OHdG as a biomarker of oxidative damage was analysed with an in vivo assay system. They found that carbonaceous particles, but not mutagens or carcinogens, promote the formation of 8-OHdG, and that as a mechanism, alveolar macrophages (AM) might be involved in oxidative damage. The authors suggest that the oxidative damage may be due to the fact that the mutation is involved with the generation of hydroxyl radicals during phagocytosis.

Dybdahl et al. (2004) investigated the short-term effects of DEP on markers of inflammation and genotoxicity in vivo. Mice were exposed by inhalation to 20 or 80 mg/m3 DEP either as a single 90 min exposure or as 4 repeated 90 min exposures (5 or 20 mg/m^3) and the effects in broncho-alveolar lavage (BAL) cells and lung tissues were examined. Inhalation of DEP induced a dose-dependent inflammatory response with infiltration of macrophages and neutrophils and elevated gene expression of interleukin-6 (IL-6) in the lungs of the mice. The inflammatory response was associated with DNA strand breaks in BAL cells and oxidative DNA damage and increased levels of bulky DNA adducts in lung tissue, indicative of genotoxicity.

DEP can enhance various respiratory diseases, but it is not clear as to which components of DEP are associated with the enhancement. Yanagisawa et al. (2006) studied the effects of DEP components on antigen-related airway inflammation, using residual carbonaceous nuclei of DEP after extraction (washed DEP), extracted organic chemicals in DEP (DEP-OC), and DEP-OC plus washed DEP (whole DEP) in the presence or absence of ovalbumin (OVA). DEP were extracted with benzene-ethanol. Briefly 10g of DEP were suspended in 800 ml of benzene-ethanol (3:3) and were ultrasonicated for 30 min. The suspension was then centrifuged at 600 g for 20 min. The supernatants were transferred to another tube, and were then filtered (0.45_m). The residual particles of DEP were prepared as washed DEP. The extracts were evaporated and dissolved in dimethyl sulfoxide (DMSO) and prepared as DEP-OC. Some of the OC are polycyclic aromatic hydrocarbons (PAH), nitroarenes, and dinitroarenes. Male ICR mice were intratracheally instilled with OVA and/or DEP components. DEP-OC, rather than washed DEP, enhanced infiltration of inflammatory cells into broncho-alveolar lavage (BAL) fluid, size of airway inflammation, and proliferation of goblet cells in the airway epithelium in the presence of OVA. Washed DEP with OVA showed less change, but increased the lung expression of IFN-γ. The combination of whole DEP-OVA caused the most significant changes in the entire enhancement. The authors found that DEP-OC, rather than washed DEP exaggerated allergic airway inflammation through the enhancement of T-helper type 2 responses. The coexistence of organic chemicals with carbonaceous nuclei caused the most marked aggravation. Although the chemicals involved have not been clearly defined, the authors suggest that DEP components might diversely affect various types of respiratory diseases, while whole DEP might mostly aggravate respiratory diseases. In addition, no detailed description of extraction method was provided. A better knowledge of the OC present will be useful in assessing the mode of interaction with biomolecules.

Ishihara and Kagawa (2003) studied long-term exposures to diesel emissions with respect to dose-dependence and the effect of components without particles from diesel emissions in Wistar rats for periods of 16h/day, 6days/wk, for 6, 12, 18 or 24 months. Changes in total cell counts and cell differentiation, total protein, mucus and surfactant components, and prostaglandin E_2

in BAL fluid, but not biomarkers in plasma, showed differences among the groups of rats during the experimental period. The authors also indicated that biological response to inhaled particles aggregated during chronic exposure to DE (<1.0mg/m^3) and inflammation and overproduction of mucus and surfactant components reached a plateau at 12-18 months of exposure during the 24-month period, and that PM plays an important role during development of lung injury induced by DE.

The NC/Ngc mouse is known to be a typical animal model for human atopic dermatitis (AD). Inoue et al. (2005) studied the effect of repeated pulmonary exposure to DEP on airway inflammation and cytokine expression in these mice. Cellular components of BAL fluid and expression of cytokines and chemokines in both the BAL fluid and lung tissues were evaluated 24 hours after the last instillation. DEP exposure significantly induced the lung expression of IL-4, keratinocyte chemo-attractant, and macrophage inflammatory protein-1 α when compared to controls. The authors suggest these results show that intratracheal exposure to DEP induces the expression of inflammatory cells, through the expression of IL-4 and chemokines in these mice.

Yin et al. (2005) conducted a study to examine whether DEP exposure exerts a sustained effect in which inhaled DEP increases the susceptibility of the lung to bacterial infection occurring at a later time. Brown Norway rats were exposed to filtered air or DEP (21.2 mg/m^3) inhalation for 4 h/day for 5 days and intratracheally instilled with saline or *Listeria* seven days after the final DEP exposure. The results showed that inhaled DEP prolonged the growth of bacteria in the lung as compared to the air-exposed controls. Pulmonary responses to *Listeria* infection were characterised by increased production of IL-1ß, tumor necrosis factor alpha (TNFα), IL-12, and IL-10 by AM and increased presence of T-lymphocytes in lung lymph nodes. We suggest that this study demonstrates that inhaled DEP, after a 7-day resting period, increased the susceptibility of the lung to bacterial infection occurring at a later time by inhibiting macrophage immune function and suppressing the development of T-cell mediated immune responses.

Yokoto et al. (2005) investigated the effect of DEP on peripheral neutrophil count and on the oxyradical production (ORP) of neutrophils in rats. Instillation of 5 mg DEP elevated circulatory neutrophil counts at 12 and 24 h post-instillation to levels of 2.1 and 2.3 fold respectively. Cytokine-induced neutrophil chemo-attractant-1, tumor necrosis factor-alpha (TNF-α) and macrophage inflammatory protein-2 levels were increased in BAL. The authors suggest that these results show that intratracheal instillation of DEP enhances systemic oxidative stress by increasing neutrophil count and ORP in the acute period.

Thioredoxin-1 (Trx-1) is a thiol protein with antioxidant and redox-regulating effects. A study by Kaimul-Ashan et al. (2005) demonstrated that Trx-1 scavenges reactive oxygen species (ROS) generated by DEP and attenuates lung injury. Intratracheal instillation of DEP resulted in the generation of more hydroxyl radicals in control mice than in human Trx-1 transgenic mice. DEP caused acute lung damage with a large infiltration of inflammatory cells in control mice, but much less damage in human Trx-1-transgenic mice. The human Trx-1 transgene protected the mice against DEP toxicity. The down-regulation of Akt (signalling factor-inhibits apoptosis) phosphorylation by DEP resulted in apoptosis, which was prevented by Trx-1. The authors suggest that Trx-1 exerts antioxidant effects and that this plays a role in protection against DEP-induced lung damage by regulating Akt-mediated anti-apoptotic signalling.

A study found that non-particulate compounds in whole DE elicit electrocardiogram (ECG) changes

consistent with myocardial ischemia, in mice exposed 6h/day for 3 days to DE (0.5 and 3.6 mg/m^3) in whole-body inhalation chambers with or without particulate filters. Significant bradycardia and T-wave depression was observed, regardless of the presence of particulates. We think that the volatile organic compounds in the vapour phase caused enhanced constriction and reduced dilation in isolated coronary arteries caused by non-particulate components of DEP (Campen 2005).

While a lot of research has been carried out in this area, researchers need to give more consideration to the toxic effects that might be caused by nano-sized particulate matter. Much of the research work cited here and in the literature involves particulate matter of PM_{10}, $PM_{2.5}$. Investigating only particulate matter of this size might not be adequate to protect the public and the workplace from the harm of nanoparticles, much of which is produced by diesel engines.

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Public Health Impact of Diesel Exhaust: Toxicity of Nano-sized Diesel Exhaust Particles-Part III

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Diesel exhaust particles (DEP) form a large component of the fine particle fraction in urban air and might constitute an important part of ultrafine (nanoparticle) particulate ambient and occupational air pollution. It is known that breathing high concentrations of diesel exhaust (DE) induces pulmonary inflammation, broncho-constriction, and oxidative stress. Epidemiological, human and in vivo studies have demonstrated an association between air pollution and various adverse health outcomes such as asthma and lung cancer, although the underlying biological mechanism is not well understood. Current research practice focuses on mainly three particle sizes, namely: PM_{10}, $PM_{2.5}$ and ultrafine (<0.1μ). It is suggested that more research should be undertaken on the smaller particles such as nanoparticles (diameter less than 100 nm), because it is hypothesised that more harm could be caused by these particles. The effect of diesel exhaust and diesel exhaust particles on various in vitro systems is discussed.

Key words: *Diesel Exhaust Particles (DEP); Diesel Exhaust (DE); Nanoparticles; Air Pollution; In Vitro Systems*

Diesel engines are known to emit high concentrations of nanoparticles (diameter less 100 nm), but the biological properties of these small particles is not well understood (Tobias et al. 2001). Diesel exhaust particles (DEP) can enhance the effects of chronic inflammatory processes as well as acute symptom responses in the respiratory tract, and diesel exhaust (DE) is recognised as a public health concern and a contributor to both ambient and occupational air pollution (Li et al. 1996; Churg & Brauer 1997). DE is composed of gaseous and particulate matter and nanoparticles are an important component. In current research practice there seems to be mainly two particle sizes investigated, these are PM_{10}, $PM_{2.5}$. We think that it might be time to revise our research focus especially in the face of the emergence of nanomaterials, in particular the effects of nanoparticles on public and occupational health.

Nanoparticles are particles with diameters between 1 and 100 nm (1 nm= 1/1000μ). Particles of this size take on novel properties and functions that differ significantly from those seen in the bulk material and allow them to perform differently. Nanoparticles have the ability to interact with complex biological functions in new ways, operating at the scale of biomolecules, e.g. DNA and proteins, and therefore have the potential to generate toxicity (Cheng 2004; McNeil 2005; Nel et al. 2006).

The soluble organic fraction of PM from diesel exhaust (DE) contains hundreds of organic constituents, particularly in the moderately polar transition fraction. The polar transition fraction contains mainly polycyclic aromatic hydrocarbon (PAH) derivatives consisting of hydroxyls, quinones, and acidic hydride compounds of PAH. Mutagenicity tests using the Ames *Salmonella* assay show that the transition fraction accounts for most of the mutagenicity when compared to other fractions (Schuetzle et al. 1981). Nakagawa et al. (1983) have identified the mutagens to be probably 1,6 and 1,8 dinitropyrenes. Quinones are reactive organic compounds

and are known to initiate reactions associated with many toxicological events. Four quinones found in DEP are 1,2-naphthoquinone, 1,4-naphthoquinone, 9,10-phenanthraquinone and 9,10-anthraquinone. Mean concentration of these quinones in DEP ranged from 7.9-40.4 μg/g (Cho et al. 2004).

A bioassay-directed chemical analysis that consists of mammalian cell bioassays in conjunction with analytical measurements, was performed by Oh and Chung (2005) to identify the most biologically active compounds of DEP on mutagenic activity. They found that mutagenic assays showed that the aromatic and slightly polar fraction of DEP induced chromosomal damage and DNA breakage in a non-cytotoxic dose. It was also observed that indirect-acting mutagens mainly contribute to the mutagenic effect of the aromatic fraction by the enzyme metabolism system. In the aromatic fraction several indirect acting mutagenic PAH such as dibenzo (a,h) anthracene, chrysene, and 1,2-benzanthracene were detected. We suggest that the aromatic fraction might be responsible for the genotoxicity of extracts of DEP.

DE particles from two sources were dispersed in aqueous mixtures of dipalmitoyl phosphatidyl choline, a major component of pulmonary surfactant, and were tested for genotoxicity by Keane et al. (1991). Both types of extracts yielded similar results in both the *Salmonella* mutagenicity assay and the sister-chromatid exchange assay using V79 cells. We believe that the results indicate that genotoxic activity associated with diesel particles inhaled into the lungs might be made available due to the solubilisation/dispersion properties of pulmonary surfactant compounds.

Hirafuji et al. (1995) examined the direct action of DEP on isolated tissues and the cytotoxicity of DEP on cultured cells of respiratory tracts of guinea pigs. DEP induced a dose-dependent relaxation in tracheal smooth muscle and lung parenchymal preparations from guinea pigs. DEP also showed concentration and time-dependent cytotoxicity on cultured tracheal smooth muscle cells and lung fibroblasts. We believe that the results indicate that the cytotoxicity of DEP might cause dysfunction of respiratory tissues, which are mediated by oxygen radicals, probably hydroxyl radicals or hydrogen peroxide.

In a study by Steerenberg et al. (1998) the effects of in vitro DEP exposure (0.04-0.33 mg/ml) on interleukin-6 (IL-6), IL-8 production by a human bronchial epithelial cell line (BEAS2B) was investigated. Transmission electron microscopy (TEM) showed that the size of the DEP particles ranged between 25 and 35 nm and that DEP was phagocytosed by BEAS2B cells. An increase in IL-6 and IL-8 production was 11 and 4-fold respectively, after 24 and 48h of exposure to DEP compared to non-exposed cells. To study the DEP effect on inflammation-primed cells, BEAS2B cells were exposed to both tumor necrosis factor-alpha (TNF-α) and then to DEP. Additive effects on the IL-6 and IL-8 production by the cells were found after TNF-α priming and subsequent exposure to DEP, and TNF-α (0.05-0.2 ng/ml).

Hiura et al. (2000) showed that methanol extracts made from DEP induce apoptosis and reactive oxygen species (ROS) in pulmonary alveolar macrophages and RAW 264.7 cells. Because DEP-induced apoptosis followed cytochrome c release, they studied the effect of DEP compounds on mitochondrially regulated death mechanisms. DEP extracts induced ROS production and upset mitochondrial function before and at the onset of apoptosis. This mitochondrial upset follows an orderly sequence of events, which starts with a change in mitochondrial membrane potential, followed by cytochrome c release, and development of membrane asymmetry. Structural damage to the mitochondrial inner membrane is shown by a decrease in cardiolipin mass, leading to O_2 generation and uncoupling of oxidative

phosphorylation, as shown by a decrease in intracellular ATP levels. We suggest that DEP chemicals induce apoptosis in macrophages by a toxic effect on mitochondria.

Amakawa et al. (2003) tested the effect of DEP on the release of cytokines from alveolar macrophages (AM). Human and murine AM were exposed to DEP (10 μg/ml) for 24 h and the concentrations of tumor necrosis factor alpha (TNF-α), interleukin (IL)-6, and IL-8 were measured by an enzyme-linked immunosorbent assay. DEP suppressed the spontaneous release of TNF-α and IL-6 from murine AM. Soluble components of DEP had a similar suppressive effect, indicating that the chemical composition of DEP is responsible for the effect.

Block et al. (2004) found that mesencephalic neuron-glia cultures treated with DEP (5-50μg/ml) resulted in a dose-dependent decrease in dopaminergic (DA) neurons, as determined by DA-uptake assay and tyrosine-hydroxylase immuno-cytochemistry. The role of microglia was shown by the failure of neuron-enriched cultures to show DEP-induced DA neurotoxicity, where DEP-induced DA neuron death was reversed with the addition of microglia to the culture. Intracellular reactive oxygen species (ROS) and superoxide were produced from enriched microglia cultures in response to DEP. Neuron-glia cultures from NADPH oxidase deficient mice were insensitive to DEP neurotoxicity. It appears that DEP selectively damages DA neurons through the phagocytic activation of microglial NADPH oxidase and subsequent oxidative attack.

Many studies focus on the biological properties of DEP-extractable components although it is possible that chemical properties inherent to the DEP itself can lead to toxicity. Pan et al. (2004) looked at the chemistry inherent to DEP. They found that DEP are capable of catalysing the consumption of oxygen by ascorbate and thiol leading to the generation of reactive oxygen species (ROS) and also they can catalyse DNA strand breakage via O2 and reductant-dependent processes. DEP were found to have paramagnetic properties and this might be important for their ability to catalyse the formation of ROS that might partially be responsible for their toxicity.

Xia et al. (2004) demonstrated that aliphatic, aromatic, and polar organic compounds, fractionated from DEP exert differential toxic effects in RAW 264.7 cells. Cellular analysis showed that the quinone-enriched polar fraction was more potent than the polycyclic aromatic hydrocarbon (PAH) enriched aromatic fraction in superoxide generation, decrease of membrane potential, loss of mitochondrial membrane mass, and induction of apoptosis. These chemical effects on isolated mitochondria could be replicated by intact DEP. We suggest that the DEP effects are mediated by adsorbed chemicals rather than by the particles themselves.

Furuyama et al. (2005) studied the production of heme oxygenase-1 (HO-1) and factors related to the fibrinolytic function by rat heart microvessel endothelial cells exposed to organic extracts of DEP, and urban fine particles (UFP), to investigate the direct effects of these soluble organic fractions in these PM on the fibrinolytic function of endothelial cells. The cell monolayer exposed to 10μg/ml DEP produced a larger amount of HO-1 than cells exposed to 10μg/ml UFP. DEP and UFP exposure reduced plasminogen activator inhibitor-1 (PAI-1) production by the cells but did not affect the production of thrombomodulin, and tissue-type plasminogen activator. Increased PAI-1 synthesis in response to treatment with 1.0 μg/ml tumor necrosis factor-alpha (TNF-α) was reduced by DEP. We suggest that exposure to the soluble organic fraction of PM and DEP induced oxidative stress and reduced the PAI-1 production of endothelial cells.

Munandhara et al. (2005) investigated the effects of DEP exposure on the human alveolar macrophage (AM) response to lipopolysaccharide (LPS) (from gram negative bacteria) in vitro, which was determined by monitoring the production of interleukin-8 (IL-8), TNF-α, and prostaglandin E_2 (PGE_2). DEP induced a decreased secretion of IL-8, TNF-α, and PGE_2 in response to LPS stimulation. DEP also showed a suppressive effect on the release of inflammatory mediators when stimulated with lipoteich acid (a product of gram positive bacteria). We think that this effect might contribute to the impairment of pulmonary defences.

Terada et al. (1999) studied the effects of DEP extract on the expression of histamine H1 receptor (H1R) mRNA in human nasal epithelial cells (HNEC) and human mucosal microvascular endothelial cells (HMMEC) and on the production of IL-8 and granulocyte macrophage colony-stimulating factor (GM-CSF) induced by histamine. HNEC and HMMEC monolayers were cultured in the presence or absence of DEP extract for 3-24 h. DEP extract increased the expression of H1R mRNA in both cell types. The amount of IL-8 and GM-CSF induced by histamine was significantly higher in DEP extract pre-treated HNEC and HMMEC than non-treated cells. We suggest that DEP accelerates the inflammatory change by directly up-regulating H1R expression and increasing histamine-induced IL-8 and GM-CSF production.

By their ability to generate ROS in macrophages and epithelial cells, DEP might lead to a worsening of the asthmatic condition. Xiao et al. (2005) looked for evidence of oxidative modification of proteins in a RAW 264.7 cell line treated with DEP chemicals. They found that the induction of oxidative stress is accompanied by 53 newly expressed proteins that are suppressed by thiol antioxidant, N-acetylcysteine. These include antioxidant enzymes, pro-inflammatory components and products of intermediatory metabolism. Inducible nitric oxide (NO) synthase was identified as a biologically relevant oxidative stress protein that is induced at the same time with increased NO production and protein tyrosine-nitration in DEP exposed RAW 264.7 cells. We think that these oxidative proteins might serve as markers for oxidative stress generation.

Ito et al. (2006) hypothesised that DEP might induce the expression of receptors for viruses and bacteria at invasion sites. They investigated the effect of DEP on the mRNA expression of intercellular adhesion molecule-1 (ICAM-1), low density lipoprotein (LDL), and platelet-activating factor (PAF) receptors in rat lung epithelial cells. All of these mRNA were up-regulated by 3, 10, and 30 μg/ml of DEP in a concentration-dependent manner. The up-regulation of each was associated with the mRNA expression of heme oxygenase-1 (HO-1), a marker of oxidative stress. We think that these results show that DEP might enhance the risk of lung infections (e.g. pneumonia) by increasing the density of bacterial and viral invasion sites in the lungs.

Bayram et al. (2006) investigated the effects of DEP on proliferation and apoptosis of lung epithelial cells. When deprived of serum, epithelial cell numbers decreased, but DEP (5-200 μg/ml) prevented this. DEP (10 μg/ml) increased cells in the S phase of the cell cycle from 12.85% to 18.7% after 48 h, reversing the serum deprivation induced cell cycle arrest. DEP also reduced the increase in apoptotic cells observed after serum starvation (28.3 to 15.4%). DEP prevented serum-starvation-led decreases in epithelial cells by inducing cell cycle progression and preventing apoptosis, processes involving oxidative stress, inhibition of p 21 expression and stimulation of JNK and NF-kappa B. We suggest that low doses of DEP exposure might lead to lung epithelial cell hyperplasia.

While much research has been carried out in this field, researchers need to give more consideration to the toxic effects that might be caused by nano-sized particulate matter.

This is because much of the research work cited here and in the literature involves diesel exhaust particles of PM_{10} and $PM_{2.5}$ rather than nano-sized ones. Nano-sized particles are very different from their larger counterparts in biological behaviour and investigating only particulate matter of this size might not be adequate to protect the public and the workplace from the harm of nanoparticles.

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A Continuous Quality Improvement Approach to Indigenous Housing and Health

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There is a growing body of evidence on the effectiveness of modern continuous quality improvement (CQI) approaches in a variety of industries. The development of CQI theory has been paralleled by an increasing recognition of the value of ecological approaches to health promotion and community interventions. In this paper we propose a long term ecological and quality improvement approach to maximise the impact of housing programs on Indigenous health, and discuss how some current key initiatives could be developed to support such an approach. State and Commonwealth data systems on Indigenous housing are patchy and inconsistent. From an ecological perspective, there are three important gaps in information, which include a lack of indicators relating to a) hygiene promotion; b) workforce development; and c) information systems development. Factors that will be important in improving health through an ecological approach to housing-related interventions can be described in three categories: infrastructure factors; behavioural factors; and factors in the policy environment. Efforts to improve health through housing interventions should adequately target all three areas of influence concurrently. A CQI approach could provide a way for community and other organisations to be effectively engaged in housing initiatives.

Key words: *Continuous Quality Improvement; Housing; Indigenous Health; Ecological Approach; National Reporting Framework; Indicators*

Indigenous housing is a national issue requiring priority action, as recognised by the government housing ministers' statement on new directions for Indigenous housing, *Building a Better Future: Indigenous Housing to 2010* (HMACSCIH 2001). This recognition of the importance of housing conditions is in large part due to their significance as a major underlying determinant of the poor health status of Indigenous people. In this paper, we propose a long term ecological and quality improvement approach to maximise the impact of housing programs on Indigenous health, and discuss how some current key initiatives could be developed to support such an approach.

The Continuous Quality Improvement Approach

There is a growing body of evidence on the effectiveness of modern continuous quality improvement (CQI) approaches in the manufacturing (Adam et al. 1997; Samson & Terziovski 1999), service (Prajogo 2006) and health care industries (Blumenthal & Kilo 1998; Shortell et al. 1998). In general, the CQI approach aims to facilitate ongoing improvement by using objective data to analyse and improve processes (Graham 1995). Emphasis is placed on efficient and effective functioning of organisational systems. At the heart of quality improvement theory is the Plan-Do-Study-Act cycle; an ongoing cycle of gathering data to understand how well organisational

systems are functioning, and developing and implementing plans for improvement.

An essential starting point for quality improvement is the systematic and objective assessment of performance and of the systems in place to support good performance (McLaughlin & Kaluzny 1994). This requires producing good quality information to inform goal setting and the development of strategies to achieve improvements in identified areas.

Key features of modern CQI approaches make them well suited to the Indigenous Australian setting. The participatory approach and 'customer focus' of CQI fits with the requirement to take account of the principles and values of Aboriginal and Torres Strait Islander people, as expressed in recent national statements on research (National Health and Medical Research Council (NHMRC) (2003) and cultural respect (Australian Health Ministers' Advisory Council [AHMAC] 2004). The emphasis given to addressing underlying causes, capacity building (including community capacity to interact with data) and improving outcomes are also central to CQI (Blumenthal & Kilo 1998; Shortell et al. 1998) as is the development of positive models and a culture of self-evaluation rather than blame (Siddiqi & Newell 2005).

The Ecological Approach

The development of CQI theory has been paralleled by an increasing recognition of the value of ecological approaches to health promotion and community interventions (see for example Green & Kreuter 1999; McLeroy et al. 1988). Based on international work on hygiene improvement (EHP et al. 2004), the factors that will be important in improving health through an ecological approach to housing-related interventions are infrastructure factors; behavioural factors; and factors in the policy environment (Figure 1). Our 'Housing and Health Improvement' model (Wayte et al. 2005) suggests that efforts to improve health through housing interventions should adequately target all three areas of influence. Priorities in terms of access to hardware should be an adequate and safe water supply and sanitation facilities. Other hardware priorities might include those required to support healthy living practices, including whitegoods and furniture. Priorities for hygiene promotion include effective strategies for community participation, communication of risk and appropriate hygiene practices, social mobilisation/ marketing strategies, and health education and health promotion strategies that reach key groups in the population. Important issues in the policy environment include general support for development of a culture of continuous quality improvement, and more specifically for development of information systems, for building partnerships and capacity of community organisations, and constructive regulatory, financing and cost-recovery policies.

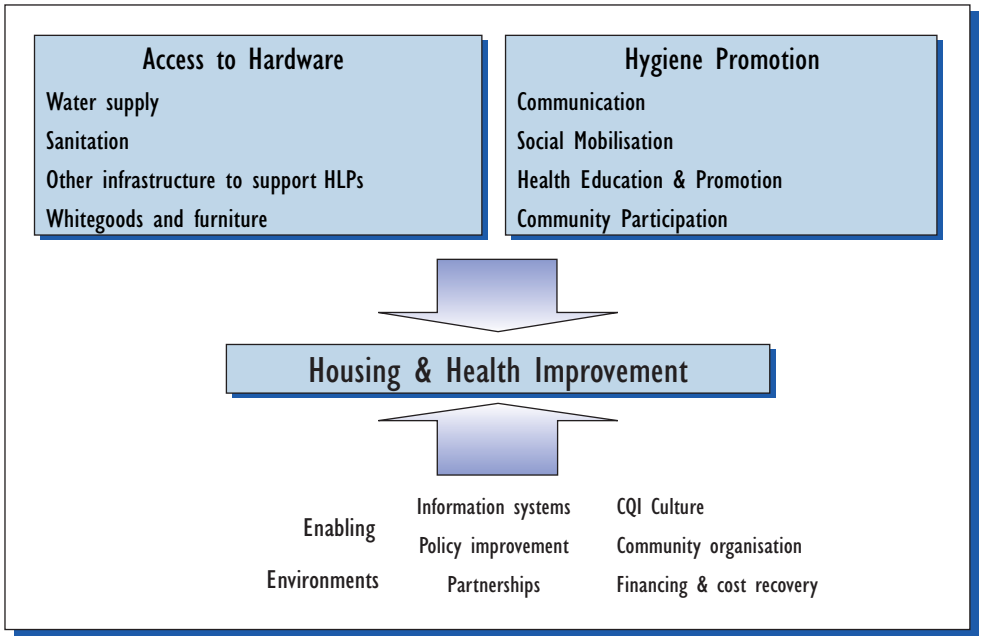
Applications to Housing and Health Improvement

All states and territories have agreed on a *National Reporting Framework* (AIHW 2004) for reporting on implementation and outcomes for all Indigenous housing programs. Identified priorities are data on crowding and dwelling condition (AIHW 2005). While the State and Territory level systems for collecting the data required for the *National Reporting Framework* have proved to be patchy and inconsistent, the definition of outcomes (HMACSCIH 2001), the definition of indicators (AIHW 2004), and the reporting of the completeness and quality of the data required for these indicators (AIHW 2005) are important advances.

Application of modern CQI theory to the proposed Housing Improvement Framework (Figure 1) would require information systems to report on indicators across all three areas: policy environment, infrastructure and home-management.

Figure 1: Housing and health improvement framework

An ecological approach includes interventions in all three areas of influence



Note: HLPs - healthy living practices

This would include indicators relating to systems development, processes and outcomes. However, the majority of the 38 indicators included in the *National Reporting Framework* relate to the condition and management of housing infrastructure, with four relating to the development of the Indigenous housing workforce, and one to the coordination between housing and other services that seek to improve the health and wellbeing of Indigenous people.

Three important gaps in the *National Reporting Framework* are the lack of indicators relating to a) hygiene promotion; b) general (including non-Indigenous) workforce development; and c) information systems development. One likely reason for the gaps in the *National Reporting Framework* is the lack of attention that these areas have received in policy and practice. They are often at the periphery of core business and tend to be insubstantial and under-resourced (Cairnduff & Guthridge 2001; Runcie & Bailie 2000). While Indigenous workforce development is a recognised priority, there is at least one example where lack of capacity in the general Indigenous housing related workforce has been a major impediment to the success of an initiative in this area (enHealth Council 2004).

In relation to information systems, published evaluations of initiatives for effective consultation and communication of housing information to community organisations and community residents have only recently emerged (Jardine-Orr et al. 2004; Wayte et al. 2005). Information has an important role in stimulating action and the lack of well developed processes for consultation and effective communication of housing information to Indigenous community organisations and community residents has led to recent proposals for improving practice in these areas (Jardine-Orr et al. 2004; Wayte et al. 2005). These continuing deficiencies in the development of Indigenous housing programs arise in part

from the lack of a culture of evaluation and research (with some exceptions) relevant to the development, implementation and outcomes of Indigenous housing programs.

The development of system, process and outcome indicators relating to the identified gaps will require the definition of clear goals, strategies, and systems in each area. This will require drawing together local community expectations, knowledge and the best available research and evaluation evidence. Housing standards, and guidelines or strategies for improvement, should also be based on the best available evidence of effective interventions. While there has been substantial and important work in developing standards and strategies or models for intervention, the health-relevant evidence base on which these standards, guidelines and interventions are based is relatively undeveloped both in the international and local environment.

The development of effective strategies to expand the workforce and to provide training in an environmental and housing relevant CQI approach should be a priority. Adaptation of a recent World Health Organization (2005) framework for workforce development for chronic illness care might provide some useful guidance in the development of a competency based approach to workforce development. Following this approach, the key competencies would include community resident centred services; partnering; quality improvement; information and communication technology; and a public health perspective.

The development of organisational structures to support initiatives in Indigenous housing is at a turning point. The effectiveness of the new arrangements in supporting a systematic ecological evidence based approach will be critical to improving housing conditions. With the abolition of the Aboriginal and Torres Strait Islander Commission, the Department of Family and Community Services has taken over Commonwealth responsibility for Indigenous housing (Office of Indigenous Policy Coordination [OIPC] 2004). The new approach by the Commonwealth government is underpinned by concepts of 'shared responsibility' (or 'mutual obligation'), 'partnerships', 'whole-of-government', 'regional focus', 'flexibility' and 'outcomes'. Across Australia multi-agency Indigenous Coordination Centres have been established, which are managed by the Office of Indigenous Policy Coordination. It is intended that these Centres will work with regional networks of representative Indigenous organisations to ensure that local needs and priorities are understood (OIPC 2004). A CQI approach could provide a way for community and other organisations to be effectively engaged in housing initiatives.

Given the policy statements of *Building a Better Future* (HMACSCIH 2001), the achievement of measurable outcomes of improved housing will be a test of the political will and imagination of various levels of government. Modern CQI theory offers a promising framework to inform the development of a model for housing and health improvement. Implementation of key aspects of the CQI approach will require significant development of capacity at the level of local communities and regional offices that support these communities. It is important for the *National Reporting Framework* to be refined to include indicators that will reflect progress in this regard. Perhaps even more importantly, effective CQI approaches for housing and health improvement will require the development of a CQI culture at all levels of Indigenous housing organisation and management, and significant development of capacity to engage with organisations and residents at the community level.

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Environmental Health for the Homeless? Creating Supportive Environments for Health and a Better Quality of Life[1]

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Homelessness and extreme poverty are no longer only associated with developing countries, with strong evidence to suggest worldwide growth in inequalities and poverty might be attributed to globalisation. One indicator of the consequences of globalisation is reflected in the increased visibility of the most extreme poor: the homeless. In Australia, and in many industrialised countries, responsibility for homelessness has tended to fall under community service departments, escaping the agendas of environmental health practitioners as a discrete area for action. This is despite the Australian National Environmental Health Strategy's *(enHealth Council 1999) call for environmental health justice to be integrated into government policies, programs and activities. This paper reports on the findings of a qualitative investigation into the lived experience of homelessness in Darwin, Australia. Using participant observation and interview as principle methods of data collection, the role of trauma in the everyday lives of homeless people is explored. First, homelessness in Australia is defined and the need for this type of study on the lived experience of homelessness explained. An overview of the study and research approach is then provided. Selected key findings are summarised before turning the focus to the role of trauma and the cumulative effects of homelessness with trauma. The paper concludes with suggestions for the integration of 'homelessness' into the delivery of mainstream environmental health services.*

Key words: *Homelessness; Trauma; Environmental Health; Qualitative Research; Darwin*

Homelessness and extreme poverty are no longer only associated with developing countries (Finley & Barton 2003; Leach 1998; Zufferey & Kerr 2004). With industrialised nations reporting a growth in the number of people living below the poverty line, there is strong evidence to suggest worldwide growth in inequalities and this might be attributed to globalisation (Bambrick 2005; Chopra 2005; Kahne 2004). One indicator of the consequences of globalisation is reflected in the increased visibility of the most extreme poor: the homeless (Hasegawa 2005). Finley and Barton (2003) comment, "being without adequate shelter is a situation that thrives in the United States and in other economically wealthy nations" (p. 483). The increased visibility of poverty has, however, generated public debate and homelessness is recognised as an unpalatable condition, without the normal safeguards for health and security experienced by other citizens.

The paper raises practical and contextualised suggestions for environmental health practitioners to contribute to the creation of supportive environments for health for this population.

Environmental Health Practitioners and Homelessness?

The environmental health profession is a dynamic one. *The National Environmental Health Strategy* (enHealth Council 1999) has highlighted the significant contribution made by environmental health to public health over the last century. Environmental health advancements in water quality, sanitation, food safety and housing conditions have led to a massive

improvement in the quality of life and longevity. The *Strategy* and the *Australian Charter for Environmental Health* (enHealth Council 1999) remind us that all Australians are entitled to live in a safe and healthy environment.

The *Strategy* recognises that some populations in Australia are disadvantaged, with an emphasis on Indigenous people. In this paper it is argued that homeless people should be explicitly understood as one such disadvantaged population under the *Strategy*. More specifically, 'homelessness' should be included in the *Strategy's* focus on the integration of environmental health justice through government policies, programs and activities.

The aim of the paper is to share knowledge on the lived experience of homelessness in Northern Australia, and to establish that homelessness is an important area in which the environmental health profession has much to offer. Environmental health practitioners have long recognised the link between health and the built and natural environments. Yet in Australia, and in many industrialised countries, responsibility for homelessness has tended to fall under community service departments, escaping the scope of environmental health practice as a discrete area for action.

By explaining homelessness through the lived experience of trauma, some of the issues that daily life presents for this population, are highlighted. This discussion aims to stimulate thought around the ways in which environmental health practitioners could incorporate the complexities of homelessness into their practice.

Defining homelessness

Homelessness is culturally and socially defined, with meaning attained through a particular community at a given point in time (Tipple & Speak 2005). As such, government policies and/or legislation that distinguish 'homeless' from 'housed' differ from place to place.

In Australia, census collection uses three definitions of homelessness: the primary, secondary and tertiary homeless (Australian Bureau of Statistics 1996, 2001). The primary homeless consists of people without conventional accommodation, such as those sleeping on streets, in parks, or squatting in derelict buildings, cars and railway carriages. The secondary homeless category includes those who move often from one form of temporary shelter to another, such as people in hostels and night shelters, and the tertiary group refers to those in boarding houses on a medium to long term basis.

The definitions used to enumerate homelessness in Australia make distinctions based on shelter type, and do little to explain the socio-cultural variations in meaning. This is illustrated by Keys Young (1998) in a study that examined the meaning of homelessness among Aboriginal Australians. The authors found five distinct types, which recognised that spiritual forms of homelessness, overcrowding, mobile lifestyles and unsafe and unstable homes all can be discussed under the umbrella of homelessness (Keys Young, 1998; see also Memmott et al. 2003).

The Commonwealth government's primary response to homelessness is affected through the *Supported Assistance Accommodation Program (SAAP) Act 1994*. The Act uses a formulation of homelessness that concentrates on eligibility to SAAP services. A person is deemed homeless if: the housing they have access to damages or is likely to damage their health and safety; marginalises a person; places a person in a position that adversely affects the adequacy, safety, security and affordability of housing; or if they have no security of tenure (Australian Institute of Health and Welfare 2003). Several of these circumstances fall into the general working environment of environmental health practitioners.

Although anomalies exist between definitions around enumeration and access to support services, homelessness is increasingly understood to be more than a

housing deficit, and is widely regarded as a multilayered and multidimensional problem.

The research participants in this study fit within the primary category of homelessness. In Darwin, they are often referred to as 'Long Grassers'. The term 'Long Grass' has evolved from its original meaning. It once referred to Aboriginal people inhabiting the grassed areas hugging the Darwin foreshores and living a 'camping' lifestyle. Although this notion remains popular, the Long Grass now has a broader interpretation. Today, both Aboriginal and non-Aboriginal people describe themselves as Long Grassers, and the Long Grass can be any place where people live or a way of living. As such, people who identify as Long Grassers can include those who camp or live rough in multiple locations. Some will be homeless and suffer from extreme poverty, whereas others are not, and have a home to return to. The Long Grassers who participated in this study represented the most extreme manifestation of homelessness and although they might have identified a place or home country of origin, they were at the time, socially, emotionally and physically disconnected from that place.

Methodology

Rationale for the research

The research and the focus of this paper are concerned with learning about the lived experience of homelessness. This work focuses on the perspective of homeless people in order to gain insights into how environments can be created that are supportive of their health and lead to a better life quality. Parker and Fopp (2004) contend that "much of the Australian literature regarding homelessness to date omits the perspective of people who are homeless...particularly in the academic literature, qualitative analysis remains relatively undeveloped" (p.145).

The qualitative inquiry used a mixed methodology. The analytical processes used reflect elements of both action research and grounded theory, in that the findings are 'grounded' in the data. Data are refined through rigorous coding, and themes emerge and contribute to the generation of theory or research propositions (Babchuk 1996; Strauss & Corbin 1998).

The paper reports on selected findings of an investigation undertaken at a Supported Assistance Accommodation Program (SAAP) funded institution, which provided a shelter and free meal service to homeless people in Darwin, the capital of the Northern Territory of Australia. Qualitative data were collected between June 2004 and June 2005. During the first seven months data were collected using participant observation and interviews as principle methods while working as a volunteer in the study setting. Participant observation involves ongoing engagement in the daily activities of a particular group of people and learning from these interactive experiences (Van Manen 1990). Through volunteering alongside homeless people, between three and seven hours each day was spent doing various jobs, such as setting tables, serving food, washing up and cooking. Much of the time was spent talking and gradually strong relationships developed between the researcher and many homeless people, which facilitated learning about their daily activities and life experiences, sometimes referred to as rapport building (Agar 1980). Later this led to the collection of data beyond the institutional setting over a period of five months, for example, when participants invited me to spend time with them, accompany them to appointments, or show me local places of importance. As van Manen (1990) points out, "the best way to enter a person's life world is to participate in it" (p. 69).

Participants were advised daily about the nature of the research by staff. This was reiterated by the researcher through one-to-one introductions. It was made clear that participants had a choice as to whether they engaged with the researcher or not. During fieldwork many participants, often those who had volunteered, became informants in

the research, and up to a dozen of these became key informants. Conversations were usually instigated and directed by participants, with some being enthusiastic about the research and the opportunity to share their experiences. The study received ethics approval from the University of Western Sydney's Human Research Ethics Committee.

During participant observation there was:

- an estimated average of 50 people per day using the lunch service
- approximately 80% of people were male
- rarely non-Indigenous women, and
- Aboriginal people constituted about 35-45% of all people.

At least half of the non-Indigenous people attended the homeless service daily. Indigenous people regularly attended the service, although would miss days and sometimes weeks, particularly if they had travelled away from Darwin.

The context of homelessness in Darwin

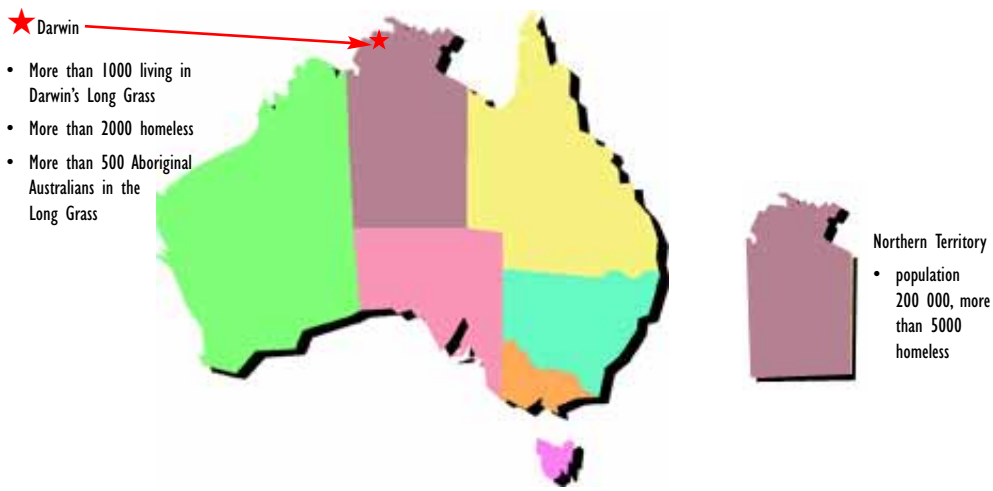
In order to understand the lived experience of homelessness in Darwin we must briefly explore the broader context that influences this way of living. The tropical climate and location of Darwin shape the experience of homelessness. Darwin is isolated from the most populated areas of the country (Figure 1) with a dry season between May and November, and a monsoon (or wet) season from December to May. Temperatures in winter range between 16 and 31°C and in summer between 26 and 34°C with high humidity and rainfall. The wet season increases the risk of mosquito borne diseases, such as Ross River Fever.

Homelessness rates in the Northern Territory remain the highest in Australia with more than 5000 people in a total population of 200,000 (Figure 1). In Darwin, more than 2000 homeless people were counted in a total population of 100,000, with more than 1000 experiencing primary homelessness. Indigenous Australians accounted for half of the primary homeless, that is 500 people (Australian Bureau of Statistics [ABS] 2005).

Participants

The primary homeless in this study experienced economic disadvantage. They were rarely engaged in employment, with several having no source of income at all. Employment was hindered by addictions, poor health, a lack of skills and education, poor literacy, along with discrimination and criminalisation. Most, if not all, welfare

Figure 1: Homelessness rates in Darwin and the Northern Territory



payments were spent on alcohol, drugs, tobacco and gambling.

Aboriginal participants in this study had less control over how their money was spent in contrast with non-Aboriginal participants. One participant explained, 'it is our way', a practice stemming from cultural expectations around sharing with kin. Pension day was often stressful, as family members would extort often all available income, leaving the individual with no money until the next pension day. Some participants used avoidance strategies to retain money, which was often associated with planned travel relating to a funeral. Many non-Indigenous participants also shared financial resources with other homeless people, most evident among those who were in relationships with Indigenous people.

Participants felt that they were regarded as criminals by the mainstream population. There is evidence to support such a perception. One example relates to the delivery of criminal sanctions under Council by-laws that prohibit camping and sleeping in public places. Between January and June 1999, 62 homeless people were jailed for non-payment of fines relating to these by-laws (Goldie 2002; Griffith 1999). Coleman (2001 cited in Goldie 2002) comments: "Public spaces form the immediate physical context in which homeless people pass much, or all, of their time" (p. 278). More than 70% of these people were Aboriginal (Goldie 2002), yet they only constituted 50% of the primary homeless population (ABS 2005). This distinction between private and public space has significant ramifications for daily life for Darwin's Long Grassers. Many research participants reported they had nowhere to go, particularly during the daylight hours, and were forced into a continual cycle of mobility around the local area. Nighttime was spent hiding to avoid receiving a fine and/or being moved on.

Participants in this study believed they were discriminated against, and went to lengths to disguise their homelessness through several mechanisms, such as attending to personal appearance and hiding belongings. Discriminatory treatment, according to Lynch and Stagoll (2002, p. 321) "exacerbates underlying causes of marginalisation and disadvantage" and was detrimental to the health and wellbeing of homeless people.

Community tensions in Darwin over 'Long Grassers' and 'itinerants' was a dominant issue in the most recent election held in 2005. The perception held by a vocal number of the housed community that this was a homogeneous population characterised by laziness, criminal behaviour, alcoholism and the 'Long Grasser' lifestyle, was a strong theme emerging through local media. The community rarely used the words 'poverty' and 'homeless' to describe any part of this population, suggesting they are not conceptualised as impoverished or homeless, with 'itinerant' implying most people were Aboriginal and had a home to return to.

Selection of Key Findings

The inquiry found that the participants:

- had poor physical and mental health, and were affected by violence, alcohol and drug addictions;
- felt tired most of the time;
- had real and imagined barriers to accessing health and other services, including rehabilitation;
- placed great value on the homeless service and used multiple other services;
- met social needs through participating in the service as a guest and/or volunteer;
- developed a 'homeless knowledge', and used this for planning and survival;

- believed mainstream society rejected them, were fearful of them, and treated them like criminals and lesser human beings;
- placed great emphasis on personal appearance and trying not to look homeless;
- experienced daily life within a culture burdened by trauma and poverty;
- developed resilience despite the context of their life world;
- had their homeless identity and homelessness reinforced through participation in homeless services and other agencies; and
- lived life in a state of constant fear, stemming from multiple traumatic events and by homelessness itself.

Trauma and homelessness

The broader context affecting life for homeless people in Darwin has been discussed. The remainder of this paper focuses on the experience of daily life within these contexts as it is played out within a framework of compounded trauma.

In this study, a significant traumatic event had been experienced by almost all participants, and was often the first event to steer someone from being 'homed' to being homeless. Traumatic events reported by participants included:

- the unnatural death of a family member or person significant to the individual (suicide, murder, freak accident or sorcery);
- multiple deaths in a family or among people significant to the individual;
- historical events (including war, conflict, displacement and the systematic forced removal of Aboriginal children from the family by the state);
- physical and sexual abuse (particularly where overcrowding was prevalent);
- unwanted pregnancies or 'wrong side' babies (pregnancies stemming from sexual assault and wrong kinship ties or incest that led to abortion or adoption);
- family breakdown (including adoption, exclusion, forced removal of children from the family by the state, death, physical and sexual abuse); and
- incarceration.

Living with the experience of a major trauma had a profound effect on the life quality and health of many participants. Trauma was generative of other events that were occasionally experienced as traumatic in themselves, referred to as secondary traumas, such as alcohol and drug abuse, prostitution, chronic gambling, disease and homelessness.

It was not uncommon for participants to experience a series of traumatic events that in turn led to, and then sustained, a homeless lifestyle. The experience of trauma played an important function in the decision making processes associated with daily life. Decisions about where to sleep or whether to drink, for example, were often made in the context of avoiding trauma, keeping safe, and as a response to trauma itself.

The National Centre for Post Traumatic Stress Disorder (NCPTSD) identifies 'avoidance' as a common approach to managing or surviving trauma. Multiple behaviours of the homeless people in this Darwin study could be regarded as avoidance strategies, for example, staying away and disconnecting with places, activities and

people which act as reminders, and avoidance of situations which invoke an emotional reaction (Carlson & Ruzek 2006).

In addition to the primary effects of trauma, the NCPTSD has documented associated problems that can emerge as a result of post-traumatic re-experiencing and avoidance strategies. Through this study, a number of behaviours that can be regarded as problems associated with this have been documented. These include:

- depression through loss (such as loved ones) or through isolation (as an avoidance strategy);
- self-harm and harm of others[2];
- self-blame, guilt and shame through an inability to meet responsibilities and through questioning their role in the traumatic event;
- relationship problems due to difficulty in feeling close or trusting others, particularly when the trauma was linked to people (rather than a natural disaster);
- feeling detached or disconnected due to difficulties with feeling or expressing positive feelings and avoidance;
- social isolation and the resultant loss of support creating fear and worry;
- physical health problems due to long-term anxiety, avoidance of medical care, and coping mechanisms such as alcohol and drug use; and
- alcohol and drug misuse as a coping strategy of avoidance of feelings, images, memories common with PTSD (Carlson & Ruzek 2006).

The NCPTSD (2006) argues that some people are more likely to develop PTSD, including those who experience:

> *…greater stressor magnitude and intensity, unpredictability, uncontrollability, sexual victimisation, real or perceived responsibility, and betrayal…those with prior vulnerability actors such as genetics, early age of onset and longer-lasting childhood trauma, lack of functional social support, and concurrent stressful life events…those with a social environment that produces shame, guilt, stigmatisation, or self hatred* (p. 2).

Trauma among Indigenous Australians, it can be argued, has been a cumulative and on-going process that has been generated through colonial and post-colonial policies (Cunneen & Libesman 2000). Zubrick et al. (2005), for example, argue that colonialisation continues to have a profound impact on the physical health of Aboriginals, and also on their wellbeing, stemming from "multiple losses and traumas experienced as a result of separation from land, family and cultural identity" (p. xvi). For Indigenous Australians in this study, trauma-surrounding homelessness is layered over colonial and post-colonial traumas.

For all the participants in this Darwin study, traumatic events led to the loss of house and therefore home and a sense of place or belonging. Other outcomes relating to avoidance included alcoholism, drug addiction and excessive gambling, which often played out in public places. Participants worked to physically and emotionally distance themselves from painful thoughts, hence the over-consumption of alcohol and other drugs. Physical and mental health conditions often deteriorated as a result of trauma, further exacerbated by homelessness. Health services were typically accessed only when the individual reached a point of crisis within the context of a homeless life.

Homelessness for the people in this study had social consequences. Participants believed they were regarded as anti-social, deviant, and a lesser class of people. Many study participants struggled with feelings of rejection, believing they were not understood or different from the people they regarded as important in their lives. This,

coupled with the broader social consequences, caused many participants to feel profoundly lonely. This experience was exacerbated for single homeless people. In an analysis of longitudinal research studies in the UK, Lenmack Consulting Pty Ltd (2005) reported, "At its extreme, single homelessness was not merely an exclusion from housing, but an exclusion from normal human relationships and society itself" (p. 52).

The following vignette details the lived experience of homelessness as it is shaped by trauma for one homeless participant in this study. Elements of this story were common among all study participants.

Polly

Polly[3] was 38 years old. She was an Aboriginal woman, largely illiterate, and unemployed. As a young teenager, she had experimented with alcohol and by the age of 13 she had contracted her first of many STDs. Polly was 17 when she became pregnant and had only ever had sex when she had been drunk. As a slight girl, she was 4-5 months gestation when she learned of the baby. She said:

> *I did not want a baby. All that humbug[4]. I was only 17… I killed my baby myself by hitting my stomach and the baby died. I spent a long time, might be 5 or 6 weeks in the hospital and they cleaned me. The baby did not come out and I got sick with an infection so they scraped my stomach inside.*

By age 18, Polly lived in the Long Grass in Darwin. She recalled her sadness and explained she drank more to feel better. This experience marked the first traumatic event in Polly's life that eventually led to homelessness.

Polly spent the next 20 years in the Long Grass, drinking every day. She lived with her father, brothers and sisters, and members from her own and other communities, linked usually through marriage and through meeting common needs. Staying in a group was a fundamental safety measure against evil spirits, with sorcery underpinning many of her daily activities.

Polly met a non-Indigenous man, 30 years her senior, who supported her in giving up alcohol. She had experienced abdominal discomfort and received advice from a doctor that she would be dead within a year if she kept drinking. This news had a profound effect on her life, marking a further traumatic event and triggering, what would become a perpetual state of fear.

Other traumatic events stemmed from Polly's sobriety. She had gained weight and was regarded as "looking good now" by her mob. Consequently, Polly experienced increased pressure to prostitute herself or share her money. This situation caused her to live and re-live her fear associated with the trauma of dying prematurely. She had come to equate the Long Grass with drinking and imminent death. As such, Polly was insistent she needed her money to pay rent in a little flat she had secured:

> *I need my money for living and for food, I do not want them to spend it on alcohol, I need it to live…if I lose my flat I will end in the Long Grass and I will die…I don't want to die, I want to live.*

In addition to the pressure placed on Polly to share her money, she was also expected to share her flat. Family would visit her flat daily to 'humbug her' after money, cigarettes, food and a place to sleep. Sometimes they wanted a place to drink without being threatened or moved on by the police or council officers. Fearful of losing her flat and dying, she resisted letting her family in, which was often met with animosity. Whether she let her family in or not, it usually caused a disturbance and complaints were made. This was despite the fact that Polly had been active in contacting the police and night patrol to manage a situation that she was unable to. She was eventually evicted from two flats. Although frustrated by her situation, being housed was very lonely. Polly longed to spend time with her family and placed great value on the opportunity to speak in her own language.

Polly had eight siblings. One brother had been incarcerated for killing his wife.

Another brother had been killed when he was hit by a taxi. A sister had been killed by electrocution. Another sister had been killed when she was shot by her husband and one sister had drowned. She had two sisters and a brother who lived in Darwin's Long Grass. Reflecting on her family, Polly said: "We can't drink this moselle [cask white wine]. We drink it, we die".

Polly was deeply affected by the death of her sister who had drowned. The death was caused by evil spirits who took her sister, as Polly would not stay with the man she was promised to, bringing this punishment to their family. She explains:

> *…when I run away they use magic…then she is in Darwin, drinking here and swimming, then she drowned…Everybody talked about it and everybody knows it was the magic…because in Aboriginal way, my sister can be punished for me, because I was running away.*

Sorcery, witchcraft and malevolent others, reinforced by the death of her sister, worked to sustain the fear experienced by Polly in everyday life. Living in the Long Grass exposed Polly to both evil and uncontrollable risks. While the death of her sister had been one of a series of traumatic events, Polly continued to live a traumatised and fearful existence.

Raising awareness among environmental health practitioners of the impact of trauma on homeless people is an important first step to responding in an appropriate manner. Polly's story describes the lived experiences of homelessness as it is affected by multiple traumas and captures some of the barriers for Long Grassers to exit homelessness and enter mainstream housing.

Supportive Environments for Health

While focusing on housing people continues to be important, there remains the need to provide ongoing support for primary homeless people if they are ever to make the transition from a Long Grass way of living to a housed lifestyle. This support must recognise the lived experience of homelessness and the cultural and social challenges that emanate from this way of life.

An alternative way of conceptualising homelessness is to shift from a house-orientated perspective of home, and work to create healthy environments in multiple habitats. Environmental health practitioners can respond more appropriately to the needs of homeless people by better understanding the context in which homelessness is lived in a particular place. Through this, there remain many opportunities for professionals to meet the challenges of the *National Environmental Health Strategy* and make a direct contribution to the creation of healthy environments for this population.

Homelessness, as a discrete area for action, can have implications for environmental health practice and public health through a number of linked strategies:

- an input into local planning and policies that build in homeless people rather than build them out through
    i. the creation of private spaces in public places;
    ii. planning for security which protects public spaces used by homeless people;
    iii. healthy housing, regardless of the form and permanency;
    iv. the identification of suitable locations for alternative and safe shelters for transient populations;
    v. adapting existing facilities used by homeless people, such as public benches;
    vi. the provision of secure and accessible lockers for the safe storage of personal items and important documents;

- challenging existing policies that adversely affect the health of homeless people, or which are discriminatory, or have the potential to criminalise poverty;
- engaging homeless people in policy development through the gradual building of trust;
- the creation of built and natural environments that are conducive to good health for all citizens with
    i. improved access to clean water and sanitation;
    ii. access to facilities for the maintenance of basic hygiene standards;
    iii. access to safe and nutritious food;
    iv. access to facilities that enable the maintenance of personal appearance, such as haircuts and dentistry;
    v. maintenance of sanitation facilities (toilet and shower) with the provision of sanitary goods, such as soap, toilet paper, sharps bins, waste bins, mirrors;
    vi. public kitchens for self catering or all-weather BBQ facilities;
    vii. access to laundry, clothes and blankets;
- strategic delivery of primary health care, health promotion and education programs; and
- community education on issues relating to homeless people.

Conclusion

Being homeless or houseless is conceived by many mainstream community members as an undesirable way to live, but for many people there is no alternative. Placing homelessness on the agenda of environmental health professionals will not be an easy task. It will be fraught with questions, challenges, fears and frustrations. Homelessness, however, must be addressed creatively, locally and across multiple disciplines for improved health and life quality outcomes to be achieved for this population.

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Endnotes

1. An earlier version of this paper was presented to the World Congress in Environmental Health, Dublin, Ireland, June 2006.

2. The NCPTSD (2006) suggests this can stem from frustration over the inability to control PTSD symptoms; a learned response to coping with anger; and limited positive connections with others. While self-harm and aggression towards others was observed among the homeless people in this study, the cause was not always clear, but alcohol

abuse was often one factor. However, it was noted that anger affected job security and relationships with partners, family and friends.

3. Not her real name.

4. Polly used the word 'humbug' in this context to describe feelings of being hassled and bothered while taking on significant responsibility.

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Design Comparison of Experimental Stormwater Detention Systems Treating Concentrated Road Runoff

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Experimental stormwater detention systems were assessed for treatment efficiencies after receiving concentrated runoff primary treated by filtration with different aggregates. Randomly collected gully pot liquor was used instead of road runoff. To test for a 'worst case scenario', the experimental system received higher volumes and pollutant concentrations in comparison to large-scale detention systems under runoff events, which are frequently longer but diluted in comparison. Gravel, sand, Ecosoil, block paving and turf were tested in terms of their influence on water quality. Concentrations of five-day at 20°C ATU biochemical oxygen demand (BOD) in contrast to suspended solids (SS) were frequently reduced to below international secondary wastewater treatment standards. The denitrification process was not completed. This resulted in higher outflow than inflow nitrate-nitrogen concentrations. An analysis of variance indicated that some systems had similar treatment performance variables including BOD and SS. The use of additional aggregates with high adsorption capacities in the primary treatment stage does not make a significant impact on water quality.

Key words: *Environment; Hydrology; Water Resources; Infrastructure Planning; Pollution; Stormwater Detention*

Sustainable Urban Drainage Systems (SUDS)

Until very recently, water quality issues relating to diffuse urban pollution were widely regarded as difficult to control and treat. Urban areas are centres of development but also depend on the environment for resources and waste disposal. Therefore, the urban environment is one of the areas in which strategies of sustainable development need to be put into practice. Over the last few decades rapid urbanisation has occurred all around the world. The fact of increased urbanisation contributes to a number of complex problems, such as the disturbance of stormwater management in developed areas. As a result of inappropriate management of stormwater, flooding of urban areas and deterioration of water quality in watercourses are likely to occur. Discharge of urban stormwater can cause many adverse effects in urban areas, including flooding, sedimentation, nutrient enrichment, toxicity, reduced biodiversity and reduced water uses.

From an environmental health perspective, pollutants are a problem in stormwater. Pollutants removed from runoff in a system like a pond can accumulate in sediments and biota (Jefferies et al. 1999). The settlement of relatively large and heavy particles contributes to sediment accumulation. The particles might contain heavy metals and trace elements originating from street surfaces or parking lots, and nutrients and pesticides might be associated with soil particulates (Pitt 1995). Traditional drainage systems have been shown to be inefficient and expensive

solutions to these problems, so sustainable urban drainage systems were introduced (Nuttall, Boon & Rowell 1998).

Sustainable Urban Drainage Systems (SUDS) are used to improve capacities in existing conventional drainage systems in urban environments. SUDS are a singular or series of management structures and associated processes designed to drain surface water runoff in a sustainable manner. This system is also known as Best Management Practice (Butler & Davies 2000; Construction Industry Research and Information [CIRIA] 2000; SEPA 1999).

New developments proposed for brownfield sites or on the periphery of urban developments might be unable to obtain planning permission if existing local sewers have no spare capacity for stormwater drainage and if the stormwater discharge from the proposed site cannot be controlled. In the absence of suitable watercourses able to accommodate direct stormwater discharges, alternative technologies such as 'at source' stormwater storage and detention systems are required (Butler & Davies 2000).

Maintenance of all public SUDS structures above ground in the United Kingdom is usually the responsibility of the local authority (The Stationery Office 1998). Above ground SUDS structures are defined as swales, ponds, basins and any other ground depression features. In contrast, the maintenance of underground SUDS structures is usually the responsibility of the local water authority. Underground SUDS structures include culverts, infiltration trenches, filter strips and underground detention systems (Butler & Davies 2000; CIRIA 2000; Nuttall, Boon & Rowell 1998). The major function of SUDS is the maintenance of as natural a flow in a watercourse as possible. Urbanisation, with greater impermeable surfaces than occur naturally, lead to more rapid flow fluctuations, with greater high flows and lower low flows (Gardiner 1994). The results are degradation of watercourses, more frequent flooding and lower flows in dry weather. SUDS, either source control or diffused pollution control techniques, should try to return the urbanised system to as close as possible to a natural regime (Jefferies et al. 1999).

Stormwater runoff is usually collected in gully pots that can be viewed as simple physical, chemical and biological reactors. They are particularly effective in retaining suspended solids (Bulc & Slak 2003). Currently, gully pot liquor is extracted once or twice per annum from road drains and transported for disposal at sewage treatment works which might be a long distance away (Butler et al. 1995; Memon & Butler 2002). A more sustainable solution would be to treat the entire road or car park runoff locally in potentially sustainable stormwater detention systems such as underground storage systems and stormwater ponds to reduce transport and treatment costs (Guo 2001). Further, runoff treated with stormwater detention systems can be recycled for irrigation purposes.

Underground stormwater storage and detention systems are sub-surface structures designed to accumulate surface water runoff and to release water when required increasing the flow hydrograph. The structure might contain aggregates with a high void ratio or empty plastic cells and act also as a water recycler or infiltration device (Butler & Parkinson 1997).

An underground stormwater detention system comprises a number of components forming a structure that is designed to reduce stormwater flow. The system captures surface water through infiltration and other methods. The filtered stormwater is stored underground in a tank. The water is often cleaned and filtered before it is infiltrated or discharged to the sewer or watercourse via a discharge control valve. The system benefits include runoff reduction of minor storms, groundwater recharge and pollution reduction. This detention system is predominantly applied in new developments.

Project Purpose

The aim is to advance knowledge and understanding by formulating design guidelines for vertical-flow stormwater detention systems treating road runoff predominantly by extended storage in a cold climate such as the Southeast of Scotland. The objectives are to assess:

1. the function of turf (absent *versus* present) and different aggregates such as Ecosoil as components of a primary treatment filtration stage before the underground detention systems; and

2. the overall passive treatment performance of vertical-flow stormwater detention systems.

Materials and Methods

System design and operation

Five detention systems (Figure 1) were located outdoors at The King's Buildings campus, The University of Edinburgh, Scotland, to assess the system performance during a relatively cold spring and summer (31 March to 19 August 2004). Inflow water, polluted by road runoff, was collected by manual abstraction with a 2L beaker from randomly selected gully pots on the campus and the nearby main roads.

Five stormwater detention systems based on plastic cells (boxes with large holes) were used. Each system had the following dimensions: height = 85 cm, length = 68 cm and width = 41 cm. Two plastic cells on top of each other made up one detention system (Figure 1). The bottom cell (almost 50% full at any time) was used for water storage only. The top cell contained the aggregates. Different packing order arrangements of aggregates and plant roots were used in the systems (Tables 1 and 2) to test for the effects of gravel, sand, Ecosoil, block paving and turf on the water treatment performance.

The filtration system was designed to operate in vertical-flow batch mode. Manual flow control was practised. Gully pot liquor compares well with concentrated road runoff (by a factor of at least 30 depending on gully pot spacing), and was used in the experiment as a worst case scenario liquid replacing road runoff. All detention systems (Tables 1 and 2) were watered approximately twice per week with 10L gully pot liquor as slowly as possible, and drained by gravity afterwards to encourage air penetration through the soils (Cooper et al. 1996; Gervin & Brix 2001). The relative quantity of gully pot liquor used per system was approximately 3.6 (the mean annual rainfall volume to simulate a worst case scenario. The hydraulic residence times were in the order of one hour. Biodegradation was enhanced by encouraging natural ventilation of the aggregates from the top via the natural air, and from the bottom via the air pocket above the storage water and between the aggregates (Figure 1). Considering industrial-scale systems, vertical ventilation pipes should be installed to encourage passive ventilation as well.

Table 1: Systematic and stratified experimental set-up of gravel-filled stormwater detention system content and operation

System	Planted	Additional media type	Natural aeration restricted
1	No	-	No
2	No	Sand	No
3	No	Sand and Ecosoil	No
4	No	Sand, Ecosoil and block paving	Yes (due to block paving)
5	Yes	Sand, Ecosoil and turf	No

Analytical methods

The five-day at 20°C ATU biochemical oxygen demand (BOD) was determined in the inflow and outflow water samples with the OxiTop IS 12-6 system (Wisenschaftlich-Technische Werkstätten [WTW], Weilheim, Germany), which is a manometric measurement device. The

Figure 1: Experimental stormwater detention systems

measurement principle is based on measuring pressure differences estimated by piezoresistive electronic pressure sensors. Nitrification was suppressed by adding 0.05 ml of 5g/L N-Allylthiourea (WTW Chemical Solution No. NTH 600) solution per 50 ml of sample water.

Concerning the analysis of nutrients in the liquid phase, oxidised aqueous nitrogen was determined in all water samples as the sum of nitrate-nitrogen and nitrite-nitrogen. However, nitrite-nitrogen concentrations were significantly low (data not shown). Nitrate was reduced to nitrite by cadmium and determined as an ago dye at 540nm (using a Perstorp Analytical EnviroFlow 3000 flow injection analyser) following diazotisation with sulfanilamide and subsequent coupling with N-1-naphthylethylendiamine dihydrocloride (Allen 1974).

Ammonium nitrate and ortho-phosphate-phosphorus were determined by automated colorimetry in all water samples from reaction with hypochlorite and salicylate ions in solution in the presence of sodium nitrosopentacyanoferrate, and reaction with acidic molybdate to form a phosphomolybdenum blue complex, respectively (Allen 1974). The coloured complexes formed were measured spectrometrically at 655 and 882nm, respectively, using a Bran and Luebbe autoanalyser (Model AAIII).

Table 2: Packing order of the stormwater detention systems

Height (mm)	System 1	System 2	System 3	System 4	System 5
861-930 (top)	Air	Air	Air	Block paving and 6 mm gravel	Air
791-860	Air	Air	Air	(within spaces)	Turf
751-790	Air	Air	Sand and Ecosoil	Sand and Ecosoil	Sand and Ecosoil
711-750	Air	Sand	Sand and Ecosoil	Sand and Ecosoil	Sand and Ecosoil
661-710	6 mm gravel	6 mm gravel	6 mm gravel	6 mm gravel	6 mm gravel
451-660	20 mm gravel	20 mm gravel	20 mm gravel	20 mm gravel	20 mm gravel
437-450	Sand	Sand	Sand	Sand	Sand
431-436	Geotextile	Geotextile	Geotextile	Geotextile	Geotextile
201-430	Air	Air	Air	Air	Air
0-200 (bottom)	Water	Water	Water	Water	Water

A Whatman PHA 230 bench-top pH meter (for control only), a Hanna HI 9142 portable waterproof dissolved oxygen (DO) meter, a HACH 2100N turbidity meter and a Mettler Toledo MPC 227 conductivity, total dissolved solids (TDS) and pH meter were used to determine DO, turbidity, and conductivity, TDS and pH, respectively. An ORP HI 98201 redox potential meter with a platinum tip electrode HI 73201 was used to measure pH. Composite water samples were analysed. All other analytical procedures were performed according to the American standard methods (APHA 1998).

Concerning the analysis of major nutrients in Ecosoil (aggregate supplied by Atlantis Water Management Ltd), 2ml sulphuric acid (strength of 98%, v/v) and 1.5ml hydrogen peroxide (strength of 30%, v/v) were used as an extraction media (Allan 1974). Approximately, 0.1g of each dried sample and the associated digestion media were placed in a tube and heated at 320°C for six hours. Aliquots were taken and digests were made up to 100ml with distilled water.

For analysis of total nitrogen, the following procedure was adopted: Ammonium (present in the digest) reacts with hypochlorite ions generated by alkaline hydrolysis of sodium dichloroisocyanurate. The reaction forms monochloroamine which reacts with salicylate ions in the presence of sodium nitroprusside to form a blue indephenol complex. This complex is measured colorimetrically at 660 nm using a Bran and Luebbe autoanalyser (model AAIII).

For analysis of total phosphorus, the following procedure was used. Orthophosphate (present in the digest) reacts with ammonium molybdate in the presence of sulphuric acid to form a phosphomolybdenum complex. Potassium antimonyl tartrate and ascorbic acid are used to reduce the complex, forming a blue colour, which is proportional to the total phosphorus concentration. Absorption was measured at 660nm using a Bran and Luebbe autoanalyser (model AAIII).

For the analysis of total potassium, the digest was analysed by a flame atomic absorption spectrometer (Unicam 919, Cambridge, UK) at a wavelength of 766.5nm and with a bandpass of 1.5nm. Standards were prepared in 100ml flasks using 2ml concentrated sulphuric acid and 1.5ml hydrogen peroxide (30% v/v) and made up to mark with de-ionised water. Caesium at a concentration of 100mg/L was added to both standards and digests to overcome ionisation.

Metal concentrations were determined in the raw gully pot liquor and the outflow waters from the experimental rig on 16 June 2004. Water samples for metal determinations were stored at -19°C until analysis.

Concerning the analysis of Ecosoil and grass cuttings, composite samples were collected and stored at -10°C prior to analysis. After thawing, approximately 2.5g of each sample was weighed into a 100ml digestion flask to which 21ml of hydrochloric acid (strength of 37%, v/v) and 7ml of nitric acid (strength of 69%, v/v) were added. The mixtures were then heated on a Kjeldahl digestion apparatus (Fisons UK) for at least two hours. After cooling, all solutions were filtered through a Whatman Number 541 hardened ashless filter paper into 100ml volumetric flasks. After rinsing the filter papers, solutions were made up to the mark with deionised water. The method was adapted from the section 'Nitric Acid-Hydrochloric Acid Digestion' (APHA 1998).

An Inductively Coupled Plasma Optical Emission Spectrophotometer (ICP-OES) called TJA IRIS and supplied by ThermoElemental (USA) was used to analyse selected wastewater, Ecosoil and grass cutting samples. The purpose was to economically screen samples to determine various trace element concentrations and potential contaminants. Analytical precision (relative standard deviation) was typically 5-10% for three individual aliquots.

Results and Discussion

Comparison of costs

The overall capital and maintenance costs were calculated for each detention system for the first year of operation. Maintenance included litter removal and grass cutting, and was based on an area of 1000m^2. Material prices were requested for a volume of 100m^3 per aggregate to obtain realistic figures for a scaled-up detention system (industrial operation size). The five system configurations have standardised cost ratios of approximately 1.0:1.1:1.2:1.3:1.6 based on Edinburgh prices in March 2004. However, the actual prices are subject to negotiation (e.g. quantities ordered) and fluctuation on the market.

Inflow water quality

Table 3 summarises the inflow water quality. The standard deviations for all inflow parameters (except for DO, pH and temperature) are high (Table 3) due to the random selection of gully pots and seasonal variations (Butler & Parkinson 1997; Scholz 2004).

The gully pot liquor was less polluted in summer than in spring. For example, BOD, SS and turbidity in summer were 42, 33 and 46% lower, respectively (Table 3). There are various reasons for this including the observation that the higher temperature in summer compared to spring results in a faster biodegradation rate within the gully pot (Table 3). Moreover, the retention time of the gully pot liquor in summer is likely to be longer than in spring due to less frequent rainfall events. A longer retention time correlated positively with a higher biodegradation rate (APHA 1998; Butler & Davies 2000; Scholz 2004).

Comparison of outflow water qualities

The overall filtration performance figures are summarised in Table 4 and should be compared with Table 3.

Reduction efficiencies for BOD and SS (Table 4) are comparable to findings reported elsewhere (Bulc & Slak 2003; Scholz 2004) for highway runoff treatment with constructed wetlands. The reductions of BOD (Table 4) were acceptable for most systems if compared to minimum American and European standards for the secondary treatment of effluent. Biochemical oxygen demand in contrast to SS (Table 4) outflow concentrations did not exceed the US thresholds of 30.0 mg/l (Tchobanoglous, Burton & Stensel 2003). However, some European standards or those of individual

Table 3: Gully pot liquor (inflow to systems): water quality variables (31/03-19/08/04)

Variable	unit	Number of samples	Mean	SD[a]	Mean (spring[b])	Mean (summer[c])
BOD[d]	mg/l	30	37.8	55.30	50.3	29.4
Nitrate-nitrogen[e]	mg/l	34	1.0	1.54	0.5	1.4
Ammonia-nitrogen	mg/l	34	2.1	1.85	2.4	1.9
Ortho-phosphate-phosphorus	mg/l	34	0.2	0.12	0.1	0.2
Suspended solids	mg/l	30	596.5	1430.40	725.6	483.5
Total solids	mg/l	30	442.8	848.58	311.4	518.9
Turbidity	NTU	35	81.3	81.67	108.0	58.7
Dissolved oxygen	mg/l	33	3.2	1.47	2.9	3.3
pH	-	35	6.99	0.286	6.79	7.16
Redox potential	mV	35	178.0	110.62	106.2	238.5
Conductivity	µS	35	224.7	223.25	338.5	128.9
Temperature (air)	°C	34	18.0	3.92	16.2	19.4
Temperature (gully pot)	°C	34	17.4	4.66	14.6	19.7

Notes: a. standard deviation; b. 31/03-21/06/04; c. 22/06/04-19/08/04; d. five-day @ 20¡C N-Allylthiourea biochemical oxygen demand; e. includes nitrite-nitrogen; na = not available.

Table 4: Relative reduction (%) of outflow variables (31/03/04-19/08/04)

Variables	change (%) per wetland systema														
	System 1			System 2			System 3			System 4			System 5		
	Y[b]	SP[c]	SU[d]	Y[b]	SP[c]	SU[d]	Y[b]	SP[c]	SU[d]	Y[b]	SP[c]	SU[d]	Y[b]	SP[c]	SU[d]
BOD[e]	92	95	89	93	95	90	90	90	90	93	93	92	94	96	92
NO3[f]	-1372	-1483	-1338	-1667	-832	-1918	-695	-482	-759	-1020	-564	-1158	-393	-853	-254
NH4[g]	81	74	87	89	86	93	86	78	94	86	76	96	89	82	96
PO4[h]	-74	16	-120	-64	12	-102	-33	12	-55	-56	8	-88	-74	2	-113
SS[i]	78	67	92	80	69	94	79	69	93	80	69	93	78	66	94
Turb[j]	91	92	90	90	91	89	84	81	88	85	81	90	71	83	51

Notes: a., where in=inflow and out=outflow; b. overall mean (31/03/04-19/08/04); c. mean of the spring (31/03/04-21/06/04); d. mean of the summer (22/06/04-19/08/04) e. five-day @ 20°C N-Allylthiourea biochemical oxygen demand (mg/l); f. nitrate-nitrogen (mg/l); g. ammonia-nitrogen (mg/l); h. otho-phosphate-phosphorus (mg/l); i. suspended solids (mg/l); j. turbidity (NTU).

regional agencies (Cooper et al. 1996; Lim et al. 2003; Shutes et al. 2001) are more stringent; e.g. BOD <20 mg/L. The BOD outflow concentration was also lower than the UK standard (Scholz 2004) for secondary treated wastewater of 20 mg/l (Table 4).

A regression analysis has shown that BOD, ammonia-nitrogen, nitrate-nitrogen and ortho-phosphate-phosphorus can be estimated with conductivity and total dissolved solids using a second order polynomial equation. For example, concerning BOD, nitrate-nitrogen and ammonia-nitrogen with conductivity, the corresponding coefficient of determination (r^2) for Filter 4 are 0.60, 0.71 and 0.76, respectively. This would result in the reduction of costs and sampling effort. However, statistical relationships between other variables were not significant.

Further, it has been suggested that mature and viable microbial biomass, in contrast to aggregates with high adsorption capacities (e.g. Ecosoil) and turf, is responsible for the high overall filtration performances (Cooper et al. 1996; Scholz & Martin 1998). However, it is difficult to classify objectively a biological system as mature without having undertaken intensive microbiological work.

Finally, analysis by ICP-OES of selected inflow and outflow samples for a suite of cations showed that all waters generally contained low concentrations of heavy metals. Measured elemental concentrations were either low (barium, calcium, magnesium and manganese), close to the detection limit (iron) and for most heavy metals (including aluminium, copper and cadmium) below the detection limit. Dissolved zinc was the pollutant measured in highest concentration. The mean inflow concentration for zinc was 0.14 mg/L and the corresponding outflow concentrations were 0.07 mg/L (standard deviation: 0.05 mg/L).

Ecosoil and turf

Ecosoil did not contribute to elevated nutrient concentrations due to very low total nitrogen, total phosphorus and total potassium concentrations of 65, 46 and 1367 mg/kg, respectively. A recent soil quality analysis for areas in Glasgow where SUDS were considered for implementation showed total nitrogen, total phosphorus and total potassium concentrations of 1612, 605 and 4562 mg/kg, respectively (Scholz, Morgan & Picher 2005). It follows that Ecosoil does function only as a very weak fertiliser, and that it is therefore unlikely to contribute to eutrophication after the release of the treated stormwater to the nearby watercourse.

Further, Ecosoil contained only trace amounts of heavy metals (except for aluminium): 1036, 24 and 7 mg/kg dry weight of aluminium, zinc and nickel,

respectively. All other metal concentrations were below the detection limit of the instrument. However, even the aluminium concentrations are similar to values reported elsewhere for urban soil (Scholz, Morgan & Picher 2005).

The influence of turf (System 5, Figure 1) on the organic matter content of the outflow was studied. The BOD and SS concentrations within the outflow from the planted system compared to the unplanted gravel and sand systems were similar (Tables 3 and 4). However, BOD in the outflow of System 5 was lower compared to all other systems.

Moreover, grass on top of Filter 6 (Figure 1) was cut when the length was greater than 10 cm for optical reasons and to reduce the overall nutrient load. Total nitrogen, total phosphorus and total potassium concentrations were 3001, 640 and 6909 mg/kg fresh weight. The presence and harvesting of grass seemed to have a positive effect on the overall nitrate-nitrogen outflow concentration that was lower for System 5 when compared to the remaining systems (Tables 3 and 4).

Conclusion

Five-day at 20°C biochemical oxygen demand outflow concentrations were below the UK threshold of 20 mg/L for secondary treated wastewater. The stormwater detention system did show signs of overloading resulting in relatively high suspended solids (SS) and nitrate-nitrogen concentrations, and further treatment would be required. Moreover, denitrification was not completed, and longer retention times are therefore suggested. Nitrate-nitrogen was lower in the outflow of the planted system (turf on the top).

Gully pot liquor (concentrated stormwater runoff) in relative quantities exceeding three times the mean annual rainfall was used for all systems. Therefore, it is likely that the SS concentration would be much lower in the field under real conditions.

An analysis of variance indicated that there was no significant difference between most systems in terms of their treatment performance (e.g. BOD and SS) despite their different set-ups. It follows that all systems, regardless of their pre-treatment, function as covered wastewater stabilisation ponds.

Sampling costs and effort can be reduced using relationships derived from regression analysis between expensive variables that can be substituted by low-cost ones. For example, BOD can be replaced by conductivity for internal control purposes.

Ecosoil did contain relatively low concentrations of nutrients and metals (except for aluminium). It follows that higher investment costs for more complex systems are not justified based on a water quality analysis alone. However, further studies are recommended, they are as follows:

1. The potential hydraulic and structural benefits of additional aggregates such as Ecosoil are required.
2. As all urban areas produce significant pollution, the amount of pollutants arriving at SUDS must be addressed, together with the ability of the system to remove the pollutants from the drainage routes.
3. There needs to be a selection of an appropriate balance between 'hard and soft' solutions for the provision of effective stormwater management leading to more sustainable systems.

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