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## Health risk assessment of kerosene usage in an informal settlement in Durban, South Africa

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### Abstract

In Durban, South Africa studies have shown that more than seven out of ten households in low-income metropolitan areas rely on kerosene for domestic purposes, leading to widespread problems of poor indoor air quality. In light of the known health effects of kerosene usage, this study aimed to quantify the health risk for people living in a densely populated informal settlement known as Cato Crest within the Durban metropolitan area. The pollutants investigated included nitrogen dioxide, benzene and toluene. Nitrogen dioxide is known to affect both respiratory and immune systems, benzene is carcinogenic while toluene has a neurological health end point. All three pollutants are harmful when inhaled. The United States Environmental Protection Agency (US EPA) health risk assessment (HRA) framework was applied. Information on the exposure patterns of residents in Cato Crest were acquired through questionnaires in which data on fuel use, building structure, cooking habits and time-activity patterns were collected. Air quality monitoring of nitrogen dioxide and volatile organic compounds was also conducted in the households. The time-activity pattern survey revealed that the exposure periods of individuals in Cato Crest were far greater than the default exposure periods used by the US EPA. The results of the HRA showed that the residents of Cato Crest may experience significant health risks as a result of kerosene usage in their homes. Exposure to 1-h nitrogen dioxide concentration is not likely to produce adverse health effects, whereas exposure over a 24-h period indicates a potential health risk to sensitive individuals in two of the households when US EPA exposure values are used and in all of the households when locally derived exposure values are used. Benzene poses a health risk to sensitive individuals in 50% of the households when local exposure parameters are used, whereas there is no health risk associated with exposure to toluene.

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### 1. Introduction

Poor indoor air quality is widely recognised as a problem in South Africa (Bank et al., 1996; Jones et al., 1996; Mehlwana and Qase, 1996; White et al., 1996), chiefly as a result of the reliance of many low-income households on fossil fuel combustion for heating, cooking and lighting. Notwithstanding the increasing

electrification of both rural and urban areas, many poor households continue to use fossil fuels because of the large capital cost of electrical appliances and perceived differences in running costs (Jones et al., 1996). According to Statistics South Africa (SSA, 2000), there are approximately 5 million households in South Africa currently using fossil fuels for domestic purposes. Of these, a fairly high proportion rely on kerosene, with 21% using it for cooking, 14% for heating and 13% for lighting. In Durban, in particular, studies have shown that these percentages are even higher. For example,

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Jones et al. (1996) reported that more than 70% of households sampled in low-income metropolitan areas of Durban relied on kerosene.

Pollutants emitted during kerosene combustion include carbon monoxide, carbon dioxide, sulphur dioxide, nitrogen dioxide (NO<sub>2</sub>), particulate matter, formaldehyde and various hydrocarbons or volatile organic compounds (VOCs) (Leaderer, 1982; Traynor and Allan, 1983). As a consequence there are many potential health effects associated with kerosene usage. Some of the compounds are mutagenic (Mumford et al., 1992), while others are known to be carcinogenic (Zhang and Smith, 1999). Sharma et al. (1998) showed that 33% of kerosene households had acute lower respiratory infection, with pneumonia and bronchiolitis being the most common ailments. Behera et al. (1994) also reported that ventilatory function was slightly impaired and blood carboxyhaemoglobin levels were significantly raised (Behera et al., 1991). Children from kerosene households were also shown to experience more respiratory symptoms than those in households using other fuels (Behera et al., 1998).

NO<sub>2</sub> is known to affect both the respiratory and immune systems (WHO, 1997; California EPA, 1999). Acute exposure to NO<sub>2</sub> is associated with respiratory irritation and can lead to long term changes such as pulmonary oedema, pneumonitis, bronchitis and bronchiolitis obliterans (California EPA, 1999). Clinical effects of NO<sub>2</sub> exposure include a shallow respiratory rate, rapid heart rate, wheezing and cyanosis. Shortness of breath and coughing lasting from 2 to 3 weeks may also occur (TOXNET, 2000). Sensitive individuals, including children, asthmatics and individuals with chronic obstructive pulmonary disease, are particularly affected (California EPA, 1997). Sandstrom et al. (1992) have shown that NO<sub>2</sub> can lead to an alteration in the immune function, causing more rapid onset of diseases such as HIV/AIDS and increased susceptibility to viral and bacterial infection (Becker and Soukop, 1999; Goings et al., 1989; Rosok et al., 1997; WHO, 1997).

Both benzene and toluene are VOCs with serious health effects. Benzene can enter the body through ingestion of contaminated food or water and through the skin, but the most common pathway is through inhalation (ATSDR, 1997). Acute inhalation causes neurological effects such as drowsiness, dizziness, rapid heart beat, tremors, headaches, confusion and sometimes unconsciousness (ATSDR, 2000a). Chronic exposure can lead to various types of anemia as well as leukemia. In addition to being carcinogenic, benzene is also known to be mutagenic (ATSDR, 2000a; CCOHS, 2000; RAIS, 1998; US EPA, 2000).

Toluene also enters the body primarily through inhalation, although it can be absorbed through the gastro-intestinal tract. It accumulates in fatty tissue and can be an internal source of exposure once the initial

external exposure has ended (TOXNET, 2000). Acute exposure to toluene by inhalation causes slight drowsiness and headaches at low concentrations (50 ppm or 13.3 mg m<sup>-3</sup>), irritation of the nose, throat and respiratory tract between 50 and 100 ppm (13.3–26.6 mg m<sup>-3</sup>) and fatigue and dizziness above 100 ppm (26.6 mg m<sup>-3</sup>). Above 200 ppm (53.2 mg m<sup>-3</sup>), depression of the central nervous system (CNS) becomes more pronounced, with symptoms of numbness and nausea being experienced. Mental confusion and lack of co-ordination occur at 500 ppm (133 mg m<sup>-3</sup>) and between 500 and 1000 ppm (133–266 mg m<sup>-3</sup>) an individual could become unconscious and die. Chronic exposure to toluene is associated with severe CNS damage and impaired liver and kidney function (CCOHS, 2000; RAIS, 1998). Some studies have shown toluene to be carcinogenic but according to CCOHS (2000), insufficient evidence is available for it to be classified as a carcinogen.

In view of the known health effects of kerosene usage and its widespread use in informal settlements in Durban, this study aimed to quantify the health risk for people living in a densely populated informal settlement known as Cato Crest, situated approximately 7 km west of the central business district of Durban. The settlement consists of approximately 4500 households and an estimated 18,000 to 23,000 people (Gielink, 2001). The pollutants targeted for investigation in this study include nitrogen dioxide, benzene and toluene.

The study is based on the premise that the default exposure values provided by the United States Environmental Protection Agency (US EPA) health risk assessment (HRA) framework are not suitable for local conditions. The behaviour patterns of South African individuals (particularly those living in informal settlements) are expected to differ somewhat from the North American behaviour patterns on which the US EPA HRAs are based. An individual's behaviour pattern can substantially increase the risk associated with exposure to different hazards. Increased exposure ultimately leads to increased health risks.

## 2. Health risk assessment framework

In order to assess the risk of exposure to kerosene and specifically NO<sub>2</sub>, benzene and toluene, the HRA framework, first introduced by the National Academy of Sciences (NAS) in 1983, and subsequently modified by the National Research Council (NRC) (1994) and now widely used throughout the world was used. The approach comprises four steps, which are outlined below.

*Hazard identification* is the process of recognising chemicals in the environment that, after exposure, might be responsible for adverse human health effects.

*Exposure assessment* identifies the population exposed to the hazard, the characteristics and behaviour of that population and the magnitude, extent and duration of exposure to the hazard. Exposure can occur via many direct and indirect pathways such as inhalation, ingestion and dermal contact.

The *dose–response assessment* attempts to quantify the relationship between a particular dose and the potential adverse effect that can be caused by that dose.

The final step of *risk characterisation* combines the information from the three previous steps to provide an indication of the nature and expected frequency of adverse health effects in exposed populations (American Chemical Society, 1998).

In determining the exposure (non-carcinogenic pollutants), the following chronic exposure rate equation for the inhalation exposure pathway is used:

$$\text{ADD} = \frac{C \times \text{IR} \times \text{ED}}{\text{BW} \times \text{AT}},$$

where ADD is the average daily dose of the chemical ( $\mu\text{g kg}^{-1} \text{day}^{-1}$ ),  $C$  the concentration of the chemical in the atmosphere ( $\mu\text{g m}^{-3}$ ), IR the inhalation rate in  $\text{m}^3 \text{day}^{-1}$ , ED the exposure duration (days), BW the average body weight of receptor over the exposure period (kg) and AT is the averaging time, the period over which exposure is averaged (days) (US EPA, 1996).

The ADD is a measure of intake rather than dose. It is unknown how much of the inhaled toxicant is actually absorbed. Exposure duration (ED) is defined as the length of time that receptors are exposed to the contaminants. It is calculated from the exposure event or time, exposure frequency, and duration of exposure using the following equation:

$$\text{ED} = \text{ET} \times \text{EF} \times \text{DE},$$

where ED is the exposure duration (days), ET the exposure time or event ( $\text{h day}^{-1}$ ), EF the exposure frequency ( $\text{days year}^{-1}$ ) and DE the duration of exposure (year) (US EPA, 1998).

The acute exposure rate equation for non-carcinogenic pollutants is given as follows (Louvar and Louvar, 1998):

$$\text{AHD} = \frac{C \times \text{IR}}{\text{BW}},$$

where AHD is the average hourly dose for inhalation ( $\mu\text{g kg}^{-1} \text{h}^{-1}$ ),  $C$  the concentration of the chemical ( $\mu\text{g m}^{-3}$ ), IR the inhalation rate ( $\text{m}^3 \text{h}^{-1}$ ) and BW the body weight (kg).

The exposure duration and averaging time are omitted from the equation as they are both 1 h for acute exposure.

In the risk characterisation step the non-carcinogenic health effects are expressed as a dimensionless ratio called a hazard quotient (HQ), which indicates the presence or absence of adverse health effects due to

exposure. The comparison between the dose, or more precisely the intake value, and the dose–response value assumes that there is a level of exposure below which it is unlikely that even sensitive individuals will experience adverse health effects. If the exposure exceeds this threshold (dose–response value), there may be concern for potential non-cancer health effects being experienced by sensitive individuals. As a rule, the higher the HQ above 1, the greater the potential for adverse health effects. The relationship between intake and dose–response value is not always linear (US EPA, 1989). A HQ that is  $< 1$  therefore indicates a negligible risk, even to a sensitive individual, while a HQ  $> 1$  indicates that there may be some risk to sensitive individuals as a result of exposure. There is currently no literature that provides an estimate of just how much risk an HQ of 4, for example, presents.

The HQ for chronic risk is estimated from:

$$\text{HQ} = \frac{\text{ADD}}{\text{DRV}},$$

where DRV is a dose response value. Examples include the Reference Concentration (RfC) which can be defined as “an estimate (with uncertainty) of the concentration that is likely to be without appreciable risk of deleterious effects to the exposed population after continuous lifetime exposure” (NRC, 1994). The RfC is based on the assumption that toxic effects will not occur until a threshold dose is exceeded (NRC, 1994). Several agencies have determined their own reference concentrations such as the ATSDR (Agency for Toxic Substances and Disease Registry) minimal risk level (MRL) and the California EPA reference exposure level (REL). The HQ for acute risk is calculated in the same manner except that AHD is substituted for ADD.

Whilst the HRA framework is widely used, there are a number of limitations which have been identified. Amongst these is the criticism that it is not able to evaluate exposure to pollutant mixtures nor take account of synergistic effects (NRC, 1994). The default values are often criticised as being too conservative and not representative of all situations, underlining the importance of conducting local exposure surveys.

### 3. Data and methodology

The application of the HRA framework required data on the exposure patterns of the people living in Cato Crest and a measure of the concentrations of the selected pollutants in the households.

Data on time-activity patterns were collected from 69 households by means of a questionnaire consisting of 48 closed-ended questions. Questions related to personal details, fuel use, building structure, cooking habits and time-activity patterns. It is recognised that the sample

size represents a very small percentage ( $\sim 1.5\%$ ) of the total number of households in Cato Crest, however, financial constraints dictated the size and moreover, this study was less concerned with statistical significance than with obtaining a preliminary understanding of time-activity patterns in the area and whether and how they differed from those of the US EPA. A multistage sampling method was used, in which the initial population was divided according to the eight recognised areas of Cato Crest. Within each area, transects were marked on aerial photographs along pathways that provided vehicular access at either end of a transect. The transects were also selected to ensure an even allocation of houses within areas. Thereafter, sampling along each transect was random, although certain difficulties were encountered. The haphazard and unplanned nature of the informal settlement made strict adherence to the sampling procedure difficult. In such cases the nearest dwelling to that designated on the aerial photograph was selected.

Women were targeted in the questionnaire as they are generally the only household members involved in cooking and other domestic activities. The questionnaires were administered by a group of 10 trained students. All interviewers were fluent in isiZulu and English and conducted the interview in the language in which the respondent was most comfortable. The questionnaires were completed by the interviewers in English.

For the air quality study, 14 households for which the questionnaires appeared to be most complete were selected. Within each dwelling air quality monitoring was performed for  $\text{NO}_2$  and VOCs. The former was measured using ChromAir<sup>®</sup> Direct Read Passive Monitor badges and total VOCs were measured using Traceair<sup>®</sup> Organic Vapour Monitor (OVM-1) badges (K&M Environmental, 1997). The badges are simple and economical diffusive samplers. They eliminate the need for expensive air sampling pumps and the maintenance associated with them, while still producing reliable and accurate results.

The  $\text{NO}_2$  badge is a colourimetric direct-read monitor, which relies on the principle of diffusion. The badge is constructed from six cells attached on one side to a flat indicator layer and on the other side to a series of different diffusive resistances. The  $\text{NO}_2$  gas diffuses to the cells through the different diffusive resistances, at the same time reacting with the indicator layer to produce a colour change from yellow to beige and then brown. The colour produced is a direct measure of the exposure dose. Colour comparison is achieved by observing the formation of the beige threshold colour on the individual cell and reading the corresponding exposure dose. The  $\text{NO}_2$  badges are able to detect a range of  $0.5\text{--}13\text{ ppm h}^{-1}$  ( $930\text{--}24,250\text{ }\mu\text{g m}^{-3}\text{ h}^{-1}$ ), with a minimum detectable concentration in 8-h of  $0.06\text{ ppm}$

( $110\text{ }\mu\text{g m}^{-3}$ ). They are able to function effectively in a relative humidity range of 15–90% and a temperature range of 10–40°C. The ability of the badge to measure  $\text{NO}_2$  is affected by the presence of ozone. The minimum sampling time that the badges can be used is 15 min and the maximum is 2 days (K&M Environmental, 1997).

The OVM-1 badges are used for determining concentrations of a variety of organic compounds. The badge contains a 300 mg coconut based charcoal strip to which the compounds adhere. They can operate in a temperature range of 10–50°C and in a relative humidity range of 10–80%.

The air quality monitoring took place over a 9-day period from 5 to 15 September 2000. In each household, the badges were hung from the ceiling near to where the cooking was done but not directly over the stove. The  $\text{NO}_2$  badges were replaced each day, while the VOC badges were left for the full period of 9 days. No results were obtained for household four (4). The  $\text{NO}_2$  badges were manually read and the VOC badges were analysed at CSIR (Council for Scientific and Industrial Research) by gas chromatography using flame ionization detection. Although a full analysis was undertaken, two compounds, namely benzene and toluene, were selected for detailed discussion here.

Air quality monitoring over a 9-day period was considered adequate since the assessment considered acute rather than chronic exposure scenarios. The results are considered to be fairly conservative. Higher values could be expected in winter months when climatic conditions predispose the area to pollution accumulation.

#### 4. Results

The results of the time-activity patterns revealed that kerosene is widely used in Cato Crest and thus has the potential to cause adverse health effects through the inhalation of combustion products. An estimated 87% of the households relied on kerosene for cooking, while only a relatively small percentage (<30%) used it for heating and lighting.

Of the 69 interviews conducted, 66 interviews were with female respondents. The average age of the interviewees was 34 years and the average weight was 68 kg. Only 10 respondents were employed full time, with 39 unemployed respondents. Two respondents were students. Questions related to the type of fuels used for cooking, heating and lighting. The responses indicated that paraffin was used predominantly for cooking (60 responses) and heating (22 responses). Twenty-six respondents reported using blankets or 'nothing' for heating. Candles were mainly used for lighting (39 responses).

In response to questions on the average time taken to cook meals, 45 respondents reported that it took between 1- and 2-h. Fifty respondents reported that there were people in their household that slept in the same room as the cooking was done in. Forty of these households had between 1 and 3 people sleeping in the 'kitchen'. Table 1 shows the average number of hours spent conducting several different daily activities in summer and winter months.

The HRA was performed for three scenarios, viz. for an adult (30 years), a child (10 years) and an infant (<1 year) for averaging periods of 1 (only for NO<sub>2</sub>) and 24 h. In the case of the latter, the HRA was first performed using US EPA default exposure values (7-h day<sup>-1</sup> for an adult and 2-h day<sup>-1</sup> for a child and an infant) and secondly, using local exposure values estimated from the survey of time-activity patterns in Cato Crest. These were estimated at 14-h day<sup>-1</sup> for an adult and an infant, since the infant is generally carried on the mother's back when she goes outside to do other chores or socialise, and 12-h day<sup>-1</sup> for a child, who is assumed to play outdoors for a portion of the afternoon but would still eat and sleep in the same room as other members of the family. The large differences in exposure values between the US EPA and the locally derived values arose because most of the houses in Cato Crest consist of a single room in which all cooking and sleeping takes place. The exposure period is taken as the total time spent indoors where *exposure* to paraffin fumes from cooking would take place, although the cooking process itself is not carried out for such long periods. In the case of other variables that constitute inputs to the model, for example weight, US EPA default values were used, as no significant differences between them and the local values could be found.

Acute exposure to NO<sub>2</sub> was estimated by comparing the AHD with a REL of 470 µg m<sup>-3</sup> for a 1-h average (California EPA, 1999). The uncertainty factor for the 1-h REL is 1 indicating a very low degree of uncertainty. The REL is based on a respiratory health endpoint, where the sensitive individual is an asthmatic. Measured NO<sub>2</sub> in the households ranged from 51.8 to 103.9 µg m<sup>-3</sup>, hence the REL was not exceeded at any of the households. HQs were all below 0.007, implying no likelihood of adverse health effects occurring at this level of exposure.

Longer term exposure over a 24-h period was compared with a 24-h REL of 100 µg m<sup>-3</sup> (New Zealand Ministry for the Environment, 1994). The uncertainty factor for the 24-h REL is not known, however, it is known that the health endpoint on which the guideline is based is a respiratory endpoint where the sensitive individual is asthmatic (New Zealand Ministry for the Environment, 1994). Measured values (cumulative) over the exposure period ranged between 1243.8 and 2493.8 µg m<sup>-3</sup>. Fig. 1 shows the HQ values at individual households for the 3 scenarios. Based on US EPA exposure values, only two households had HQs > 1 for the adult scenario. It was noted that one of these households was a one-roomed dwelling occupied by seven people and where all cooking was done indoors. The second dwelling had three rooms but no windows in any of the rooms, thus poor ventilation was likely to account for the relatively higher measured NO<sub>2</sub> values. Based on local exposure patterns, all HQs were above 1 for all three scenarios. The minimum HQ value was 1.2 and the maximum 5.6, indicating that all households faced a potential risk of adverse health effects from NO<sub>2</sub> exposure over a 24-h period. Risks were highest for infants and lowest for adults.

Cumulative benzene concentrations over a 24-h period ranged between 31 and 1644 µg m<sup>-3</sup>. These values were compared with the 24-h MRL for benzene of 161.3 µg m<sup>-3</sup> (ATSDR, 2000b). The uncertainty factor for this MRL is 300, indicating a medium degree of uncertainty. It is based on an immunological health endpoint, where the sensitive individual would be someone who has a blood disorder/disease (ATSDR, 2000b). Based on US EPA exposure values, all estimated HQs were below 0.4, indicating no likelihood of adverse health effects. However, based on local exposure values, 7 of the households indicated a risk of adverse health effects for infants and one for a child as well. Sensitive adults are also likely to be affected negatively.

Exposure to toluene over a 24-h period was estimated to pose no potential risk based on both US EPA and local exposure values as all HQs were below 1. Cumulative measured values ranged between 514 and 3055 µg m<sup>-3</sup> and were compared with a 24-h MRL of 1064 µg m<sup>-3</sup> (ATSDR, 2000b) based on a 1–14 day exposure period. The uncertainty factor for this MRL is 10 indicating a high degree of confidence in the guideline

Table 1  
Average hours spent per daily activity in summer and winter months

	Indoors, cooking	Indoors, other	Outdoors, cooking	Outdoors, other	Nearby road	Travel	Indoors, at work	Outdoors, at work	Sleeping
Winter	2	2.6	2	2	1	1.5	8.2	2.8	10
Summer	2	2.4	2	4.3	1	1.5	8.3	2.4	9

Note that the hours do not add up to 24-h day<sup>-1</sup> as these are average hours per activity.

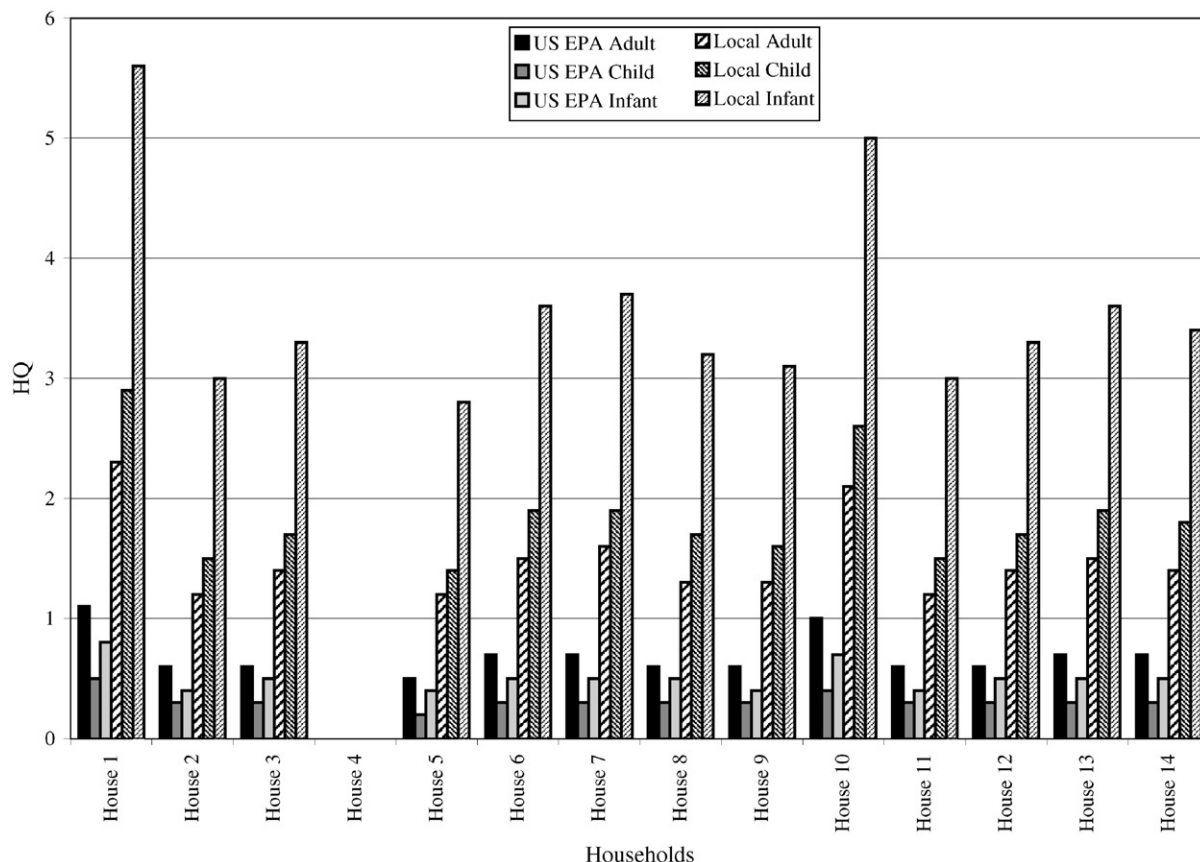


Fig. 1. HQs for 24-h  $\text{NO}_2$  exposure at individual households (1–14) for an adult (30 years), child (10 years) and an infant (<1 year). The solid blocks are based on US EPA exposure values and the hatched blocks on locally derived exposure values.

value. The MRL is based on a neurological health end point, where the sensitive individual would be someone who already has a neurological disease or disorder or someone with a liver disease (ATSDR, 2000b; TOXNET, 2000).

## 5. Conclusion

The results of the HRA have shown that residents in the informal settlement of Cato Crest experience significant health risks as a result of kerosene usage in their homes. Exposure to 1-h  $\text{NO}_2$  concentration is not likely to produce adverse health effects, whereas exposure over a 24-h period indicates a potential health risk in two of the households when US EPA exposure values are used and in all of the households when locally derived exposure values are used. Benzene poses a health risk in 50% of the households when local exposure parameters are used, whereas there is no health risk associated with exposure to toluene.

The time-activity pattern survey revealed that the exposure periods of individuals in Cato Crest were far

greater than the default exposure periods used by the US EPA. The adult exposure period was twice as great (14-h compared with 7-h) and the exposure period of a child increased from 2- to 12-h and that of an infant from 2- to 14-h. These differences are largely due to cultural differences between individuals in North America (the population on which US EPA values are based) and individuals living in informal settlements, particularly in South Africa. The residents of Cato Crest tend to live in smaller dwellings, often comprising only one room, which serves as a multi-function room in which most people slept and in which cooking was undertaken. In addition, ventilation was often very poor, with many dwellings having no windows. The greater exposure of the residents results in a greater potential for adverse health effects to occur. It is also important to bear in mind that many people living in informal settlements have poor nutrition and are more likely to have diseases that suppress their immune system, making them more vulnerable to the risk of health effects due to exposure to environmental pollutants.

This study has underlined the importance of conducting research into time-activity patterns and not relying

on default values. It is recognised that these surveys are costly and time consuming but this small study has demonstrated that the US EPA default values are not an accurate reflection of exposure patterns in informal settlements in South Africa. The US EPA default values are indeed culturally specific and should be treated with caution when applying them to populations other than North Americans. Although the time-activity pattern survey cannot be considered as a valid statistical sample, the consistency of the results provides some measure of confidence in the results. Furthermore, the data provided here may be more useful for studies in other informal communities rather than relying on US EPA default values. Future studies should try to improve on the accuracy of the time-activity pattern data, perhaps by increasing the sample size and by allowing people to keep diaries.

One of the limitations of this study was that no information on other possible sources of exposure to pollutants, such as occupational exposure was recorded. Further, the role of disease, nutritional status and other exogenous factors in causing potential health effects have not been considered.

The air quality study was conducted over a limited period and as such no indication of seasonal fluctuations in pollutants could be obtained, precluding the possibility of estimating long term health risks. Future studies should be conducted over a longer period that would allow an estimate of the risk of cancer effects and should include the assessment of the risk posed by exposure to other pollutants.

It has been shown that the use of kerosene poses a potential health risk to individuals in the informal settlement in Cato Crest and by implication in other informal settlements in South Africa. Interventions to improve the quality of life of residents include switching to a cleaner fuel, a change that has not met with much success in informal settlements. An improvement to the ventilation of the dwelling could be effected at little or no cost and may lead to a substantial improvement in air quality.

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### References

American Chemical Society, 1998. Understanding Risk Analysis: A Short Guide for Health, Safety and Environmental

- Policy Making, USA, [http://www.rff.org/misc\\_docs/risk\\_book.htm](http://www.rff.org/misc_docs/risk_book.htm)
- ATSDR, 1997. Toxicological profiles, CD-Rom issue 97-2. Agency for Toxic Substances and Disease Registry, USA.
- ATSDR, 2000a. ToxFAQs. Agency for Toxic Substances and Disease Registry, USA, <http://www.atsdr.cdc.gov>
- ATSDR, 2000b. Minimal risk levels (MRLs) for hazardous substances. Agency for Toxic Substances and Disease Registry, USA, <http://www.atsdr.cdc.gov/mrls.html>
- Bank, L., Mlomo, B., Lujabe, P., 1996. Social determinants of energy use in low-income metropolitan households in East London. Department of Minerals and Energy Report Number EO9421.
- Becker, S., Soukop, J.M., 1999. Effect of nitrogen dioxide on respiratory viral infection in airway epithelial cells. US EPA Environmental Research Section A, 81, 159–166.
- Behera, D., Dash, S., Yadav, S.P., 1991. Carboxyhaemoglobin in women exposed to different cooking fuels. Thorax 46 (9), 688.
- Behera, D., Jindal, S.K., Malhotra, H.S., 1994. Ventilatory function in nonsmoking rural Indian women using different cooking fuels. Respiration 61, 89–92.
- Behera, D., Sood, P., Singhi, S., 1998. Respiratory symptoms in Indian children exposed to different cooking fuels. Journal of the Association of Physicians of India 46 (2), 182–184.
- California EPA, 1997. Determination of noncancer chronic reference exposure levels, USA, [http://www.oehha.ca.gov/air/chronic\\_rels/](http://www.oehha.ca.gov/air/chronic_rels/)
- California EPA, 1999. Determination of acute reference exposure levels for airborne toxicants, USA <http://www.oehha.org/air/pdf/acuterel.pdf>
- CCOHS, 2000. CHEMInfo database. Canadian Centre for Occupational Health and Safety, <http://ccinfoweb.ccohs.ca/cheminfo/search.html>
- Gielink, S., 2001. Cato Manor development association communications manager. Personal Communication.
- Goings, S., Kulle, T.J., Bascom, R., Sauder, L.R., Green, D.J., Hebel, R., Clements, M.L., 1989. Effect of nitrogen dioxide exposure on susceptibility to influenza A virus infection in healthy adults. American Review of Respiratory Disease 139, 1075–1081.
- Jones, S., Aitken, R., Luckin, L., 1996. Power, poverty and posterity: the social determinants of energy use in low-income metropolitan households in Durban. Department of Minerals and Energy Report Number EO9422.
- K&M Environmental, 1997. Operating instructions for nitrogen dioxide monitor and OVM-1 Badge, USA.
- Leaderer, B., 1982. Air pollutant emissions from kerosene space heaters. Science 218, 1113–1115.
- Mehlwana, M., Qase, N., 1996. Social determinants of energy use in low-income metropolitan households in the Western Cape. Department of Minerals and Energy Report Number EO9420.
- Mumford, J.L., Lewtas, J., Williams, K., Tucker, W.G., 1992. Mutagenicity of organic emissions from unvented kerosene heaters in a chamber study. Journal of Toxicology and Environmental Health 36, 151–159.
- New Zealand Ministry for the Environment, 1994. Ambient Air Quality Guidelines. Ministry for the Environment, New Zealand.

- NRC (National Research Council), 1994. Science and Judgment in Risk Assessment. National Academy Press, USA.
- RAIS, 1998. Toxicity profiles. Risk Assessment Information System, <http://risk.lsd.ornl.gov>
- Rosok, B., Voltersvik, P., Larsson, B., Albert, J., Brinchmann, J.E., Asjo, B., 1997. CD8+ T cells from HIV Type 1-seronegative individuals suppress virus replication in acutely infected cells. *AIDS Research and Human Retroviruses* 13 (1), 79–85.
- Sandstrom, T., Helleday, R., Bjermer, L., Stjernberg, N., 1992. Effects of repeated exposure to 4ppm nitrogen dioxide on bronchoalveolar lymphocyte subsets and macrophages in healthy men. *European Respiratory Journal* 5, 1092–1096.
- Sharma, S., Sethi, G.R., Rohtagi, A., Chaudhary, A., Shankar, R., Bapna, J.S., Joshi, V., Sapir, D.G., 1998. Indoor air quality and acute lower respiratory infection in Indian slums. *Environmental Health Perspectives* 106 (5), 291–297.
- SSA, 2000. Stats in Brief. Statistics South Africa, Pretoria.
- TOXNET, 2000. Hazardous Substances Data Bank. National Library of Medicine, Internet Edition, <http://toxnet.nlm.nih.gov>
- Traynor, G., Allan, J., 1983. Pollutant emissions from portable kerosene-fired space heaters. *Environmental Science and Technology* 17, 369–371.
- US EPA, 1989. Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual (Part A), <http://www.epa.gov/superfund/programs/risk/ragsa/>
- US EPA, 1996. Exposure Factors Handbook: draft update to EPA/600/8-89/043, May 1989.
- US EPA, 1998. Human health risk assessment protocol for hazardous waste combustion facilities, <http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm>
- US EPA, 2000. Benzene, integrated risk information system, <http://www.epa.gov/iris/subst/0276.htm>
- White, C., Crankshaw, O., Mafokoane, T., Meintjies, H., 1996. Social determinants of energy use in low-income metropolitan households in Soweto. Department of Minerals and Energy Report Number EO9423.
- WHO, 1997. World Health Organisation's Environmental Health Criteria 188. Nitrogen Oxides, Second Edition.
- Zhang, J., Smith, K.R., 1999. Emissions of carbonyl compounds from various cook-stoves in China. *Environmental Science and Technology* 33 (14), 2311–2320.