Quantification of the human health risks associated with kerosene use in the informal settlement of Cato Manor, Durban

by

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TABLE OF CONTENTS

ABSTRACT

The main objective of this study was to investigate the application of the United States Environmental Protection Agency (US EPA) human health risk assessment framework for quantifying the adverse human health effects of exposure to inhaled kerosene pollutants in the South African context. The study was based in the informal settlement of Cato Crest in Cato Manor, Durban. This dissertation includes a theoretical review of the environment/health relationship, the US EPA health risk assessment approach, it's history, and the health effects of kerosene combustion products. Chapter three outlines the methodology for the study, detailing how time-activity pattern data and air quality results were collected from the community of Cato Crest. Chapter four presents the results of the health risk assessments conducted for nitrogen dioxide, benzene and toluene exposure – using both local and US EPA exposure values in the health risk assessments. A critical evaluation of the US EPA human health risk assessment framework in the South African context is provided in chapter five.

The results of the study revealed that a 1-hour exposure to the nitrogen dioxide concentrations measured in Cato Crest would not present any adverse health effects. A 24-hour exposure to NO_2 using US EPA default exposure values provided a slight possibility of adverse health effects being experienced in sensitive individuals in some houses. 24-Hour exposure to NO_2 using local exposure values could result in both sensitive individuals and even some healthy individuals experiencing adverse health effects in all houses. Potential adverse health effects include coughing, wheezing, chest tightness, broncho-constriction and increased airway resistance. Sensitive individuals include those with asthma or other respiratory diseases. Exposure to 24-hour benzene concentrations (using US EPA default exposure values) is not likely to result in individuals experiencing adverse health effects. Exposure to the same benzene concentrations at local exposure times will cause potential adverse health effects in sensitive individuals. Sensitive individuals are those with respiratory ailments and blood diseases or disorders. Exposure to monitored toluene concentrations over a 24-hour period (using both US EPA default exposure values and local exposure values) is unlikely to result in adverse health effects being experienced by any individuals. The US EPA human health risk assessment framework is seen as applicable to South Africa where developed areas are concerned (as these areas are quite similar to North American populations). In areas of South Africa that are considered less developed or undeveloped, local conditions need to be substituted into health risk assessments where possible.

PREFACE

The work described in this dissertation was carried out in the School of Life and Environmental Sciences, University of Natal, Durban, from January 2000 to March 2001, under the supervision of Professor R.D. Diab of the University of Natal and Mr. R. Hounsome of the CSIR.

These studies represent original work by the author and have not otherwise been submitted in any form for any degree or diploma to any tertiary institution. Where use has been made of the work of others it is duly acknowledged in the text.

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DEFINITIONS

Acute:	short-term exposure, typically 1 hour
Anemia:	reduced number of erythrocytes or red blood cells
Aplastic anemia:	anemia characterised by a greatly decreased formation of erythrocytes or red blood cells
Asphyxiant:	a poison depriving the tissues of oxygen thus causing asphyxiation or suffocation
Asthma:	a condition of the lungs in which there is widespread narrowing of airways, varying over short periods of time either spontaneously or as a result of treatment
Ataxia:	failure of muscular coordination, irregularity of muscle action
Attention deficit:	an inability to control behaviour due to difficulty in processing neural stimuli
Bronchitis:	inflammation of one or more bronchial tubes, usually secondary to infection
Bronchiolitis obliterans:	inflammation of the bronchioles with obstruction by fibrous granulation tissue or bronchial exudates, often follows inhalation of irritating gases/foreign bodies and complicates pneumonia
Broncho-constriction:	reduction in the size of the bronchus or bronchial tube
Carboxyhaemoglobinemia:	the presence of carboxyhaemoglobin (stable union of carbon monoxide with haemoglobin) in the blood
Carcinogen:	any substance that causes cancer
Cardiac arrhythmia:	irregularity of the heart beat
Cerebral atrophy:	wasting of the tissues in the cerebrum
Chronic:	long-term exposure, typically annual
Chronic bronchitis:	chronic inflammation of the mucous membrane of the bronchial tubes
Chronic lymphocytic leukemia:	a slowly progressing form of leukemia, characterised by an increased number of lymphocytes
Craniofacial and limb abnormalities:	congenital structural deformities, malformations or other abnormalities of the cranium and facial bones or of the extremities

CNS dysfunction:	disturbance, impairment or abnormality of the functioning of the central nervous system
Congenital abnormalities:	abnormalities of organs or parts existing at or before birth
Conjunctival infection:	infection of the mucous membrane covering the anterior surface of the eyeball and lining the lids.
Cyanosis:	a dark bluish or purplish colouration of the skin and mucous membrane due to deficient oxygenation of the blood
Developmental decrements:	loss or reversal of developmental state
Dysmorphism:	abnormality of shape
Dyspnea:	shortness of breath, difficulty or distress in breathing, usually associated with serious disease of the heart or lungs
Genotoxic:	a substance which damages the DNA of an organism
Haemolytic anemia:	anemia resulting from reduced red cell survival time, either due to an intrinsic defect in the erythrocyte or an extrinsic damaging agent
Hematologic:	relating to the blood and blood-forming tissues.
Hematologic neoplasms:	abnormal growths in the blood and blood-forming tissue (bone marrow and lymphatic tissue)
Hemorrhagic lungs:	profuse bleeding in the lungs
Hepatomegaly:	enlargement of the liver
Hodgkin's lymphoma:	a malignant disease characterised by progressive enlargement of the lymph nodes, spleen and general lymphoid tissue, and the presence of large, usually multi-nucleate Reed-Sternberg cells of unknown origin
Hypoxia:	reduction of oxygen supply to tissue below physiological levels despite adequate perfusion of tissue by the blood
Ischaemia:	a low oxygen state usually due to obstruction of arterial blood supply or inadequate blood flow causing tissue hypoxia
Lacrimation:	excessive secretion of tears
Leukemia:	progressive proliferation of abnormal leukocytes found in hemopoietic tissues, other organs, and usually in the blood in increased numbers

Leukopenia:	a condition in which the total number of leukocytes in the blood is less than normal
Lymphoma:	a general term that includes various, abnormally prolific, probably neoplastic diseases of the lymphoid tissues
Menorrhagia:	excessive or prolonged menstruation
Methaemoglobinemia:	a clinical condition in which 1% of haemoglobin in the blood has been oxidized to the Ferric (+3) state. Cyanosis occurs due to the inability of haemoglobin to transport oxygen. This condition is often caused by nitrites
Multiple myeloma:	a malignant neoplasm that originates in bone marrow and involves chiefly the skeleton
Mutagen:	any agent that causes the production of a mutation
Myelodysplastic syndrome:	consists of a clonal group of disorders which represent steps in the progression to the development of leukemia
Narcosis:	general and non-specific reversible depression of neuronal excitability, produced by a number of physical and chemical agents, usually resulting in stupor rather than anesthesia
Narcotic:	any substance producing stupor associated with analgesia
Non-cardiogenic pulmonary oedema:	a severe state of increased interstitial fluid within the lung that leads to flooding of the alveoli with fluid. This results in a severe disturbance of gas exchange across the alveolar surface
Nonlymphocytic leukemia:	distinguished from lymphocytic leukemia by the morphology of the marrow and blood cells
Obstructive lung disease:	a form of lung disease that manifests as acute or chronic narrowing or blockage of the smaller airways in the lungs, causing increased resistance to airflow in the bronchial tubes (e.g. asthma, silicosis). (Also called chronic obstructive pulmonary disease or COPD)
Pneumonia:	inflammation of the lungs with consolidation
Pneumonitis:	inflammation of the lung secondary to chemical, viral or bacterial infection

Preleukemia:	a general term referring to some non-cancerous blood disorders, such as myelodysplasia which carry an increased risk of the patient developing acute leukaemia
Pulmonary:	relating to the lungs
Pulmonary oedema:	a severe state of increased interstitial fluid within the lung leading to flooding of the alveoli with fluid, causing severe disturbance of gas exchange
Sinusitis:	inflammation of a sinus, especially paranasal
Small airways function:	capacity of the site of gas exchange in the lungs
Teratogen:	any substance that causes abnormal development
Thrombocytopenia:	a decrease in the number of platelets in the blood, resulting in the potential for increased bleeding and decreased ability for clotting
Ventilatory function:	breathing capacity, movement of gases into and out of the lungs
Whooping cough:	a bacterial infection with symptoms of a runny nose, fever, conjunctivitis, and characteristic coughing spells that end in a whoop caused by forceful inspiration of air

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LIST OF FIGURES

LIST OF TABLES

CHAPTER ONE: INTRODUCTION

1.1 Background

Traditional air quality studies have tended to focus on ambient air quality, with the assumption that the indoor environment provides some degree of protection from the outdoor air. Once stricter controls were enforced on ambient air quality it became clear that indoor air quality has a far greater potential to impact on human health than previously thought (Gammage and Kaye, 1987). Many studies have shown that the indoor concentration of a pollutant can often be higher than the outdoor concentration (for example: Biersteker *et al.*, 1965; Benson *et al.*, 1972; Cote *et al.*, 1974; Yocum, 1982). This can have a vast impact on human health as many people now spend far more time indoors than they do outdoors. Several of these studies have also been conducted in South Africa where poor indoor air quality is problematic for various reasons (Mehlwana and Qase, 1996; Bank *et al.*, 1996; Jones *et al.*, 1996; White *et al.*, 1996; Mehlwana and Qase, 1998).

The four main sources of pollutants that are associated with indoor air pollution include combustion, building materials, the ground beneath buildings and biological agents such as mould, mildew and dust mites. Within the category of 'combustion', domestic fuel use and passive smoking are most likely the main cause for concern (Behera, 1995).

Many low-income households in South Africa rely on fossil fuel combustion for heating, cooking and illuminating purposes. Notwithstanding the increasing electrification of both rural and urban areas, many of these households continue to use fossil fuels because of the large initial outlay required to purchase electrical appliances and the perceived difference in costs for fossil fuels vs. electricity (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 the 5 million households reported to rely on fossil fuels for cooking, heating and lighting purposes, a fairly high proportion rely on kerosene, also known as paraffin. Approximately 21% of the 5 million households use kerosene for cooking; about 14% use it for heating and approximately 13% use kerosene for lighting (SSA, 2000). (The full range of fuels that are used in South African households is outlined in Appendix 1.) Kerosene is also a popular fuel choice in the city of Durban. In a study conducted by Jones *et al.* (1996) it was shown that kerosene is used by more than 70% of the households sampled in low-income metropolitan areas of Durban.

Kerosene is an odourless, oily liquid that is pale yellow or white in colour. It is insoluble in water. After kerosene has been released into the atmosphere it may degrade by reaction with photochemically produced hydroxyl radicals (Baker, 2000).

Kerosene is positioned towards the middle of the 'energy ladder'. The 'energy ladder' is a term that is used to describe the hierarchy of energy forms available and used by humans. As one moves further up the ladder or hierarchy, energy sources become cleaner yet more expensive. At the bottom of the 'energy ladder' lie biomass fuels such as animal dung, crop residues and wood. These are followed by coal, coke, kerosene and gas. Finally, electricity lies at the top of the ladder, being one of the cleanest combustible fossil fuels (Behera, 1995).

Exposure to the air pollutants that arise during kerosene combustion is likely to have an impact on (or cause) certain health problems within the households using kerosene for cooking, heating and illuminating purposes. The actual pollutants emitted during kerosene combustion depend on the fuel composition,

the appliance type and condition and the ventilation conditions. The list of pollutants most often includes:

- carbon monoxide
- carbon dioxide
- sulphur dioxide
- nitrogen dioxide
- particulate matter
- formaldehyde
- and various hydrocarbons (Leaderer, 1982; Traynor and Allan, 1983).

Hydrocarbons can be classified according to the following groups:

- aliphatics (open chain hydrocarbons)
- cyclo-aliphatics (closed chain hydrocarbons), and
- aromatics (benzene rings and derivatives thereof) (Hart, 1987).

The hydrocarbons that are typical of kerosene combustion can be grouped as follows:

- aliphatics
    - alkanes
        - nonane
        - decane
        - *n*-undecane
        - *n*-dodecane
        - *n*-tridecane
        - *n*-tetradecane
        - *n*-pentadecane
        - *n*-hexadecane
    - alkenes
        - 1, 3 butadiene
- aromatics
        - benzene

- toluene
- ethylbenzene
- isopropylbenzene
- propylbenzene
- xylenes
- trimethylbenzenes

o polycyclic aromatics
- naphthalene
- methylnaphthalene (B.J. de Vos, *pers. comm.*, October 2000)

The pollutants listed above can cause various health effects once exposure has occurred. The health effects associated with exposure to these pollutants vary according to the length of time an individual has been exposed to the pollutant, and the concentrations they have been subjected to. The health effects of each of these pollutants will be discussed in further detail in Chapter 2.

1.2 Study Area

This study will focus on kerosene use in an informal settlement in Durban known as Cato Manor. Cato Manor has a history that is perhaps typical of many settlements in South Africa during the apartheid era. In 1845 the first mayor of Durban, George Cato, was granted the land now known as Cato Manor in exchange for property on the beachfront that had been expropriated by the military. This land was farmed until 1900 and then subdivided into plots that were either hired out or sold to Indian market gardeners. From 1900 to 1959 Cato Manor was inhabited by many Indian families as well as Black South Africans despite Apartheid laws that prevented them from living in urban areas. It was only in the 1960's, after much violent conflict, that the area was forcefully cleared of its 'illegal' inhabitants. In the mid-1980's re-settlement of the area began. In 1992 the Cato Manor Development Forum was set up and in 1993 the Cato

Manor Development Association was established as a Section 21 company which began to implement the re-development of Cato Manor (CMDA, 1998).

Cato Manor is situated 7km to the west of the central business district of Durban. The Greater Cato Manor area is approximately 2000 hectares of land that is divided into 19 geographical units called precincts (CMDA, 1998). Cato Manor is bordered in the north by the Pavilion Shopping Centre and the N3 freeway (see Figure 1.1). The boundaries to the east and west are created by the residential area of Manor Gardens and the University of Natal, and Westville Prison respectively. In the south Cato Manor is demarcated by Sarnia Road. The Greater Cato Manor area is home to approximately 80 000 people (S. Gielink, *pers. comm.*, March 2001).

The area of Cato Crest (shown in dark grey in Figure 1.1) is the area under consideration in this study. Cato Crest houses roughly 4 500 households, and with an estimated 4 to 5 people per household there are approximately 18 000 to 23 000 people living in the area (S. Gielink, *pers. comm.*, March 2001). Population surveys have not been conducted in the area of Cato Crest therefore figures are an approximation of what the population is thought to be.

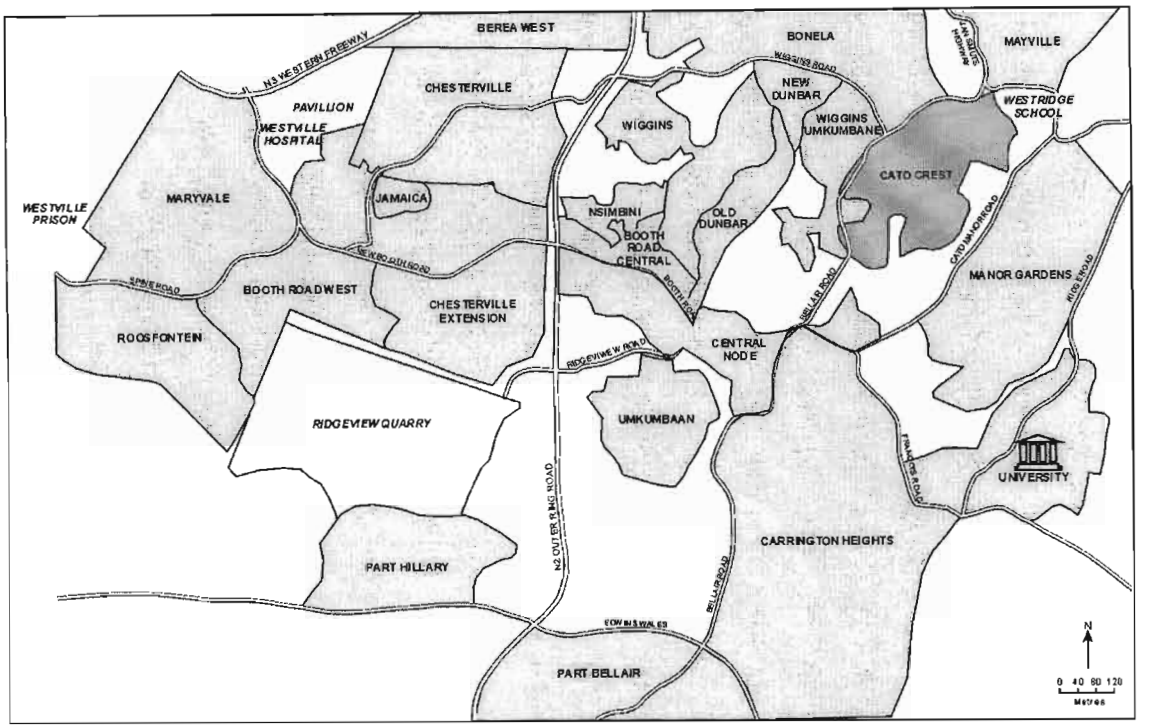

Figure 1.1: Location of Cato Crest within Greater Cato Manor. (Source: University of Natal, Durban.)

Of the 2000 hectares of land, only 900 hectares have been made available for development. These 900ha have been apportioned for development in the following manner:

- 51% for housing,
- 25% for roads and transport routes,
- 13% for social facilities, and
- 11% for industrial / commercial use (CMDA, 1998).

Development of the remaining land is restricted due to unfavourable topographical and geological conditions, and conservation areas that have already been established (CMDA, 1998).

1.3 Study Objectives

The objective of this study is to investigate the application of the United States Environmental Protection Agency (US EPA) human health risk assessment framework for quantifying the adverse human health effects of exposure to inhaled kerosene pollutants in the South African context. The study will focus on the informal settlement of Cato Crest, Durban and will be based on indoor air quality data collected in the area as well as time-activity pattern data that will be collected from the community through questionnaires.

The key questions that will be asked in this study are as follows:

- What are the potential adverse human health effects associated with the inhalation of kerosene combustion products in informal urban settlements?
- How do time-activity patterns in a typical South African informal settlement differ from those provided by the US EPA Exposure Factors Handbook?
- Are the default values provided by the US EPA for the exposure assessment an accurate representation of local conditions?
- Is the calculated risk value affected in any way when the risk assessment uses US EPA default exposure values as opposed to locally collected information?
- Is the US EPA human health risk assessment framework applicable in the South African context?

CHAPTER TWO: THE ENVIRONMENT AND HUMAN HEALTH

2.1 Environmental Health

Diseases are caused or influenced by either environmental factors, or alternatively, by genetic factors. Environmental factors affecting disease tend to influence each other, whereas genetic factors influence the way in which the environment will affect an individual (Beaglehole *et al.*, 1998).

Environmental factors that can have an impact on health include psychological factors, biological and accidental factors, physical factors and chemical factors. It is the chemical factors such as dust, tobacco smoke and pollutants arising from increasing industrialisation and urbanisation that contribute to increased adverse health effects in urban areas. In addition, the combustion of fossil fuels in the domestic environment increases an individual's exposure to certain pollutants that would otherwise have been fairly limited (Beaglehole *et al.*, 1998).

Each individual also has certain characteristics that make him or her susceptible to disease. These characteristics include:

- Genetics
- Nutritional state
- Presence or absence of disease
- Age and gender
- Physical condition
- Personality (Beaglehole *et al.*, 1998).

Exposure to a chemical will cause a wide range of effects depending on the actual dose received. The effects can range from *not noticeable* through *slight* to *severe illness* and eventually *death*. Generally, the severity of the effect is increased as the dosage increases. At a low dose there are few people who

experience an effect, while at a high dose almost everyone that has been exposed exhibits an effect.

2.1.1 Previous Studies

Many studies have been conducted in developing countries on the domestic use of fossil fuels such as kerosene. All of these studies recognise that domestic cooking is a significant and important source of indoor air pollution as more than half the world's households cook daily with unprocessed fuels (Behera *et al.*, 1998). The location of the kitchen in the house, the ventilation conditions and the fuel type are all factors contributing to the indoor air quality in many homes in developing countries, while the severity of the health risk depends on the length and level of exposure of each individual.

A study by Behera *et al.* (1994) was conducted in India on the ventilatory function of women using different cooking fuels. This study showed that the ventilatory function of non-smoking women using kerosene and other fossil fuels was impaired to a certain extent. Small airways function is reported to be lowest in kerosene users (Behera, 1997). Blood carboxyhaemoglobin levels were also found to be high in all four fuel user groups (biomass, liquefied petroleum gas or LPG, kerosene and mixed use) indicating significant indoor air pollution associated with cooking with fossil fuels. Earlier observations reported in this paper revealed interstitial fibrosis type lesions due to domestic cooking with fossil fuels such as kerosene.

Behera *et al.* (1998) in a study entitled "Respiratory symptoms in Indian children exposed to different cooking fuels" reported that the children from 'kerosene households' showed the most respiratory symptoms compared to other households using biomass, LPG and mixed fuels (52% of the 'kerosene households' group were symptomatic). Symptoms that were reported by the children in the study included a cough persisting for more than 4 days per week, chest congestion with a cold and phlegm for more than 1 week. Historical

accounts of other illnesses such as sinusitis, bronchiolitis, whooping cough and pneumonia were similar for all 4 fuel groups. Behera and Jindal (1991) showed respiratory symptoms in 11.4% of women using kerosene stoves with dyspnea being the primary complaint.

Carboxyhaemoglobin concentrations of women using either biomass, LPG, kerosene or all 3 fuels were studied by Behera *et al.* (1991). The women using kerosene showed significantly higher levels of carboxyhaemoglobin in the blood than any of the other fuel use groups and the control group (7.52% vs. 3.52% for the control group). Individuals sensitive to carbon monoxide poisoning include infants, the elderly and patients with cardiovascular disease, anaemia and lung disease (Behera, 1995).

Leaderer *et al.* (1998) showed significantly higher levels of PM_{10}, $PM_{2.5}$, sulphate, NH_4^+ and SO_2 inside homes using kerosene heaters in winter compared to outdoor concentrations of the same pollutants. The indoor pollutant concentrations correlated closely with the number of hours of heater use.

Sharma *et al.* (1997) tried to determine the incidence of acute lower respiratory infection (ALRI) in relation to indoor air pollution from kerosene and wood used for cooking in urban slums in India. Of the households using kerosene for cooking, 33% showed ALRI with pneumonia and bronchiolitis being the most common ailments.

Mumford *et al.* (1992) showed that kerosene heaters released organic compounds known to be mutagenic (e.g. nitropolycyclic hydrocarbons). Zhang and Smith (1999) showed that kerosene stoves produce aldehydes and carbonyl compounds known to produce genetic damage and cancer.

No studies on the health effects of kerosene use in Cato Manor have been undertaken, although fuel use studies have been done in the area.

2.1.2 Environmental Health Linkage Methods

As environmental management became commonplace in many countries around the world it became important to be able to predict the potential health effects likely to arise from an industrial or other activity. A means of determining the health effects of different pollutants was necessary in order to quantify the effects of environmental pollution.

Currently there are three categories of environmental health linkage methods used for this purpose: analytical epidemiology, observational studies and health risk assessments.

Analytical Epidemiology

Analytical epidemiology is used to statistically prove the relationship between an observed health effect in a population and the environmental pollutant to which it is exposed. This method requires mostly primary data on environmental exposures and the health status of individuals. The most popular analytical study design methods include cross-sectional studies, cohort studies and case-control studies. Disadvantages include:

- the requirement of a large study population,
- the time taken to conduct the study (usually longer than 1 year),
- the expense of conducting such a study, and
- the possibility that the study may not always yield results due to confounding factors and bias (Briggs *et al.*, 1996).

Cross-sectional studies measure the number of diseased cases in a defined population. The measurements of exposure and effect are made at the same point in time making it a useful method for studying individuals with fixed characteristics such as socio-economic status or blood group. This method is relatively cheap and easy to carry out, however it provides results that have low

statistical power. The results also cannot reveal whether exposure to a pollutant precedes or follows a health effect (Briggs *et al.*, 1996).

Cohort studies begin with a disease-free group of people who are classified into smaller groups according to their exposure to a potential cause of disease. These studies are major undertakings as they must continue to track individuals until the disease manifests itself (often years later), thus providing fairly accurate information about the causation of disease. This method is often used in an occupational health environment. The cost of this type of study is the biggest disadvantage (Briggs *et al.*, 1996).

Case-control studies begin with a group of people afflicted by a certain disease and a control group who are not afflicted with the disease of interest. The two groups of people are then examined to see how a particular environmental exposure affects them. A case-control study can be done with retrospective or prospective environmental exposure and it is fairly simple to conduct (Briggs *et al.*, 1996).

Observational Studies

These studies are used to associate patterns of disease with environmental exposure. Health trends in specific geographic locations can be identified quickly but cannot be statistically proven to be as a result of environmental exposure. Observational studies can be carried out on a much lower budget than analytical studies and are often used as 'pilot investigations' for analytical epidemiology studies. Traditional observational studies include ecological studies, time-series studies and GIS (Geographical Information Studies) analyses (Briggs *et al.*, 1996).

Ecological studies can be used to identify disease prevalence across time and space and in relation to social, behavioural and other factors. Ecological studies use group rather than individual data and are most often used to see the results

of intervention. The ecological study is able to detect relatively small increases in risk due to a specific variable, however this can only be done for groups and the potential for bias is very high making the interpretation of results difficult (Briggs *et al.*, 1996).

Time-series analysis examines the relation between exposure and health observations recorded at consecutive (equally spaced) points in time. Health outcomes are predicted using regression modelling. This method is mainly used for exploring patterns in a series of observations in order to quantify causal relationships. Disadvantages of this method include:

- no explanation of toxicological probability of various health effects,
- time consuming, and
- interpretation of the results can be difficult (Briggs *et al.*, 1996).

Geographical Information Systems (GIS) is not an environmental health linkage method but rather an environmental health linkage *tool*. GIS can be used to analyse environmental health data for spatial correlations between disease prevalence and environmental exposure. GIS can be very useful for determining the relationship between air pollution and adverse health effects. GIS can be a relatively inexpensive tool if existing data are used, however, where data acquisition and cleaning are necessary the costs can become significant. GIS is also a very powerful tool for data presentation. Results from a GIS study can be subject to bias and uncertainty because of poor or unknown data quality (Briggs *et al.*, 1996).

Health Risk Assessment

Health risk assessment (HRA) is used to determine the potential adverse health effects that are likely to arise from exposure to an environmental pollutant. The advantage of a health risk assessment over observational studies or analytical epidemiological studies is that a health risk assessment is *predictive* in nature and uses existing exposure data to quantify health effects of exposure to a

certain substance (Briggs *et al.*, 1996). Validated epidemiological and toxicological data are used to estimate potential health effects.

The risk assessment process used in environmental management seeks to identify which environmental hazards are likely to occur and what type of health effect each hazard could cause. In addition, exposure levels are either measured or estimated and then used in conjunction with dose-response relationships to calculate the likely risk.

Advantages of risk assessment include:

- Quantification of health effects,
- Prediction of both long-term and short-term health effects is possible by changing the exposure time and concentration,
- The most important route of exposure can be determined,
- Can be used in areas where data on local health effects are insufficient,
- HRA requires the least resources and is the fastest and easiest of the environmental health linkage methods presented above,
- It allows for predictions of health effects for adults and children,
- The framework ensures consistency in the process, and
- It is the most sensitive of the methods (able to predict low levels of risk) (NRC, 1994).

The disadvantages of HRA include:

- Reliable epidemiological or toxicological data may not be available for the pollutant of interest,
- Synergistic effects of various pollutants in combination may complicate the study and their effects will not be known, and
- The study population may exhibit unknown behavioural patterns causing the actual exposure to influence the results (NRC, 1994).

2.2 The History of Human Health Risk Assessment

Approximately sixty years ago toxicologists began to set limits for exposure to hazardous substances in order to protect human health. It was widely held that every substance was toxic at some concentration, however, it was believed that for each substance there would be a threshold level below which no health effects would occur. Carcinogenic substances were then discovered to have no safe level of exposure. For these chemicals it was necessary to determine a maximum permissible human exposure which would still have some degree of risk associated with it. The US EPA then began to quantify low-dose risks for carcinogens using risk quantification methods. Through the 1970's and 1980's risk assessment became more popular and in 1983 the National Research Council (NRC) was asked to investigate the use of risk assessment in the federal government. This report included the risk assessment framework, specific definitions of risk assessment and its steps which have become widely adopted and are still in use today (NRC, 1994).

After the NRC report was produced there were many advances in the science of risk assessment such as:

- Development of guidelines for carcinogen risk assessments,
- Specification of default options,
- Distinction between risk assessment and risk management, and
- Development of guidelines for evaluation of mutagenicity, developmental toxicity, effects of chemical mixtures and reproductive risk (NRC, 1994).

Section 12 of the Clean Air Act (USA) required that the US EPA set emissions standards for hazardous air pollutants that would protect human health with an 'adequate margin of safety'. This ambiguous statement resulted in only 7 pollutants having standards set, as the EPA first had to determine what 'safe' levels were and then set the standards. The acceptable degree of risk was set at 1 in 10 000 people for the most exposed person and 1 in 1 000 000 for the majority of the population. Emphasis was initially placed on quantitative methods

of estimating individual lifetime risk, however, Section 12 was subsequently adjusted to reduce the emphasis on quantitative risk assessment (NRC, 1994).

Health risk assessment can also be used in the assessment of adverse human health effects arising from exposure to non-carcinogenic air pollutants. For this an inhalation reference concentration (RfC) is determined, 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 p.39). The RfC is based on the assumption that toxic effects will not occur until a threshold dose is exceeded (NRC, 1994). Several agencies have since determined their own reference concentrations, such as the ATSDR Minimal Risk Level (Agency for Toxic Substances and Disease Registry MRL) and the Californian EPA Reference Exposure Level (REL).

Risk assessment has come under much criticism since its inception. Some of these criticisms are listed below:

- The default options have been seen as too conservative or not consistent with current scientific knowledge and are too rigid;
- Several issues of human exposure have been given too little consideration;
- Aspects such as synergism and risk aggregation have been ignored;
- Little attention has been given to non-cancer risks;
- Uncertainties are not adequately dealt with and some people believe there is still insufficient knowledge to make risk estimates;
- Upper-bound point estimates are too frequently used for decision-making purposes;
- Separation of risk assessment and risk management in the conceptualisation of risk assessment has resulted in separation of the procedures;
- Risk assessment is seen as too resource intensive; and

- There is a perceived lack of improvement in risk assessment methods (NRC, 1994).

2.3 Human Health Risk Assessment Approach

A human health risk assessment is defined by the US EPA as a qualitative and/or quantitative process conducted to characterise the nature and magnitude of risks to public health from exposure to hazardous substances released from specific sites (NRC, 1994). A qualitative human health risk assessment *describes* the potential risks that a population could be exposed to while a quantitative human health risk assessment seeks to put a *measure* to that potential risk. Risk assessment as a process can be extended further to incorporate risk communication and risk management.

A risk is the potential adverse effect that could be caused by a hazard. A hazard is a chemical, physical or biological agent or set of conditions that has the potential to cause harm. Risk is determined by the nature of the hazard, the exposure potential, the exposure population characteristics, the likelihood of occurrence and the magnitude of exposure (NRC, 1994).

Any health risk assessment follows a defined procedure in order to determine the risk potential. The procedure, developed by the National Academy of Science in the USA, consists of the following steps (as shown in Figure 2.1):

- Hazard identification
- Exposure assessment
- Dose-response assessment
- Risk characterisation

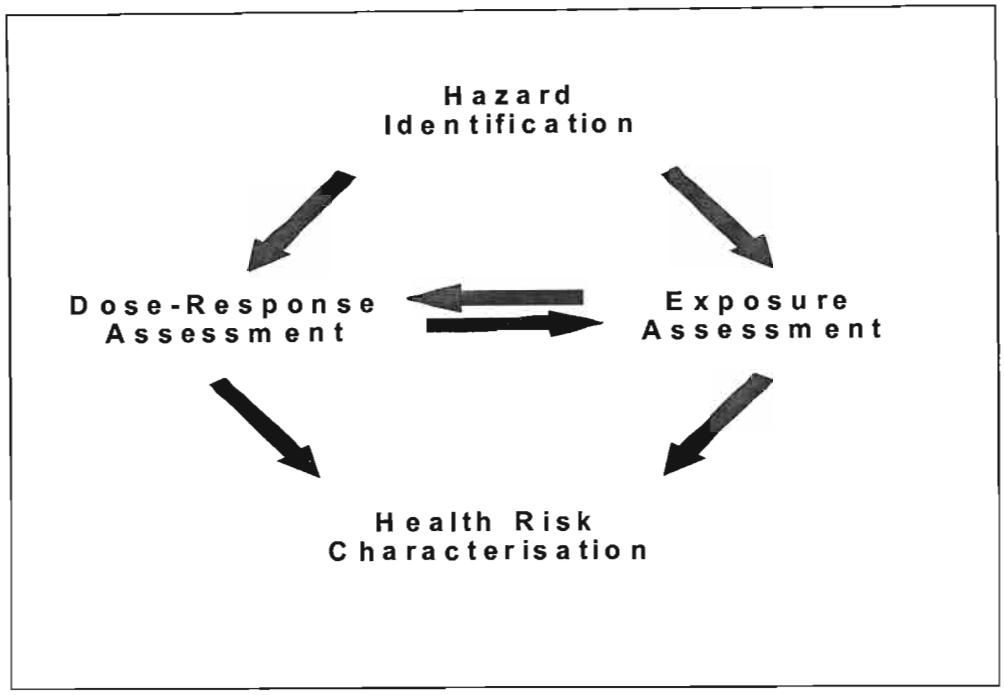

Figure 2.1: Human Health Risk Assessment Framework, as outlined by the National Academy of Science (American Chemical Society, 1998).

The hazard identification step is aimed at determining if exposure to a particular substance could result in 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).

The actual risk associated with a hazard can only be assessed and measured once damage due to exposure to that hazard has occurred. Human health risk assessment is a *predictive* process that is able to assess the *likelihood* of adverse health effects occurring as a result of exposure to a hazardous substance. The risks can therefore only be *estimations* of what *could* occur, and thus have uncertainty associated with them. Human health risk assessments are generally quite conservative as they include many safety factors that are built into the process. The final risk estimate is therefore likely to overstate the actual risk.

2.3.1 Hazard Identification

A hazard is described by van Leeuwen and Hermens (1995) as the potential of a chemical or mixture of chemicals to cause adverse human health effects at a particular exposure level. Hazard identification is therefore the process of recognising which chemicals or mixture of chemicals in the environment are likely to be responsible for adverse human health effects should exposure to that chemical or chemical mixture occur. Hazard identification also involves identifying which adverse human health effects are likely to occur following both acute and chronic exposure to the hazardous substances (Paustenbach, 1989; van Leeuwen and Hermens, 1995).

2.3.2 Exposure Assessment

The exposure assessment aims to identify the following:

- emissions, pathways, rates of movement, transformation reactions and fate of hazardous substances in the environment,
- estimated concentrations of the hazardous substances to which the target population is exposed,
- target population exposed to the hazardous substances, and the target organs in the body which are affected by exposure to the hazardous substances,

- magnitude, frequency and duration of exposure of the target population, as well as behaviour patterns, geographic distribution and population size of the target population, and
- estimated dose for carcinogenic and non-carcinogenic substances (Paustenbach, 1989; van Leeuwen and Hermens, 1995).

The exposure assessment can be based either on default values suggested by the US EPA, on local values collected from the community where the study is taking place, or a combination of the two. This study has been based on both US EPA default values as well as local data collected from Cato Crest.

The US EPA default exposure values are obtained from the Exposure Factors Handbook (US EPA, 1996). The values that are in this handbook are developed through numerous studies on North American populations. They are provided as guidance exposure factors to those conducting risk assessments.

2.3.3 Dose-Response Assessment

The dose-response assessment is the process of identifying the relationship between the exposure level or dose and the severity of the health effects likely to be experienced. This analysis uses benchmark values derived from quantitative studies such as epidemiological studies or experimental laboratory or field studies on animals and/or humans. Dose-response relationships often indicate the presence of different toxic effects of a substance at different concentrations. Dose-response is also affected by population characteristics such as age, gender, lifestyle, occupation and existing diseases, among other things (Paustenbach, 1989; van Leeuwen and Hermens, 1995; Gratt, 1996).

There are currently several sources of these benchmark values, namely the ATSDR, the US EPA and the Californian EPA Office of Environmental Health Hazard Assessment.

Agency for Toxic Substances and Disease Registry

The ATSDR benchmark values are known as MRLs or Minimal Risk Levels and are "an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse *non-cancer* health effects over a specified duration of exposure" (ATSDR, 2000 p.1). The MRLs are derived through extensive examination of all available toxicological and epidemiological information on the substance, and are only derived once the ATSDR concludes that sufficient reliable data exist to identify the most sensitive health effect or target organ for a specific exposure pathway. The MRLs are set at a level below which sensitive individuals are not likely to experience adverse health effects and often have an uncertainty factor associated with them. This is due to the fact that the MRL can be derived from studies done on animals rather than humans, as well as from studies done on healthy rather than sensitive individuals.

United States Environmental Protection Agency

The US EPA benchmark value is known as the reference concentration or RfC (for inhalation only). The RfC is derived from previous dose-response studies and indicates the level at which no adverse non-carcinogenic health effects will occur. Another US EPA benchmark value is the inhalation unit risk or IUR. The IUR is used to determine the carcinogenicity or cancer potency of inhaled pollutants (Gratt, 1996).

Californian Environmental Protection Agency

The Californian EPA benchmark values are known as Reference Exposure Levels or RELs. They are derived for both acute and chronic exposure to toxic air pollutants and can be described as the concentration at or below which adverse health effects are unlikely to occur. Cancer Potency Factors are used by the Californian EPA for assessment of exposure to carcinogenic air pollutants. The RELs and Cancer Potency Factors are derived in a very similar way to the US EPA benchmark values and also include a margin of safety to account for data

uncertainty (Californian EPA, 1997; Californian EPA, 1999*a*; Californian EPA, 1999*b*).

Uncertainty Factors

Each benchmark value has a certain amount of uncertainty associated with it. When deriving benchmark values for the dose-response assessment, there is often a lack of data on the effects of a particular substance on humans. For this reason, surrogate data are sometimes used to derive a level at which humans will not experience adverse health effects, thus leading to uncertainty about the accuracy of the benchmark value. Data extrapolations that can be made when deriving benchmark values are as follows:

- animal to human extrapolation (UF_a)
- healthy individual to sensitive individual extrapolation (UF_h)
- sub-chronic to chronic extrapolation (UF_s)
- lowest observable adverse effect level (LOAEL) to no observed adverse effect level (NOAEL) extrapolation (UF_l)

In addition to the extrapolation uncertainty there is also a

- modifying factor (MF), and a
- data quality / quantity factor (DF)

that account for any additional uncertainty (Californian EPA, 1997; Californian EPA, 1999*a*; ATSDR, 2000).

The data extrapolations are not only used in the calculation of the benchmark value, but are also used to calculate the uncertainty factor. The uncertainty factor is reported together with the benchmark value but is a totally separate entity. It provides an indication of the confidence that can be placed on the benchmark value. For example, if a benchmark value is based on animal data of poor quality rather than human data, the UF_a will be given a value between 1 and 10. If UF_a is said to be 10, DF is said to be 10 because of poor quality data and each other value is said to be 1 (meaning no other surrogate data were used), the total uncertainty factor for that benchmark value will be:

$$\text{Uncertainty Factor} = UF_a \times UF_h \times UF_s \times UF_l \times MF \times DF$$
$$= 10 \times 1 \times 1 \times 1 \times 1 \times 10$$
$$= 100$$

This uncertainty factor then provides an indication of high, medium or low confidence in the benchmark value and the data it was derived with.

2.3.4 Risk Characterisation

The process of risk characterisation should be clear and transparent, showing consistency and reason. This final step of the risk assessment is where both cancer and non-cancer risks are calculated (US EPA, 1998). The final risk result is a calculation of the upper-bound excess lifetime cancer risk and/or non-carcinogenic hazards for each media pathway and receptor considered. Risks and/or hazards can be summed for individual receptors across exposure pathways in order to get a total individual risk and/or hazard. It has been suggested by the US EPA that both individual risk and population risk be calculated in a risk assessment (US EPA, 1998). Population risk is defined as the aggregate risk of the exposed population and will incorporate risks associated with different exposure scenarios as well as the proportion of the population represented by each exposure scenario (US EPA, 1998).

Carcinogenic health effects are expressed as a cancer risk which is defined as the incremental probability that an individual will develop cancer over a lifetime as a result of an exposure to a carcinogen (US EPA, 1998). The cancer risk is expressed, for example, as a 1 in a million chance of developing cancer due to long-term exposure to the carcinogen.

Non-carcinogenic health effects are expressed as a hazard quotient (HQ) which indicates the presence or absence of adverse health effects due to exposure to a pollutant. The HQ can also provide an indication of whether only sensitive individuals will be affected, or if both healthy and sensitive individuals will be

affected. Non-cancer risks can be calculated for both acute and chronic exposure scenarios (US EPA, 1998).

2.3.5 Uncertainties and Limitations of the Study Approach

Uncertainty is introduced into every risk assessment in every step of the process. The uncertainty occurs primarily due to the fact that a risk assessment integrates information of the following nature: pollutant releases into the environment; the fate and transport of those pollutants in changeable environments through poorly understood and often unquantifiable methods; the potential for adverse human health effects obtained through extrapolation from human and animal studies and the probability of adverse human health effects given the genetic and other causes of diversity within the human population (US EPA, 1998).

The risk assessment process used in this study relies on the use of several point values in order to determine a point estimate of risk. It is unknown at what percentile of the distribution of risk this point estimate lies (US EPA, 1998). The actual risk associated with a hazard can also be assessed and measured only once damage due to exposure to that hazard has occurred. Human health risk assessment is a *predictive* process that is able to assess the likelihood of adverse health effects occurring as a result of exposure to a hazardous substance. The risks can therefore only be *estimations* of what could occur, and as such have uncertainty associated with them. Human health risk assessments are generally quite conservative as they include many safety factors that are built in along the process. The final risk estimate is therefore likely to overstate the actual risk.

Variability is often mistaken for uncertainty, however the two terms are distinctly different. Variability is the natural variation within physical and biological processes and populations. Variability is not reducible through further research or information gathering. Uncertainty is the imperfect or incomplete knowledge that a person has about the value of a variable or the variability within an individual or

a population (US EPA, 1998). The uncertainty in a study can be reduced through further study or better analysis techniques.

The uncertainty associated with this health risk assessment study can be categorised under the headings of variable uncertainty, model uncertainty and decision-rule uncertainty (Finkel, 1990, in US EPA, 1998).

Variable uncertainty occurs when the variables used in equations throughout the assessment cannot be accurately measured for a particular reason. This is either due to the limitations of any measuring or analysis equipment being used or the spatial and/or temporal variances in the measurements. Errors such as random or sample errors are included in the category of variable uncertainty (US EPA, 1998).

Model uncertainty is associated with all phases of the risk assessment and is introduced in various ways. Firstly, animal models used as proxy for human toxicity and carcinogenicity testing introduce uncertainty through interspecies variability in sensitivity to different chemicals. The dose-response models used in extrapolations introduce uncertainty through limited or poor understanding of the mechanisms of toxicity and related dose-response relationships. Thirdly, the computer models used to predict fate and transport of pollutants in the different environmental media introduce uncertainty by being oversimplifications of reality and through the exclusion of certain variables due to lack of data or the resulting increased complexity (US EPA, 1998).

Decision-rule uncertainty arises out of each decision that is taken and each assumption that is made through the risk assessment. It is subject to the risk assessor's knowledge and skill as well as the decision-maker's knowledge and ultimate objectives. Decision-rule uncertainty is also influenced by the use of standard US EPA default values during the analysis. This uncertainty could be reduced by using a range or distribution for each variable, although one could

also collect locally relevant information for these variables from the study area being looked at. This is particularly relevant in this study as the area under consideration contains a South African rather than a North American population. Decision-rule uncertainty is also influenced through the use of benchmark values in the dose-response assessment. The benchmark values have both uncertainty and variability associated with them.

2.4 Health Effects of Kerosene Combustion Products

Each of the products of kerosene combustion has certain health effects on the human body. These health effects are outlined below. The list includes all those pollutants found in the air after kerosene combustion, and while some may be more important than others, they may all contribute to adverse human health effects.

2.4.1 Carbon monoxide

Carbon monoxide is produced when carbon-containing fuel burns incompletely. It is a toxic gas that reacts with the iron in haemoglobin thereby displacing oxygen from the blood causing suffocation. Carbon monoxide poisoning is largely due to acute exposure to very high concentrations of approximately 1000ppm (Kotz and Purcell, 1991).

Acute exposure to carbon monoxide can cause many different health effects that are due to tissue hypoxia and cellular poisoning (TOXNET, 2000). Sensitive individuals include people with chronic bronchitis or asthma. Other people at a higher risk of CO poisoning are infants, pregnant women, elderly people and those with chronic obstructive pulmonary disease (COPD) or ischemic heart disease.

Mild toxicity after minor exposure to CO is indicated by the presence of a temporal headache, dizziness and shortness of breath during physical exertion.

Individuals with cardio-vascular or cerebro-vascular diseases could suffer a heart attack or stroke. Moderate exposure can lead to severe headaches, dizziness, nausea, vomiting and a feeling of weakness. Individuals may also experience a rapid heart beat, shallow respiratory rate, fainting and cardio-vascular toxicity. Severe CO toxicity is characterised by fainting, seizures, coma and cardio-vascular toxicity. Respiratory failure may occur and can lead to death (TOXNET, 2000).

Inhalation of carbon monoxide can cause carboxyhaemoglobinemia, although the level of carboxyhaemoglobin in the blood does not always correlate with the severity of exposure. Carbon monoxide has an affinity for haemoglobin that is 240 times that of oxygen, in addition to binding more strongly to haemoglobin than oxygen. Carboxyhaemoglobin formation is reversible although accumulation of carboxyhaemoglobin may occur as the halftime for elimination is 2 to 6½ hours (Godish, 1997). A carboxyhaemoglobin concentration in the blood of 25% is generally considered toxic (TOXNET, 2000).

Certain studies have shown that exposure to carbon monoxide can cause an increased risk of multiple myeloma (Richardson, 1993). Animal studies have shown that inhalation of carbon monoxide can lead to reduced foetal growth and viability (Richardson, 1993).

2.4.2 Carbon dioxide

Carbon dioxide (CO_2) is one of the main greenhouse gases. It is one of the end products of incomplete combustion of organic compounds such as fossil fuels.

Carbon dioxide is an asphyxiant that displaces oxygen from the breathing atmosphere and causes hypoxia (TOXNET, 2000). Susceptible individuals include those with pre-existing cardiac, pulmonary or hematologic diseases. Carbon dioxide poisoning is generally due to acute exposure.

There are four stages that characterise acute CO_2 exposure depending on the arterial oxygen saturation. The first stage is known as the 'indifferent stage', where the arterial oxygen concentration is still 90%. The only clinical effect evident in this stage is decreased night vision. The second or 'compensatory stage' is characterised by an arterial oxygen concentration of 82 to 90%. Clinical effects include an increase in respiratory rate and pulse, a further decrease in night vision and reduced alertness and performance ability. Stage three is the 'disturbance stage', where arterial oxygen saturation is between 64 and 82% and the clinical effects include a hunger for air, fatigue, tunnel vision, headaches and dizziness, numbness, poor judgement, memory loss, hyperventilation and cyanosis. It becomes increasingly difficult for the exposed individual to escape the harmful environment. The 'critical stage' is the final stage where arterial oxygen saturation is 60 to 70% or even less. At this point individuals will experience a decline in judgement and co-ordination, followed by incapacitation and loss of consciousness (TOXNET, 2000).

2.4.3 Nitrogen dioxide

Nitrogen dioxide is a brownish, highly reactive but relatively insoluble gas present in the urban atmosphere. Nitrogen dioxide (NO_2) pollution in South Africa arises mainly from combustion processes of power stations using fossil fuels, motor vehicles (using both petrol and diesel fuel sources), and from the use of coal and other fossil fuels (such as kerosene) as an energy source in many homes in the country.

The acute toxic health endpoint of concern for NO_2 exposure is the respiratory system. Acute exposure to NO_2 appears to cause chronic changes in the respiratory system (Californian EPA, 1999*a*). Clinical effects of nitrogen dioxide exposure include a shallow respiratory rate, rapid heart beat, wheezing and cyanosis. Shortness of breath and coughing lasting from 2 to 3 weeks may also occur (TOXNET, 2000).

The chronic toxic health end point of concern for NO_2 is both the respiratory system and the immune system. Nitrogen dioxide is not a carcinogen. Chronic exposure of humans to NO_2 has shown that there are 3 types of response: firstly, an increased sensitivity to broncho-constrictors; secondly, increased airway resistance; and thirdly, increased susceptibility to respiratory infections. Sensitive individuals include children, asthmatics, and individuals with chronic obstructive pulmonary disease (COPD) (Californian EPA, 1997).

2.4.4 Sulphur dioxide

Sulphur dioxide or SO_2 is a colourless, toxic gas that has a choking, acrid odour (3 to 5ppm). It dissolves easily in water to form sulphurous acid and is a key component of acid rain (Kotz and Purcell, 1991). Sulphur dioxide is a product of kerosene combustion and is also used as a preservative in many foods, as well as a disinfectant and bleaching agent in several industries. Industry contributes significantly to the atmospheric sulphur dioxide concentrations through the combustion of sulphur-containing coal and other fossil fuels (Richardson, 1993).

The health endpoint of concern for sulphur dioxide exposure is primarily the mucous membranes (e.g. eyes, nasopharynx) and respiratory system. The clinical effects of acute exposure to sulphur dioxide include broncho-constriction, coughing and wheezing, upper respiratory obstruction and pulmonary oedema (TOXNET, 2000). Other effects include lacrimation and conjunctival infection. Individuals who survive exposure to elevated concentrations may develop reactive airway disease, obstructive and restrictive lung disease or chronic bronchitis.

Sulphur dioxide, in the presence of particulate matter and other photochemical pollutants, aggravates chronic pulmonary disease and also increases the risk of acute and chronic respiratory illness. Pulmonary mucociliary clearance is also impaired due to the deposition of hydrogen ions on the bronchial lining (TOXNET, 2000).

2.4.5 Formaldehyde

Formaldehyde is a colourless gas with a pungent, irritating odour. It is a highly soluble compound that combines easily with other substances and is soluble in water. Formaldehyde is a product of incomplete combustion and arises from kerosene combustion as well as various other sources, namely vehicles, refineries, stone/clay/glass production, resins and is an ingredient of household detergents, cosmetics and chemicals (EDF, 2000).

The non-cancer health effects from formaldehyde exposure include irritation of the respiratory tract and mucous membranes. Exacerbation of asthma can occur as well as coughing, wheezing, chest pain and bronchitis. Some studies have reported menstrual disorders and problems during pregnancy. Formaldehyde is a probable human carcinogen with a limited number of studies showing lung and nasopharyngeal cancer (EDF, 2000; US EPA, 2000*b*).

2.4.6 Particulate Matter

Particulate matter is the solid and/or liquid particles that are found in the air, having originated from any combustion activity or other dust generating activity. Particulate matter reduces visibility and can also harm crops and other plants. Particulates may be of variable sizes, the smaller the particle the more likely it is to travel deep into the lungs resulting in adverse health effects. Individuals with asthma, lung and heart disease are most susceptible to the effects of particulate matter, as are the elderly and young children. Particulates are likely to cause persistent coughs, wheezing and excess phlegm (EPA, 1997).

2.4.7 Nonane

Nonane is harmful through inhalation and is also a skin and mucous membrane irritant (Oxford Chemistry Department, 2000). Very little information could be found on this chemical.

2.4.8 Decane

Decane is a colourless liquid that is an eye, skin and respiratory irritant (Oxford Chemistry Department, 2000). Very little other information was available for this chemical.

2.4.9 Undecane

Undecane is a colourless liquid that is an irritant to the mucous membranes of the eyes and respiratory tract. It is also an irritant to skin. Inhalation of this chemical may be harmful (Oxford Chemistry Department, 2000).

2.4.10 Dodecane

Dodecane or *n*-dodecane is a colourless liquid which may be harmful if inhaled and can act as an irritant (Oxford Chemistry Department, 2000). Very little other information could be found on dodecane.

2.4.11 Tridecane

Tridecane is also known as *n*-tridecane and the health effects are similar to dodecane. Tridecane also acts as an irritant and may be harmful if inhaled (Oxford Chemistry Department, 2000). It is also a colourless liquid.

2.4.12 Tetradecane

Very little information could be found on tetradecane or *n*-tetradecane. Like dodecane and tridecane, it is also an irritant and may be harmful if inhaled (Oxford Chemistry Department, 2000).

2.4.13 Pentadecane

Pentadecane is a colourless liquid which is an irritant and may be harmful if inhaled. Very little is known about the health effects of pentadecane (Oxford Chemistry Department, 2000).

2.4.14 Hexadecane

Hexadecane is a clear, colourless liquid that is insoluble in water. It causes severe skin irritation and is extremely destructive to certain tissues (e.g. respiratory tract, mucous membranes, eyes and skin) (Oxford Chemistry Department, 2000). Inhalation of hexadecane is harmful and prolonged exposure can lead to narcotic effects (Baker, 2000). Hexadecane is not degraded through photolysis when released into the atmosphere. The chemical has a half-life of 10 to 30 days in the air.

2.4.15 1,3 Butadiene

1,3 Butadiene is a chemical that is produced during the manufacture of fuel. It is a colourless gas that has a petrol-like odour. 1,3 Butadiene is used to manufacture synthetic rubber for tyres and plastics such as acrylics and is also a product of kerosene combustion. In the air 1,3 butadiene breaks down rapidly in sunlight (approximately 2 hours) and more slowly in cloudy weather (a couple of days) (ATSDR, 2000*a*).

The health effects of exposure to 1,3 butadiene occur mainly in the central nervous system resulting in blurred vision, headaches, fatigue and tiredness, lower blood pressure, lower pulse rate and unconsciousness. Irritation of the mucous membranes of the eyes, nose and throat occurs at lower concentrations. This may lead to coughing and upper respiratory tract irritation (TOXNET, 2000). 1,3 Butadiene is a probable human carcinogen and carcinogenicity has been shown in some animals (TOXNET, 2000).

2.4.16 Benzene

Benzene (an aromatic hydrocarbon) is a clear, colourless liquid with a sweet odour. Benzene is a natural component of coal and petroleum and is mainly used in the production of other chemicals and as a solvent in chemical laboratories. It is also used to produce rubbers, lubricants, dyes, detergents, drugs and pesticides. Natural sources of benzene include volcanoes and forest

fires. The main source of benzene in the environment is industry. Benzene is one of the pollutants arising during kerosene combustion that is of great concern to human health (RAIS, 1998; ATSDR, 2000*a*; CCOHS, 2000; US EPA, 2000*c*).

Benzene is harmful if inhaled and will cause depression of the central nervous system below the normal functional level with symptoms including headache, nausea, dizziness, drowsiness and confusion. Benzene causes irritation of the mucous membranes, is carcinogenic and may be considered a mutagen. Long-term inhalation of benzene is responsible for its effects on the blood, mainly carcinogenic effects. Benzene vapour enters the human body after inhalation and also through the skin (approximately one quarter to one third of benzene enters the body via the skin). In order for benzene to actually become toxic to the human body it must be metabolised. The metabolites of benzene account for its toxicity (RAIS, 1998; ATSDR, 2000*a*; CCOHS, 2000; US EPA, 2000*c*).

As mentioned above, acute exposure to benzene causes depression of the central nervous system. Effects have not been reported below 25ppm. Exposures as high as 20 000ppm may result in death of the individual. Chronic exposure to benzene can lead to various types of anemia as well as various types of leukemia (RAIS, 1998; ATSDR, 2000*a*; CCOHS, 2000; US EPA, 2000*c*).

Benzene is able to cross the placenta and has been found in the umbilical cord in concentrations equal to those found in the mothers blood (RAIS, 1998).

2.4.17 Toluene

Toluene is an aromatic hydrocarbon. It is a clear, colourless liquid with a benzene-like odour. The odour threshold for toluene varies widely and the chemical may not be detected through odour after short exposure periods. Toluene is used in the manufacture of chemicals, dyes and explosives and is also used as a solvent in paints, resins, cleaners, glues and adhesives. It is also

found in certain fuels. Toluene, like benzene, is a great hazard to human health if exposure to kerosene pollutants occurs (RAIS, 1998; CCOHS, 2000).

Toluene's primary health effect is depression of the central nervous system. The symptoms experienced are related to the concentration that an individual is exposed to. Headaches and slight drowsiness are reported to occur at 50ppm. From 50 to 100ppm the nose, throat and respiratory tract become irritated, and individuals may experience fatigue and dizziness. At toluene concentrations above 200ppm, depression of the central nervous system becomes more pronounced with symptoms of numbness and nausea being experienced. Mental confusion and incoordination will occur at 500ppm, from 500 to 1000ppm the individual will become unconscious and could die. Chronic exposure to toluene or mixtures containing toluene could lead to permanent adverse central nervous system effects. Prolonged exposure has also been known to cause changes in kidney and liver function and may lead to an enlarged liver (RAIS, 1998; CCOHS, 2000).

2.4.18 Ethyl benzene

Ethyl benzene is a colourless liquid with a pungent odour which has narcotic properties in high concentrations. It is also an irritant to the skin and respiratory tract and is able to cause severe eye irritation. Ethyl benzene is also an experimental teratogen. It is commonly used as a solvent and in the manufacture of styrene. Ethyl benzene is also an additive in some fuels and is produced during the combustion of kerosene (Oxford Chemistry Department, 2000; CCOHS, 2000).

2.4.19 Isopropylbenzene

Very little information could be found on the effects of isopropylbenzene. Certain studies have shown that inhalation by humans can lead to behavioural changes, such as depressed activity and irritability. Animal studies have shown damage on the liver, kidney, bladder and spleen (CCOHS, 2000).

2.4.20 Propylbenzene

Propylbenzene is a colourless to light yellow liquid that is used in textile dyeing, printing processes and as a solvent. Propylbenzene occurs naturally in petroleum and bituminous coal. It is released to the atmosphere from combustion emissions such as those produced during domestic kerosene use. Health effects of exposure include irritation to mucous membranes, eyes, nose, throat and skin (CCOHS, 2000).

2.4.21 o, m, p Xylene

Xylene is a colourless, sweet-smelling (aromatic) liquid which exists in three isomers (ortho-xylene, meta-xylene and para-xylene). Xylene exists primarily as synthetically manufactured chemicals. It occurs naturally in petroleum and coal tar and is formed during forest fires. Xylene is used as a solvent and cleaning agent, and as a thinner for paint and varnish. It is used in the printing, rubber and leather industries and is also added to aviation fuel and petrol. Xylenes are found in the air after kerosene has been burned. Once xylene has entered the atmosphere it is broken down by sunlight into less harmful chemicals (ATSDR, 2000*a*; CCOHS, 2000).

Xylene is known to be a central nervous system depressant and also causes skin irritation. Acute exposure to high concentrations of xylene causes eye and mucous membrane irritation as well as breathing difficulties, memory loss, delayed reaction times and stomach discomfort. In addition, one can experience headaches, lack of muscle co-ordination, dizziness and confusion and changes in balance. Exposure to extremely high concentrations for prolonged periods of time may lead to narcosis, irritation of the respiratory tract, non-cardiogenic pulmonary oedema, loss of consciousness and eventually death. Animal studies have shown xylene exposure to delay growth and development and increase the number of deaths (ATSDR, 2000*a*). Xylene has not been classified as a human or animal carcinogen due to insufficient evidence (CCOHS, 2000).

Inhalation of xylene at a concentration of 1000ppm is considered to be immediately dangerous to health, symptoms range from loss of consciousness to respiratory failure and eventually death. Symptoms of pulmonary oedema may be delayed until several hours after exposure. Exposure to 6000ppm for longer than 12 hours can lead to death (TOXNET, 2000). These effects are not often seen however, as xylene can be detected through its odour at much lower concentrations (CCOHS, 2000).

2.4.22 1,3,5 Trimethylbenzene

1,3,5 Trimethylbenzene is an aromatic hydrocarbon that is a clear, colourless liquid with a distinct aromatic odour. It is derived from petroleum refining processes and the distillation of coal tar. The chemical is used as a solvent and paint thinner, and is found in the air after domestic paraffin use. Inhalation of 1,3,5 trimethylbenzene leads to depression of the central nervous system with symptoms such as headaches, nausea, dizziness, confusion and drowsiness being experienced (CCOHS, 2000). Skin irritation may also occur. Long-term exposure may increase the tendency to contract bronchitis and may cause blood clotting problems.

2.4.23 1,2,4 Trimethylbenzene

1,2,4 Trimethylbenzene is a colourless, flammable liquid that is an aromatic hydrocarbon. It is present in petroleum and petroleum products. The largest user of 1,2,4 trimethylbenzene is the chemical industry but it is also used as a dye carrier solvent and is a component of some paint solvents. Exposure to this substance can occur while using petrol or certain paints and cleaners or in the domestic environment where kerosene is used as a fuel for cooking and lighting. 1,2,4 Trimethylbenzene is a central nervous system depressant. The side effects of acute exposure to large amounts of this chemical include headaches, nausea, dizziness, drowsiness, confusion and even loss of consciousness. Irritation of the skin and mucous membranes can also occur (EPA, 1994; CCOHS, 2000).

2.4.24 1,2,3 Trimethylbenzene

The characteristics and effects of exposure to 1,2,3 trimethylbenzene are very similar to those of 1,3,5 and 1,2,4 trimethylbenzene (CCOHS, 2000).

2.4.25 Naphthalene

Naphthalene is a polycyclic aromatic hydrocarbon. It has a distinctive odour of mothballs. Naphthalene is used in the production of phthalic anhydride and is also an intermediate in the production of synthetic resins, celluloid, solvents and lubricants. Naphthalene was formerly used in the production of moth repellant, wood preservative, insecticide and certain pharmaceuticals. It is also a product of kerosene combustion (RAIS, 1998; CCOHS, 2000).

Naphthalene can be absorbed through inhalation and can cross the placenta in amounts that are sufficient to cause foetal toxicity. In addition, this chemical is a respiratory and skin irritant and can also affect the central nervous system. Acute exposure via inhalation causes haemolytic anemia (destruction of the red blood cells). Sub-chronic exposure via inhalation can lead to neurotoxic effects such as confusion and lethargy, gastrointestinal effects, hepatic and renal effects and ocular damage (cataracts have been reported) (RAIS, 1998; CCOHS, 2000). Naphthalene is believed to be a possible carcinogen (Oxford Chemistry Department, 2000).

2.4.26 2 Methylnaphthalene

2 Methylnaphthalene has been shown to cause pulmonary damage in rats and is thought to cause pulmonary damage after prolonged periods of exposure (CDC, 1993). Very little information could be found on the effects of this chemical.

2.4.27 1 Methylnaphthalene

1 Methylnaphthalene is used in insecticide manufacture and as a solvent. The chronic health effects due to inhalation of this substance are unclear, acute

health effects from exposure to the liquid include eye and skin irritation (CDC, 1993). Very little information could be found on the effects of this chemical.

CHAPTER THREE: METHODOLOGY AND DATA

3.1 Introduction

This dissertation provides an insight into domestic kerosene use in the city of Durban, with particular emphasis on the potential health effects likely to arise after exposure to kerosene combustion products. The study took place in the informal settlement of Cato Crest within the Greater Cato Manor area. The objective of this study was to investigate the application of the US EPA human health risk assessment framework in the South African context. The study consisted of two components of local data collection, namely a time-activity pattern questionnaire and an air quality study.

3.2 Time-Activity Pattern Questionnaire

The time-activity pattern survey was conducted through the Lead programme project entitled *Technologies for Enhanced Environmental Management in South Africa*. The study was originally meant to form a single component of a larger social study. During the course of the Lead project it was discovered that the social study could not be implemented at the time it was intended. It was then decided that this study (time-activity pattern questionnaires) should be conducted as a pilot study for the Lead project social study.

3.2.1 Area Identification

Cato Crest was identified as one of the informal areas within Cato Manor and was considered the most appropriate area in which to conduct the study due to the fairly high degree of organisation within this community. Previous studies such as Jones *et al.* (1996) also showed high kerosene usage in the area. Cato Crest is an extremely dense informal settlement that is divided into eight areas. Each of the building structures within the 8 Cato Crest areas is assigned a 'CC'

number. The 'CC' numbers also made it easier to locate households for follow-up visits during the air quality study. According to Mr. G. Mgenge (*pers. comm.*, June 2000) permission to conduct the study had been given by the Working Group reporting to the Metropolitan Mayor.

3.2.2 Questionnaire Design

The questionnaire consisted of 48 closed-ended questions divided into the categories of personal details, fuel use, building structure, cooking and time-activity patterns. The questionnaire was designed to be completed by the interviewer and was kept short in order to ensure that interviewees did not lose interest. The questionnaire is presented in Appendix 2.

3.2.3 Sampling method

As this study was investigating domestic kerosene use it was considered necessary to target women in the time-activity pattern questionnaire as they are generally the only household members involved in cooking and other domestic activities. A few men were also interviewed in order to verify this assumption. A multistage sampling method was used where the initial population (Cato Crest) was clustered (each of the eight areas of Cato Crest, as shown in Appendix 3, became a cluster) after which random sampling took place within each cluster (University of Oregon, 1999). The implementation of this sampling method is described in more detail below in Section 3.2.4. A sample size of 50 was chosen due to financial constraints and the fact that the exercise was being conducted as a pilot project for the Lead programme project. Although the sample represents a small percentage of the total population of Cato Crest (less than 0.5%), this study was not so concerned with the statistical significance of the results as with gaining an understanding of the time-activity patterns in the area. This would allow the author to indicate whether the time-activity patterns differ from those of the US EPA or not.

3.2.4 Implementation

The questionnaires were administered by a group of ten students from the Universities of Natal (UND) and Durban-Westville (UDW). The students attended a training session where they were guided through the questionnaire and any problems resolved.

On 28 June 2000, after a final briefing session, a vehicle was taken into Cato Crest to scout the area for potential transect routes and meeting points. Mr. Goodman Mgenge (Durban City Health Department) met the team in Cato Crest and gave them a guided tour of the area. The boundaries of the eight areas were delineated on an aerial photograph of Cato Crest (see Appendix 3). Meeting points were determined where there would be easy vehicular access for conducting the study. Transects through each of the eight areas were marked on the map ensuring a fairly even allocation of households between areas, and allowing for easy access at the end of a transect route in order to collect team members. The sampling along each transect route was random, with most houses being chosen simply if a main member of the household was at home and willing to answer the questionnaire.

The team members were paired into 'interviewer and observer' teams as outlined below:

Thursday 29 June 2000
Team A: Welcome and Ruth
Team B: Themba and Vincent
Team C: Philip and Phumla
Team D: Sizwe and Mamopeli

Friday 30 June 2000
Team E: Philip and Bongi
Team F: Themba and Futhi
Team G: Vincent and Phumla
Team H: Sizwe and Welcome

On the first day of the study Teams A and B were required to sample area 2 of Cato Crest along a transect from point X1 towards Bellair Road (see Appendix 3). Teams C and D were required to visit area 4, sampling from point X2

northwards towards Bellair Road. These areas are the two largest areas within Cato Crest, requiring a larger number of households to be included in the sample (24 households in total were sampled for the first day). On the second day of the study Team E was required to visit areas 7 and 8 (relatively small geographical areas) walking southwards from point X3 and then west towards Bellair Road. Team F was required to visit area 6, walking north-west from point X3 towards Bellair Road. Team G visited area 5 (walking from point X4 towards Bellair Road) and Team H visited areas 1 and 3 (walking parallel to Bellair Road in an easterly direction). A total of 45 households were visited on the second day of the study.

A cumulative total of 69 questionnaires were completed during the study. This was more than the original sample size in order to allow elimination of incomplete questionnaires where necessary without affecting the sample size. Sixty six of the questionnaires were completed by women and only three were completed by men.

Data from the questionnaires were entered into an Excel spreadsheet and subsequently analysed. Twenty households were identified for the indoor air quality study as outlined in Section 3.3.1.

3.2.5 Limitations

Several limitations were obviously apparent during the implementation of the time-activity pattern questionnaire. The list below provides an indication of these limitations.

- The author was unable to take part in the implementation of the time-activity pattern study due to racial tension in the study area. On the recommendation of the Environmental Health Officer only black research workers were advised to enter the area. The author was therefore dependent on the results gathered by others. She did, however, play a role in briefing sessions at which the team was prepared for the study.

- Some misinterpretation may have occurred between the study team reading the interview questions and the community members although this is unlikely as interviews were conducted in the first language of the interviewees.
- Questionnaires were administered in the language respondents felt most comfortable with, and were completed by the students in English. A small amount of insight may have been lost when the English questions were translated into Zulu, and then Zulu answers back into English.
- Bias may have occurred when houses were chosen for interviews. Male team members were unable to interview female respondents if the respondent was not already outside their home.
- The haphazard nature of the Cato Crest settlement (and the lack of a housing plan) made it impossible to randomly choose from an aerial photograph which houses to conduct interviews in. Team members therefore had to chose households while walking along the transect, often selecting those households where a main member of the household was already outside doing household chores.
- The time-activity pattern sample is not a valid statistical sampling of the total population. This was not a concern as a statistical sample was not required, but rather an indication of the time-activity patterns of several households in a typical informal settlement in South Africa.

Some of the results of the questionnaire can be found in Appendix 4.

3.3 Air Quality Study

Indoor air quality has been shown to have a great impact on human health with many studies proving the relationship between pollutants and certain diseases (Gammage and Kaye, 1987). It is often in poor households where the diseases caused by indoor air pollution can be ill-afforded and are exacerbated by nutritional state and existing diseases. This air quality study was undertaken to

assess whether in fact the use of kerosene as a domestic fuel was causing poor health in the community of Cato Manor, Durban.

3.3.1 Household Identification

The sample size for the air quality study was limited by the availability of funds and to a lesser extent, time. Households for which questionnaires appeared to be most complete were chosen, resulting in a total of 20 households. Only 14 of the original 20 households participated in the air quality study

The households chosen for the air quality study were as follows:

CC 0536	Area 4
CC 2316	Area 1
CC 4038	Area 8
CC 2909	Area 1
CC 0548	Area unknown
CC 2662	Area 3
CC 0834	Area 5
CC 1843	Area 2
CC 0823	Area unknown
CC 1839	Area 2
CC 1230	Area unknown
CC 0797	Area 5
CC 1635	Area 2
CC 0715	Area 5

3.3.2 Study implementation

Within each of the houses listed above, air quality monitoring was performed for both nitrogen dioxide (NO_2) and volatile organic compounds (VOCs). Nitrogen dioxide was measured using ChromAir© Direct Read Passive Monitor badges and total VOCs were measured using Traceair© Organic Vapour Monitor (OVM-1) badges (K&M Environmental, 1997).

The NO_2 ChromAir© Direct Read Passive Monitor 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 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 on the indicator layer 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 nitrogen dioxide badges are able to detect a range of 0.5 to 13 ppm per hour (930 to 24 250 $\mu g/m^3$ per hour), with a minimum detectable concentration in 8 hours of 0.06 ppm (110 $\mu g/m^3$). They are able to function effectively in a relative humidity range of 15% to 90% and a temperature range of 10°C to 40°C. The ability of the badge to measure NO_2 is interfered with by the presence of ozone. The minimum sampling time that the badges can be used for is 15 minutes and the maximum is 2 days (K&M Environmental, 1997).

The Traceair© Organic Vapour Monitor (OVM-1) badges were originally developed by DuPont and are simple and economical diffusive samplers used for determining concentrations of organic contaminants. They can be used to sample for hundreds of organic solvents. The OVM eliminates the need for expensive air sampling pumps and the maintenance associated with them. The OVM-1 badge contains a 300mg coconut-based charcoal strip for the compounds to adhere to. The badges can operate in a temperature range of 10°C to 50°C and in a relative humidity of 10% to 80% (K&M Environmental, 1997).

The air quality monitoring study took place over a 9 day period from 5 to 15 September 2000. NO_2 and VOC badges were placed in each house on 5 September, the badges were hung from the ceiling near to where the cooking

was being done but not directly over the stove. The NO_2 badges were replaced each day while the VOC badges were left for the entire 9 day period. After the replacement of each NO_2 badge the results were read off a colour comparison chart and recorded. The VOC badges were sent to the GC/MS Laboratory (Fire and Engineering Services, CSIR Building and Construction Technology Division), where the aromatic compounds were individually quantified using the NIOSH 1500/1501 analysis method. In this method the total VOC samples collected using coconut shell charcoal sorbent tubes are analysed by gas chromatography with flame ionization detection. These methods are evaluated and validated for NIOSH accuracy of 25% at the 95% confidence level. However, many of the methods have an accuracy greater than 25%, such as toluene which has an accuracy of 7% (CDC, 1994). Benzene and toluene were chosen to use in the health risk assessments as they have been subject to fairly comprehensive research to date and also had readily available dose-response values.

3.3.3 Limitations

There were several limitations to the air quality study. The list below provides an indication of these:

- Severe restrictions in the budget available for the study meant that not every house involved in the time-activity pattern questionnaire could be involved in the air quality study, as originally intended.
- Homeowners were not always at home when technicians arrived to replace the NO_2 monitors each day. Some NO_2 monitors were therefore left out for 48 hour periods. This may have resulted in saturation of the monitors although this is unlikely as the monitors can be left out for a maximum period of 2 days.
- The air quality monitoring study was based on a 9-day sample in September. The results are not representative of conditions throughout the year and can be regarded as fairly conservative. Higher values could be expected in winter when climatic conditions predispose the area to pollution accumulation.

3.4 Quantitative Human Health Risk Assessment

The health risk assessments in this study have been conducted according to the US EPA method as outlined above in Chapter 2. The methodology for the risk assessments is outlined below according to the four steps of a health risk assessment.

3.4.1 Hazard Identification

A hazard is described by van Leeuwen and Hermens (1995) as the potential of a chemical or mixture of chemicals to cause adverse human health effects at a particular exposure level. Hazard identification is therefore the process of recognising which chemicals or mixture of chemicals in the environment is likely to be responsible for adverse human health effects should exposure to that chemical or mixture of chemicals occur. Hazard identification also involves identifying which adverse human health effects are likely to occur following both acute and chronic exposure to the hazardous substances (Paustenbach, 1989; van Leeuwen and Hermens, 1995).

The process of hazard identification relied on the use of literature such as medical and environmental health journals, books, previous environmental health studies and the internet. The use of kerosene appliances in the informal settlement of Cato Manor, Durban was identified as a hazardous activity with the effect of the inhaled combustion products being the hazard. Various literature sources were again used to ascertain the health effects related to the inhalation of kerosene combustion products

3.4.2 Exposure Assessment

In the exposure assessment the following were identified or calculated:

- emissions and pathways,

- estimated concentrations (or doses) of the hazardous substances that the target population is exposed to,
- target population exposed to the hazardous substances, and the target organs in the body which are affected by exposure to the hazardous substances,
- magnitude, frequency and duration of exposure of the target population, as well as behaviour patterns, geographic distribution and population size of the target population, and
- estimated dose for carcinogenic and non-carcinogenic substances (Paustenbach, 1989; van Leeuwen and Hermens, 1995).

In the exposure assessment step of the health risk assessment, the estimated dose received is calculated for both carcinogenic and non-carcinogenic substances. For carcinogenic substances the Lifetime Average Daily Dose or LADD is determined for both oral and dermal routes of exposure, whereas for non-carcinogenic substances the Average Daily Dose (ADD) or Average Hourly Dose (AHD) is determined. The calculation of exposure can be achieved in a very simple way using an equation for a single medium, single pathway exposure assessment. Alternatively, for multimedia, multiple pathway exposures a software model can be used. For the purposes of this study (which addresses a single pathway exposure) a simple equation was used.

The equation that is used to determine the Lifetime Average Daily Dose for an individual exposed to carcinogenic compounds is taken from the Exposure Factors Handbook (US EPA, 1996) and is shown below.

$$LADD = \frac{C \cdot IR \cdot ED}{BW \cdot AT} \qquad (3.1)$$

where LADD = Lifetime Average Daily Dose for inhalation ($\mu g/kg/day$)

C = pollutant concentration in the air ($\mu g/m^3$)

IR = inhalation rate (m^3/day)
ED = exposure duration (days)
BW = body weight (kg), and
AT = averaging time (days).

The units for this equation are determined as follows:

$$\mu g/kg/d = \frac{\mu g/m^3 \times m^3/d \times d/1}{kg/1 \times d/1} \quad (3.2)$$

The LADD is then used to determine the Cancer Risk as outlined in Section 3.4.4.

The calculation of chronic (non-cancer) health effects used the following equation to determine the Average Daily Dose or ADD (US EPA, 1996).

$$ADD = \frac{C \,.\, IR \,.\, ED}{BW \,.\, AT} \quad (3.3)$$

where ADD = Average Daily Dose for inhalation (μg/kg/day)
C = concentration of the chemical (μg/m^3)
IR = inhalation rate (m^3/day)
ED = exposure duration (days)
BW = body weight (kg), and
AT = averaging time period (days).

The Exposure Duration is based on total exposure time and is expressed as the number of hours exposure per day multiplied by the number of days exposed per year multiplied by the number of years of exposure to the chemical. The units for this equation are calculated in the same manner as Equation 3.2. The ADD is then used to determine the Hazard Quotient for chronic exposure as outlined in Section 3.4.4.

The calculation of acute (non-cancer) health effects required the use of the following equation to determine the average hourly dose or AHD (Louvar and Louvar, 1998).

$$AHD = \frac{C \cdot IR}{BW} \qquad (3.4)$$

where AHD = Average Hourly Dose for inhalation (μg/kg/hr)

C = concentration of the chemical (μg/m^3)

IR = inhalation rate (m^3/hr), and

BW = body weight (kg).

The exposure duration and averaging time are omitted from the equation as they are both 1-hour for an acute exposure. The units for this equation are calculated in the same manner as Equation 3.2 with the substitution of 'hours' instead of 'days' into the inhalation rate. The AHD is then used to determine the Hazard Quotient for acute exposure as outlined in Section 3.4.4.

3.4.3 Dose-response Assessment

The dose-response assessment is the process of identifying the relationship between the exposure level or dose and the severity of the health effects likely to be experienced. This analysis is based on quantitative studies such as epidemiological studies or experimental laboratory or field studies on animals and/or humans. Dose-response relationships often indicate the presence of different toxic effects of a substance at different concentrations. Dose-response is also affected by population characteristics such as age, gender, lifestyle, occupation and existing diseases, among other things (Paustenbach, 1989; van Leeuwen and Hermens, 1995).

Dose-response information for each of the pollutants identified in the first step of the health risk assessment (hazard identification) were found through an

extensive search conducted on all known, reliable databases such as the US EPA Integrated Risk Information System or IRIS, the ATSDR Minimal Risk Levels for Hazardous Substances and the Californian EPA Office of Environmental Health Hazard Assessment (OEHHA) Reference Exposure Levels. The dose-response value can also be termed the critical level.

3.4.4 Risk Characterisation

In the risk characterisation step both cancer and non-cancer health effects are estimated. Non-carcinogenic health effects are expressed as a hazard quotient (HQ) which indicates the presence or absence of adverse health effects due to exposure. The HQ can also provide an indication of whether only sensitive individuals will be affected, or if both healthy and sensitive individuals will be affected. Both acute and chronic health effects can be assessed.

Calculation of chronic health effects

The Average Daily Dose (obtained in the exposure assessment) is divided by the relevant dose-response value obtained from the dose-response assessment (such as RfC, MRL or REL) to determine the hazard quotient or HQ (NRC, 1994).

$$HQ = \frac{ADD}{DRV} \tag{3.5}$$

where HQ = hazard quotient
ADD = average daily dose, and
DRV = dose-response value (such as RfC, MRL or REL).

Calculation of acute health effects

Following the calculation of the Average Daily Dose or ADD, the Hazard Quotient (HQ) for acute exposure to chemical pollutants is determined through the following equation (NRC, 1994).

$$HQ = \frac{ADD}{DRV} \quad (3.6)$$

where HQ = hazard quotient
ADD = average daily dose, and
DRV = dose-response value (such as RfC, MRL or REL).

This equation is, in essence, the exposure assessment value divided by the dose-response value.

Calculation of cancer risks

The cancer risk is calculated in a similar manner as the non-cancer hazards. The Lifetime Average Daily Dose (LADD) is multiplied by the Inhalation Unit Risk (or another similar value) in order to obtain the cancer risk.

$$\text{Cancer Risk} = \text{LADD} \cdot \text{IUR} \quad (3.7)$$

where LADD = Lifetime Average Daily Dose
IUR = Inhalation Unit Risk (US EPA, 1998).

The cancer risk is defined as the incremental probability that an individual will develop cancer over a lifetime as a result of an exposure to a carcinogen (US EPA, 1998). The cancer risk is expressed, for example, as a 1 in a million chance of developing a specific cancer due to long-term exposure to a specific carcinogen.

3.4.5 Limitations

This study did not address adverse human health effects that might arise from:

- The possible synergistic effects between each of the combustion products being studied as well as with other pollutants present in the atmosphere.
- The long-range transport of pollutants into and out of the study area.

- Occupational exposure of any individual in the study area to these and other pollutants.
- The health effects arising from dermal contact or ingestion of kerosene or pollutants of kerosene combustion.
- The carcinogenic health effects of benzene due to a lack of annual indoor benzene concentrations.

CHAPTER FOUR: RESULTS AND DISCUSSION

4.1 Introduction

The results of this study are presented in three sections according to the 3 species of chemicals considered in the air quality study. Firstly, the results of the nitrogen dioxide monitoring are presented in a health risk assessment with a discussion of the results. Secondly, the benzene results are presented, also in a health risk assessment. A cancer health risk assessment was not calculated for benzene as the air quality results were conducted over an inadequate averaging period. Thirdly, the toluene health risk assessment results are presented and discussed.

The results of the time-activity pattern revealed that 87% of the sample group relies on kerosene for cooking (60 out of 69 households) while less than 30% rely on kerosene for heating and lighting purposes (22 and 20 households out of 69 respectively) (see Appendix 4). The results clearly show that kerosene fuel use is fairly widespread throughout Cato Crest and has the potential to cause adverse health effects through inhalation of the combustion products. The following sections will discuss the health effects related to nitrogen dioxide, benzene and toluene concentrations found in certain Cato Crest homes.

4.2 Nitrogen Dioxide

4.2.1 Hazard Identification

The process of hazard identification in this study revealed that there are a wide range of pollutants released during domestic combustion of kerosene in Cato Manor, Durban. One of these pollutants is nitrogen dioxide. Nitrogen dioxide is

known to affect both the respiratory system and the immune system (WHO, 1997; Californian EPA, 1999*a*).

Acute exposure to nitrogen dioxide is associated with respiratory irritation and can lead to long-term changes in the respiratory system such as pulmonary oedema, pneumonitis, bronchitis and bronchiolitis obliterans (Californian EPA, 1999*a*). Acute health effects may or may not occur within 1 or 2 hours of exposure. Clinical effects include a shallow respiratory rate, excessively rapid heart beat, wheezing and cyanosis. Shortness of breath and coughing may occur and can last from 2 to 3 weeks (TOXNET, 2000). NO_2 toxicity can be enhanced if certain medical or chemical conditions are present. Medical conditions such as asthma or other pulmonary diseases would predispose an individual to be more sensitive to the effects of nitrogen dioxide exposure.

For chronic exposure to nitrogen dioxide the health endpoint of concern is again the respiratory and immune systems. Chronic exposure to NO_2 causes increased pulmonary sensitivity, increased airway resistance and increased susceptibility to respiratory infections. Those individuals who are sensitive to these effects include children, asthmatics, and individuals with chronic obstructive pulmonary disease (COPD) (Californian EPA, 1997).

Recent studies are showing evidence that chronic exposure to NO_2 can cause a decline in immunological function. The inflammatory response often associated with exposure to pollutants suggests that immune function is altered in various ways. Sandstrom *et al.* (1992*a*) have shown that exposure of healthy adult males to various concentrations of nitrogen dioxide causes reductions in certain immune components. The cytotoxic T lymphocyte population was significantly reduced following NO_2 exposure, as was the natural killer cell population. At higher concentrations of NO_2 the alveolar macrophage count and B lymphocyte count was also reduced. The reduction in these specific components of the immune system could cause more rapid onset of diseases such as HIV/AIDS and

increased susceptibility to viral and bacterial infection (Goings *et al*, 1989; Rosok, *et al.*, 1997; WHO, 1997; Becker and Soukup, 1999).

4.2.2 Exposure Assessment

The exposure parameters used for the calculation of the Average Hourly Dose (AHD) and Average Daily Dose (ADD) are outlined in Table 4.1. These exposure parameters were substituted into the equations shown in Section 3.4.2 in order to determine the ADD (or AHD).

The scenarios presented in Table 4.1 are as follows:

- A 1-hour indoor exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year;
- A 24-hour exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year, using US EPA default exposure values (US EPA, 1996);
- A 24-hour exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year, using local exposure values.

Table 4.1: Exposure Parameters and Exposure Assessment Results

Indoor 1-Hour concentration	Inhalation Rate (m^3/hour)	US EPA Exposure Duration (hrs)	Body Weight (kg)	Averaging Time (hrs)
Adult	1.2	1	71.8	1
Child	1.2	1	36.3	1
Infant	0.3	1	11.3	1
Indoor 24-Hour concentration, EPA values	**Inhalation Rate (m^3/day)**	**US EPA Exposure Duration (days)**	**Body Weight (kg)**	**Averaging Time (days)**
Adult	11.3	(7h/d) (350d/y) (30y)	71.8	(30y) (365d)
Child	8.7	(2h/d) (350d/y) (10y)	36.3	(10y) (365d)
Infant	4.5	(2h/d) (350d/y) (1y)	11.3	(1y) (365d)
Indoor 24-Hour concentration, Local values	**Inhalation Rate (m^3/day)**	**Local Exposure Duration (days)**	**Body Weight (kg)**	**Averaging Time (days)**
Adult	11.3	(**14**h/d) (350d/y) (30y)	71.8	(30y) (365d)
Child	8.7	(**12**h/d) (350d/y) (10y)	36.3	(10y) (365d)
Infant	4.5	(**14**h/d) (350d/y) (1y)	11.3	(1y) (365d)

Inhalation rate, body weight and US EPA exposure duration taken from Exposure Factors Handbook (US EPA, 1996). For an explanation of the 'Exposure Duration' see Equation 3.3.

The US EPA default exposure values were taken to be 7 hours per day for an adult and 2 hours per day for a child and infant. The adult was assumed to cook for 1 hour per day, to be in the kitchen for 1 hour per day (not cooking), to perform chores in the kitchen for 2 hours per day, and to be present in the kitchen either eating or entertaining for 3 hours per day. The child was assumed to spend 2 hours per day in the kitchen either eating or performing chores. The infant was assumed to spend the majority of the day in a nursery or room other than the kitchen, therefore the exposure time was set to 2 hours (US EPA, 1996).

The time-activity pattern questionnaire revealed that many of the houses in Cato Crest consist of a single room (Question 21, Appendix 2) in which cooking and all other activities take place. In addition, the room functions as a bedroom (Question 39, Appendix 4) with 60% of households having between one and

three people sleeping in the same room that meals are cooked in. With this in mind, the local exposure time was set to 14 hours exposure for both adult and infant. The 14 hour period is taken as the total time spent indoors where *exposure* to kerosene fumes from cooking would take place although the cooking process itself is not carried out for 14 hours (see Questions 47 and 48 in Appendix 4). This 14 hour period therefore includes the time spent sleeping (averaging between 9 and 10 hours) as the houses consist mainly of a single room. The exposure time for the infant was taken to be the same as that of the adult, based on the assumption that the infant would be carried on the mother's back when she went outside the house to do other chores or to socialise. The exposure time for children was taken as 12 hours, assuming a child would spend a portion of the late afternoon playing outside after school but would still eat and sleep in the same room as other members of the family. Household chores were also included in the time spent indoors. The assumption for both these scenarios is that the mother cooks and does chores indoors for approximately 4 hours per day (see Questions 47 and 48 in Appendix 4). The estimated exposure durations presented here are based on data collected in the time-activity pattern questionnaire and shown in Appendix 4.

The Cato Crest study also showed that the average age of respondents was 34 years old and the average weight was 68kg. The average age and weight of the respondents was so similar to the default US EPA values that a decision was taken to leave the age and weight for the local adult scenario as the US EPA default values in order to assess more clearly the effect of exposure time on the hazard quotient.

The Average Hourly Dose and Average Daily Dose have been calculated for each house in which nitrogen dioxide was monitored. The AHD and ADD for each house are shown in Tables 4.2 to 4.4. The AHD is expressed in μg/kg/hr which can be interpreted as μg pollutant per kg of body weight per hour. The

ADD is expressed in μg/kg/day which is interpreted as μg pollutant per kg of body weight per day.

Table 4.2: Average Hourly Dose (μg/kg/hr) for 1-Hour Nitrogen Dioxide Exposure

	CC0536	CC4038	CC0548	CC0834	CC0823	CC1230	CC1635
Adult	1.7	0.9	1.0	No results	0.9	1.1	1.2
Child	3.4	1.8	2.0	No results	1.7	2.2	2.3
Infant	2.8	1.5	1.6	No results	1.4	1.8	1.9
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	1.0	0.9	1.6	0.9	1.0	1.1	1.1
Child	2.0	1.9	3.1	1.9	2.0	2.2	2.1
Infant	1.6	1.5	2.5	1.5	1.6	1.8	1.7

Table 4.3: Average Daily Dose (μg/kg/day) for 24-Hour Nitrogen Dioxide Exposure using EPA Exposure Values

	CC0536	CC4038	CC0548	CC0834	CC0823	CC1230	CC1635
Adult	109.8	58.6	64.3	No results	54.7	71.4	73.8
Child	47.8	25.5	28.0	No results	23.8	31.1	32.1
Infant	79.4	42.4	46.5	No results	39.6	51.7	53.3
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	63.8	61.2	98.5	59.1	64.6	71.2	67.6
Child	27.8	26.6	42.9	25.7	28.1	31.0	29.4
Infant	46.1	44.2	71.2	42.7	46.7	51.5	48.9

Table 4.4: Average Daily Dose (μg/kg/day) for 24-Hour Nitrogen Dioxide Exposure using Local Exposure Values

	CC0536	CC4038	CC0548	CC0834	CC0823	CC1230	CC1635
Adult	231.8	123.7	135.8	No results	115.6	150.9	155.8
Child	286.6	152.9	167.9	No results	142.9	186.5	192.6
Infant	555.5	296.5	325.5	No results	277.1	361.6	373.3
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	134.7	129.2	208.1	124.9	136.4	150.3	142.8
Child	166.5	159.7	257.3	154.4	168.6	185.8	176.5
Infant	322.8	309.6	498.7	299.2	326.9	360.2	342.2

4.2.3 Dose-response Assessment

For this health risk assessment, the health risk of exposure to nitrogen dioxide was determined for a 1-hour and a 24-hour exposure period. The Reference Exposure Levels (RELs) that were used in these health risk assessments were as follows:

- 1-Hour REL : 470 $\mu g/m^3$
- 24-Hour REL : 100 $\mu g/m^3$

The 1-hour REL was obtained from the Californian EPA (1999*a*) while the 24-hour REL was obtained from the New Zealand Ministry for the Environment (1994). Other countries with a 24-hour nitrogen dioxide guideline level are South Africa (187 $\mu g/m^3$) and Finland (70 $\mu g/m^3$) (DEAT, no date; Hamekoski, 1998). The South African guideline could not be used as it has not been set for a health endpoint. Very little literature could be found on the Finnish guideline.

The uncertainty factor for the 1-hour 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. The uncertainty factor for the 24-hour 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 (NZ Ministry for the Environment, 1994).

Nitrogen dioxide is an irritant to the lungs and at very high concentrations can produce pulmonary oedema and even death. After inhalation, the NO_2 is deposited along the respiratory tract, specifically the lower respiratory tract where it affects firstly the bronchioles and then the alveolar ducts and alveoli. Exercise increases the inhaled quantity of NO_2 and alters the distribution of deposited NO_2 in the lung, where more NO_2 is deposited in the lower lung during exercise than during rest. Nitrogen dioxide is also a strong oxidant which is able to oxidise unsaturated lipids and membrane proteins. The oxidation of membrane proteins

at the site of NO_2 deposition in the respiratory tract results in a loss of control of cell permeability leading to the development of pulmonary oedema. The oxidative action of NO_2 on lipids can be counteracted by the presence of both Vitamin C (ascorbic acid) and Vitamin E (α-tocopherol) (WHO, 1997).

The respiratory health effects of nitrogen dioxide exposure have been extensively studied and are well documented in the literature. Both acute and chronic exposure to nitrogen dioxide affects the respiratory system. Acute exposure appears to cause pulmonary oedema, pneumonitis, bronchitis and bronchiolitis obliterans (Californian EPA, 1999). Exposure to NO_2 at concentrations between 190 000 and 900 000 $\mu g/m^3$ (100 to 500 ppm) can lead to sudden death. Studies done on healthy human subjects at various NO_2 concentrations reveal different levels at which responses occur. Healthy adults do not usually respond to nitrogen dioxide concentrations below 1880 $\mu g/m^3$ (or 1 ppm), however, it has been noted that there is no consistent pattern of response to acute exposure (Californian EPA, 1999).

Asthmatic individuals are generally more responsive to the effects of NO_2 exposure, although responses vary widely in severity. Typical responses of asthmatics (at levels above 380 $\mu g/m^3$) include increased airway resistance and decreased forced expiratory volume (FEV). The total lung capacity is often decreased, and airway responsiveness to broncho-constrictors can be increased.

Individuals with chronic obstructive pulmonary disease (COPD) represent a potentially sensitive sub-population. Certain studies show significant decreases in FEV (at 564 $\mu g/m^3$ or 0.3 ppm), with the magnitude of the response being unaffected by the severity of the disease. However, individuals with COPD have relatively inelastic lungs which would limit the ability to respond to NO_2 exposure.

Other individuals in a population may be predisposed to nitrogen dioxide toxicity due to several factors. One such factor is the presence of high nitrate levels in

drinking water, which increases the likelihood of development of methaemoglobinemia (WHO, 1997; Californian EPA, 1999). As mentioned earlier, studies done by Postlethwait and Mustafa (1981) showed the metabolic products of inhaled NO_2 in rat lungs to be NO_2^- and NO_3^- (once in contact with the blood). This increase in NO_2^- levels after NO_2 exposure could cause increased incidence of Blue Baby Syndrome due to raised methaemoglobin levels in the blood (Colvin and Genthe, undated).

Individuals sensitive to the effects of chronic NO_2 exposure are the asthmatic and the individual with COPD. Chronic exposure to NO_2 is reported to enhance sensitivity to broncho-constrictors, increase airway resistance, and increase the likelihood of acquiring respiratory infection (Californian EPA, 1997). Personal monitoring studies with children have shown that chronic exposure to ambient NO_2 at a level of 28.2 $\mu g/m^3$ (0.015 ppm) is significantly associated with asthma prevalence (Infante-Rivard, 1993 in Californian EPA, 1997). Correlations between ambient nitrogen dioxide concentration and alterations in lung function in children have also been shown (Californian EPA, 1997).

Animal studies have shown that exposure to nitrogen dioxide can cause a modification of the lung structure (a change in cells lining the affected regions, or tissue thickening) and function, changes in the biochemistry and metabolism of the lung, emphysema (of the same type found in humans), increased bacterial and viral susceptibility (at 9400 $\mu g/m^3$ or 5 ppm), and strong oxidative effects on unsaturated lipids (at 75 $\mu g/m^3$ or 0.04 ppm), proteins and enzyme pathways (at 1880 $\mu g/m^3$ or 1 ppm) (Ospital *et al.*, 1981; WHO, 1997).

Humoral and cell-mediated immune responses are also altered by chronic exposure to NO_2 (Goings *et al.*, 1989; Sandstrom *et al.*, 1992*a* and 1992*b*; WHO, 1997; Becker and Soukup, 1999). Animal studies have shown that host defence mechanisms of the respiratory tract can be affected in the following ways:

- cilia and ciliated epithelial cells lose certain functions at high levels of NO_2

(>9400 $\mu g/m^3$ or 5 ppm),

- alveolar macrophages (responsible for removing inhaled particles, dead cells etc by phagocytosis, among other immunological functions) undergo structural, functional and biochemical changes at 3760 $\mu g/m^3$ or 2 ppm,
- various humoral and cell-mediated immune changes (for example, changes in lymphocyte populations and immunoglobulin levels), and
- increased susceptibility to both bacterial and viral infections (WHO, 1997).

Human exposure studies on the effects of nitrogen dioxide on the immune system show similar results. Using a method known as bronchoalveolar lavage, or BAL (washing of the bronchoalveolar region with saline solution), the components of the immune system present in the affected areas of the respiratory tract can be determined. Sandstrom *et al.* (1992*a* and 1992*b*) performed sub-chronic NO_2 exposure studies on healthy adult males to assess changes in lymphocyte populations. A significant reduction in the CD8+ cytotoxic T lymphocytes was observed in the BAL fluid. This reduction in cytotoxic T lymphocytes caused an increase in the ratio of CD4+ helper T lymphocytes : CD8+ cytotoxic T lymphocytes. The natural killer cell (NK) count was also significantly reduced. Changes in the B lymphocyte population could not be detected. The CD8+ cytotoxic T lymphocytes are responsible for containment of HIV retrovirus replication (Kundu and Merigan, 1991; Hsueh *et al.*, 1994; Rosok, *et al.*, 1997; Stranford, *et al.*, 1999). A reduction in the CD8+ cytotoxic T lymphocyte population could therefore possibly result in an earlier onset of conversion to full-blown Acquired Immune Deficiency Syndrome or AIDS (Famularo, *et al.*, 1997).

4.2.4 Risk Characterisation

The risk calculation results for both the acute and chronic risks were obtained by dividing the average daily dose (ADD) by the Reference Exposure Level (REL) (see Section 3.4.4). The risk calculation is then expressed as a hazard quotient (HQ) which indicates the presence or absence of adverse health effects due to

exposure. A hazard quotient that is less than 1 indicates a negligible risk, even to a sensitive individual, while a hazard quotient greater than 1 indicates that there will be some risk as a result of exposure. A basic rule can be followed, whereby, the greater the hazard quotient the greater the risk.

The monitored 1-hour nitrogen dioxide concentrations shown in Appendix 5 ranged from 51.8 $\mu g/m^3$ at CC 0823 to 103.9 $\mu g/m^3$ at CC 0536. The Californian EPA 1-hour NO_2 guideline value of 470 $\mu g/m^3$ was not exceeded at any of the houses. The hazard quotients calculated for each house in this scenario (shown in Table 4.5) showed no likelihood of adverse health effects occurring at this level of exposure for an adult, child or infant.

Table 4.5: Hazard Quotients for 1-Hour Nitrogen Dioxide Exposure

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	0.004	0.002	0.002	No results	0.002	0.002	0.002
Child	0.007	0.004	0.004	No results	0.004	0.005	0.005
Infant	0.006	0.003	0.003	No results	0.003	0.004	0.004
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	0.002	0.002	0.003	0.002	0.002	0.002	0.002
Child	0.004	0.004	0.007	0.004	0.004	0.005	0.005
Infant	0.003	0.003	0.005	0.003	0.003	0.004	0.004

The monitored 24-hour nitrogen dioxide concentrations shown in Appendix 5 ranged from 1243.8 $\mu g/m^3$ at CC 0823 to 2493.8 $\mu g/m^3$ at CC 0536. The New Zealand 24-hour NO_2 Reference Exposure Level of 100 $\mu g/m^3$ was exceeded at each of the houses and most of the hazard quotients calculated for the 24-hour exposure scenario using US EPA exposure values (shown in Table 4.6) showed no likelihood of adverse health effects occurring at this level of exposure for an adult, child or infant. However, CC 0536 had an HQ of 1.1 for an adult and CC 2662 had an HQ of 1.0 for an adult. These were the only two houses showing 24-hour nitrogen dioxide concentrations over 2000 $\mu g/m^3$. Both houses used paraffin for cooking and candles for lighting. The houses were made of metal sheeting,

wood and plastic. CC 0563 is a one-roomed house with a single door and window, and is home to seven people. All the cooking is done indoors. CC 2662 is a three-roomed house with no windows in any of the rooms and three doors. Four people live in CC 2662 with only one person sleeping in the room where the cooking is done. Confounding factors such as smoking were considered and it was noted that none of the occupants of the two dwellings were smokers. The ventilation in these two homes is likely to be very poor due to the lack of windows.

Sensitive adults living in either of these two houses are likely to experience adverse health effects associated with exposure to nitrogen dioxide. Adults who are likely to be affected include those with asthma and other respiratory diseases. The infants and children showed HQ's of less than 1 for these two houses as the time they were exposed to the nitrogen dioxide was set to 2 hours whereas the adult exposure was for 7 hours. This is based on the assumption that infants would spend most of the day sleeping in a nursery or in a room other than the kitchen.

Table 4.6: Hazard Quotients for 24-Hour Nitrogen Dioxide Exposure using EPA Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	1.1	0.6	0.6	No results	0.5	0.7	0.7
Child	0.5	0.3	0.3	No results	0.2	0.3	0.3
Infant	0.8	0.4	0.5	No results	0.4	0.5	0.5
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	0.6	0.6	1.0	0.6	0.6	0.7	0.7
Child	0.3	0.3	0.4	0.3	0.3	0.3	0.3
Infant	0.5	0.4	0.7	0.4	0.5	0.5	0.5

The next scenario is based on the same 24-hour nitrogen dioxide values as shown in the previous scenario, however, the exposure assessment has been determined according to local exposure assessment values collected from the

time-activity pattern questionnaire. All the hazard quotients (presented in Table 4.7) calculated for this scenario were above 1, with the minimum HQ being 1.2 and the maximum being 5.6 (for an infant in CC 0536, the house with the highest 24-hour NO_2 concentration). CC 0536 appears to have very low ventilation as it is a one-roomed house with one window and one door. Assuming the local exposure times are correct, it would appear that nitrogen dioxide fumes from kerosene combustion will very likely cause adverse respiratory effects in individuals living in these houses. These adverse health effects include coughing, wheezing, chest tightness, broncho-constriction and increased airway resistance. Not only sensitive individuals, but also some healthy individuals will begin to experience adverse health effects.

Table 4.7: Hazard Quotients for 24-Hour Nitrogen Dioxide Exposure using Local Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	2.3	1.2	1.4	No results	1.2	1.5	1.6
Child	2.9	1.5	1.7	No results	1.4	1.9	1.9
Infant	5.6	3.0	3.3	No results	2.8	3.6	3.7
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	1.3	1.3	2.1	1.2	1.4	1.5	1.4
Child	1.7	1.6	2.6	1.5	1.7	1.9	1.8
Infant	3.2	3.1	5.0	3.0	3.3	3.6	3.4

4.3 Benzene

4.3.1 Hazard Identification

The process of hazard identification revealed that in addition to nitrogen dioxide being a pollutant released during kerosene combustion, there is a whole range of other pollutants such as volatile organic compounds (VOCs) that are also released. Benzene is one of these VOCs that is released during kerosene

combustion. Benzene is a known carcinogen which also affects the central nervous system and the blood (RAIS, 1998; ATSDR, 2000*a*).

Benzene is a volatile, colourless, highly flammable liquid that dissolves easily in water. It has a sweet odour that is characteristic of aromatic hydrocarbons with an odour threshold of 4.7 ppm (15 mg/m^3) (TOXNET, 2000). Inhalation accounts for more than 90% of the total daily intake of benzene (ATSDR, 1997). Intake can also occur through ingestion of contaminated water or food, and through dermal absorption (ATSDR, 1997).

Acute or short-term inhalation of benzene causes neurological effects such as drowsiness, dizziness, rapid heart beat, tremors, headaches, confusion and sometimes unconsciousness (ATSDR, 2000*a*). Inhalation of extremely high concentrations can lead to death through depression of the central nervous system (RAIS, 1998).

Chronic exposure to benzene has a major effect on the blood. Benzene produces a progressive depletion of the bone marrow and an inability of the hematopoietic system to function correctly leading to anaemia (reduced erythrocytes or red blood cells), leukopenia (reduced leukocytes or white blood cells) or thrombocytopenia (reduced platelets or thrombocytes) (RAIS, 1998; ATSDR, 2000*a*). In addition to this, benzene is also known to affect the immune system, cause skin irritation and excessive bleeding, and is a known carcinogen and mutagen (RAIS, 1998; ATSDR, 2000*a*; CCOHS, 2000; US EPA, 2000*c*). Some women have experienced menstrual disorders and decreased ovary size (ATSDR, 1997).

4.3.2 Exposure Assessment

The exposure parameters used for the calculation of the Average Daily Dose (ADD) are outlined in the last two sections of Table 4.1. These exposure

parameters were then substituted into the equations shown in Section 3.4.2 in order to determine the ADD.

The scenarios presented in the table below are as follows:

- A 24-hour exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year, using US EPA default exposure values (US EPA, 1996);
- A 24-hour exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year, using local exposure values.

The 1-hour scenario was not considered as a 1-hour dose-response value for benzene could not be found in the literature. A cancer risk was not calculated because the benzene values from the 9-day air quality monitoring study could not be converted into an annual average. Pollutant concentrations collected via passive sampling are cumulative and cannot be used to determine averages for time periods greater than the actual monitoring period (i.e. convert a weekly total into an annual average). They can only be used to determine averages for time periods less than the actual monitoring period (i.e. convert a weekly total into daily average). In order to calculate cancer risks one must have an annual average pollutant concentration as the cancer risk is a long-term risk.

The ADD for 24-hour benzene exposure are shown in Tables 4.8 and 4.9 below.

Table 4.8: Average Daily Dose (μg/kg/day) for 24-Hour Benzene Exposure using EPA Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	24.3	47.8	40.2	23.9	32.7	1.4	16.8
Child	10.6	20.8	17.5	10.4	14.2	0.6	7.3
Infant	17.6	34.6	29.1	17.3	23.6	1.0	12.2
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**

Adult	41.5	16.6	45.2	1.6	9.9	45.2	72.4
Child	18.1	7.2	19.6	0.7	4.3	19.6	31.5
Infant	30.0	12.0	32.6	1.2	7.2	32.6	52.3

Table 4.9: Average Daily Dose (μg/kg/day) for 24-Hour Benzene Exposure using Local Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	51.3	101.0	85.0	50.5	69.1	2.9	35.5
Child	63.4	124.9	105.0	62.4	85.4	3.6	43.9
Infant	123.0	242.1	203.6	121.0	165.5	6.9	85.1
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	87.7	35.0	95.4	3.4	21.0	95.4	152.8
Child	108.4	43.2	117.9	4.3	26.0	117.9	188.9
Infant	210.1	83.8	228.5	8.2	50.3	228.5	366.2

4.3.3 Dose-response Assessment

The health risk of exposure to benzene was determined for a 24-hour exposure period only. From the data that was collected it was impossible to determine an annual average as explained above. The Minimal Risk Level (MRL) that was used in these health risk assessments was:

- 24-Hour MRL : 161.3 μg/m^3 (ATSDR, 2000)

The 24-hour MRL (based on a 1-14 day exposure period) was obtained from ATSDR (2000). No other 24-hour guideline value for benzene could be found.

The uncertainty factor for this MRL is 300, indicating a medium degree of uncertainty (see Section 2.3.3 for an explanation on how the uncertainty factor is calculated). The MRL is based on an immunological health endpoint, where the sensitive individual would be someone who has a blood disorder / disease (ATSDR, 2000).

Benzene is easily absorbed through inhalation and is rapidly distributed throughout the body, especially in fatty tissue. Metabolism of benzene occurs in the liver and to a lesser extent in bone marrow. Benzene metabolites appear to give benzene its high toxicity. Benzene is metabolised by cytochromes in the liver to form phenol, hydroquinone and catechol. The intermediate benzene oxide is also able to convert to muconic acid. Recent evidence suggests that the genotoxicity of benzene results from a synergistic combination of phenol with hydraquinone, muconaldehyde or catechol (US EPA, 2000*c*). There are two schools of thought as to how benzene metabolites actually affect DNA. Firstly, benzene metabolites may covalently bind to the DNA resulting in replication problems and secondly, benzene metabolites may cause oxidative stress which may lead to damaged DNA (TOXNET, 2000).

Benzene is a known carcinogen of medium carcinogenic hazard. The carcinogenic effects of benzene have been well studied, with many epidemiological studies coming from the chemical industry, shoemaking and oil refineries. There is clear evidence of a causal association between benzene and the blood disorders of acute nonlymphocytic leukemia (ANLL), chronic nonlymphocytic leukemia (CNLL) and chronic lymphocytic leukemia (CLL). Other conditions are also associated with benzene exposure, these include hematologic neoplasms, preleukemia, aplastic anaemia, Hodgkin's lymphoma and MDS or myelodysplastic syndrome. In addition, animal studies have shown increased risk of cancer in multiple organ systems including the liver, lungs, ovaries, mammary glands, oral and nasal cavities and various other organs and glands (US EPA, 2000*c*; TOXNET, 2000).

Acute exposure to benzene causes adverse non-cancer health effects on the central nervous system. Symptoms of acute exposure include drowsiness, headaches, nausea and loss of co-ordination as well as confusion and unconsciousness. At 25 ppm (80 mg/m^3) no effects are likely to be experienced. Exposure to 50 to 100 ppm (161 mg/m^3 to 322 mg/m^3) will lead to tiredness and

headaches. At concentrations above 3000 ppm acute poisoning will occur, characterised by the narcotic action of benzene on the central nervous system. Because benzene is similar to anesthetic gases it has an anesthetic action, where there is an initial state of excitement followed by depression and ultimately death through respiratory failure (TOXNET, 2000). An individual exposed to 20 000 ppm (64 500 mg/m^3) for 5 to 10 minutes is likely to die (CCOHS, 2000). Respiratory effects include hemorrhagic lungs with confluent alveolar hemorrhage and pulmonary oedema, in addition to irritation, cough, pulmonary oedema and pneumonia (TOXNET, 2000).

Chronic exposure to benzene causes adverse non-cancer health effects on the blood and immune system such as reduced numbers of erythrocytes, leukocytes and thrombocytes, although these effects are still considered to be reversible. Continued exposure can lead to aplastic anaemia or leukemia. Two studies where workers were exposed to benzene in the occupational setting showed that levels of 30 to 210 ppm (97 to 678 mg/m^3) for a period of 3 months to 17 years resulted in the classical blood effects described above. Other studies have shown that low level exposures (lower than 1.4 ppm or 4.5 mg/m^3) for long periods of time (up to 21 years) did not result in any blood effects. Occupational studies have also shown effects on the immune system and central nervous system (CCOHS, 2000). The onset of chronic poisoning is slow with the symptoms being vague (headaches, dizziness, nausea, loss of weight) and developing into different symptoms later (such as nosebleeds, bleeding gums, menorrhagia, pallor) (TOXNET, 2000).

Information relating to the reproductive toxicity of benzene as well as its teratogenicity and mutagenicity is conflicting and there is no conclusive evidence that any of these effects definitely occur. Benzene is, however, able to cross the placenta (CCOHS, 2000).

4.3.4 Risk Characterisation

The risk calculation results were obtained by dividing the Average Daily Dose (ADD) by the Minimal Risk Level (MRL) (see Section 3.4.4). The risk calculation is expressed as a hazard quotient in the same way as for nitrogen dioxide.

The monitored 24-hour benzene concentrations are shown in Appendix 5. They range from 31 $\mu g/m^3$ at CC 1230 to 1644 $\mu g/m^3$ at CC 0715. The ATSDR Minimal Risk Level for acute benzene exposure of 161.3 $\mu g/m^3$ was exceeded at 12 of the 14 houses. The hazard quotients calculated for the first scenario (using US EPA default exposure values) show no likelihood of adverse health effects being experienced by any individuals in these homes (see Table 4.10).

Table 4.10: Hazard Quotients for 24-Hour Benzene Exposure using EPA Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	0.2	0.3	0.2	0.1	0.2	0.008	0.1
Child	0.1	0.1	0.1	0.1	0.1	0.004	0.01
Infant	0.1	0.2	0.2	0.1	0.1	0.006	0.1
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	0.3	0.1	0.3	0.01	0.1	0.3	0.4
Child	0.1	0.04	0.1	0.004	0.03	0.1	0.2
Infant	0.2	0.1	0.2	0.01	0.04	0.2	0.3

The second scenario is based on the same 24-hour benzene values as shown in Appendix 5, however, the exposure assessment was done using local exposure assessment values. The exposure times remain the same as those shown in the third section of Table 4.1.

Several of the hazard quotients calculated for this scenario are 1.0 or above, indicating that it is likely that sensitive individuals will begin to experience adverse health effects associated with acute benzene exposure (see Table 4.11). The houses in which infants (and in one case the child too) showed a hazard quotient greater than or equal to 1.0 all had benzene concentrations over 700

$\mu g/m^3$. Only the house with the greatest monitored benzene concentration (CC 0715) showed both infant and child as having HQs greater than 1. Infants are generally more sensitive than adults and children as the same amount of pollutant for a lower body weight results in a higher dose. Sensitive individuals likely to be affected by these benzene concentrations are those with respiratory ailments or those with blood disorders or diseases (TOXNET, 2000).

Table 4.11: Hazard Quotients for 24-Hour Benzene Exposure using Local Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	0.3	0.6	0.5	0.3	0.4	0.02	0.2
Child	0.4	0.8	0.7	0.4	0.5	0.02	0.3
Infant	0.8	1.5	1.3	0.7	1.0	0.04	0.5
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	0.5	0.2	0.6	0.02	0.1	0.6	0.9
Child	0.7	0.3	0.7	0.03	0.2	0.7	1.2
Infant	1.3	0.5	1.4	0.05	0.3	1.4	2.3

4.4 Toluene

4.4.1 Hazard Identification

Toluene was identified as one of the hazardous VOCs emitted during kerosene combustion. Toluene is a colourless liquid that has a sweet, pungent odour (TOXNET, 2000). There is wide variation in odour threshold from detection of the chemical from 0.16 to 37 ppm (0.04 to 9.8 mg/m^3) to recognition of the chemical from 1.9 to 69 ppm (0.5 to 18.4 mg/m^3) (CCOHS, 2000). The health endpoint of concern following toluene inhalation exposure is the central nervous system.

Acute exposure to toluene by inhalation causes slight drowsiness and headaches at lower concentrations, through to fatigue, dizziness, numbness and mild

nausea, mental confusion, unconsciousness and ultimately death at very high concentrations. Toluene also causes respiratory irritation.

Chronic exposure is associated with severe central nervous system damage (symptoms include ataxia, tremors, involuntary eye movements, impaired speech, hearing and vision and cerebral atrophy) as well as hepatomegaly and impaired liver and kidney function (RAIS, 1998). Some studies have shown toluene to be carcinogenic but the overall evidence is not sufficient for toluene to be classified as a carcinogen.

4.4.2 Exposure Assessment

The exposure parameters used for the calculation of the Average Daily Dose (ADD) are outlined in the last two sections of Table 4.1. These exposure parameters were substituted into the equations shown in Section 3.4.2 in order to determine the ADD.

The scenarios presented in the table below are as follows:

- A 24-hour exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year, using US EPA default exposure values (US EPA, 1996);
- A 24-hour exposure period for an adult of 30 years, a child of 10 years and an infant aged less than 1 year, using local exposure values.

A 1-hour exposure period was not considered for toluene as no dose-response values could be found.

Table 4.12 and 4.13 below show the Average Daily Dose calculated for the scenarios mentioned above.

Table 4.12: Average Daily Dose (μg/kg/day) for 24-Hour Toluene Exposure using EPA Exposure Values

	CC0536	CC4038	CC0548	CC0834	CC0823	CC1230	CC1635
Adult	42.2	134.5	78.7	64.6	81.2	43.5	56.7
Child	18.3	58.5	34.3	28.1	35.3	19.0	24.7
Infant	30.5	97.2	56.9	46.7	58.7	31.5	41.0
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	91.0	52.8	63.1	22.6	75.8	92.0	98.3
Child	39.6	23.0	27.4	9.8	33.0	40.0	42.8
Infant	65.8	38.2	45.6	16.4	54.8	66.5	71.1

Table 4.13: Average Daily Dose (μg/kg/day) for 24-Hour Toluene Exposure using Local Exposure Values

	CC0536	CC4038	CC0548	CC0834	CC0823	CC1230	CC1635
Adult	89.0	284.0	166.3	136.4	171.4	91.9	119.8
Child	110.1	351.1	205.6	168.6	211.9	113.6	148.1
Infant	213.4	680.5	398.5	326.8	410.8	220.3	287.1
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	192.1	111.5	133.2	47.8	160.1	194.2	207.6
Child	237.5	137.9	164.7	59.1	197.9	240.0	256.6
Infant	460.4	267.3	319.2	114.5	383.6	465.3	497.4

4.4.3 Dose-response Assessment

The health risk of exposure to toluene was determined for a 24-hour exposure period only (due to a lack of long term monitored data, and the expense in collecting new long term data). The Minimal Risk Level (MRL) that was used in these health risk assessments was:

- 24-Hour MRL : 1064 $\mu g/m^3$

This 24-hour MRL (based on a 1-14 day exposure period) was obtained from ATSDR (2000). No other 24-hour guideline value for toluene could be found.

The uncertainty factor for this MRL is 10 indicating a high degree of confidence in the guideline 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, 2000; TOXNET, 2000).

Toluene enters the body primarily through inhalation although it can be absorbed through the gastro-intestinal tract. It is rapidly metabolised into benzyl alcohol and then is converted into benzaldehyde. Further oxidation produces benzoic acid. Hippuric acid is the metabolite that is excreted via urine. Very little toluene is re-exhaled via the lungs. Toluene accumulates in fatty tissue and can be an internal source of exposure once the initial external exposure has ended (TOXNET, 2000).

According to TOXNET (2000), simultaneous exposure to benzene and toluene results in mutual suppression of their metabolism. However, CCOHS (2000) reports that exposure to benzene and toluene at the same time slows the rate of clearance of toluene from the body leading to enhanced toluene toxicity.

Acute toluene exposure causes depression of the central nervous system. Reversible central nervous system dysfunction and narcosis have been observed, as has cardiac arrhythmia. At 50 ppm (13.3 mg/m^3) headaches and drowsiness occur, from 50 to 100 ppm (13.3 to 26.6 mg/m^3) irritation of the nose, throat and respiratory tract occur, while over 100 ppm (26.6 mg/m^3) individuals become dizzy, numb and report nausea. Over 500 ppm (133 mg/m^3) it has been reported that individuals experience inco-ordination and mental confusion. From 10 000 ppm (2660 mg/m^3) depression of the central nervous system is so severe that one becomes unconscious and may die (ATSDR, 1997; CCOHS, 2000).

Chronic toluene exposure also causes central nervous system effects. Symptoms of chronic exposure include ataxia, tremors, cerebral atrophy,

involuntary eye movements and impaired speech, vision and hearing. Irritation of the respiratory tract and mucous membranes can also occur. Kidney and liver damage have been reported to occur, although some studies have not shown an increase in kidney or liver damage (ATSDR, 1997; TOXNET, 2000).

Both human and animal studies have shown developmental decrements and congenital abnormalities. Occupational toluene concentrations of 19-94 mg/m^3 have been reported to cause hormonal changes in men while higher rates of spontaneous abortions have been noted in pregnancies where there was paternal (and not maternal) exposure to toluene. Pregnant women exposed to toluene or mixtures of solvents have given birth to children with central nervous system dysfunction, attention deficits, minor craniofacial and limb abnormalities, delays in development, retardation of growth and dysmorphism. These study results could have been affected by the confounding factors reported (NZ Ministry for the Environment, 2000).

There is insufficient evidence to suggest that toluene is a carcinogen although some studies have shown possible links between toluene exposure and cancer (CCOHS, 2000).

4.4.4 Risk Characterisation

The risk calculation results were obtained by dividing the Average Daily Dose (ADD) by the Minimal Risk Level (MRL) (as explained in Section 3.4.4). The risk calculation is expressed as a hazard quotient in the same way as for nitrogen dioxide and benzene.

The monitored 24-hour toluene concentrations are shown in Appendix 5. They range from a low of 514 $\mu g/m^3$ at CC 1843 to a high of 3055 $\mu g/m^3$ at CC 4038. The ATSDR Minimal Risk Level for acute toluene exposure of 1064 $\mu g/m^3$ was exceeded marginally at 10 out of the 14 houses, with the 11th exceedance being almost triple the MRL (the maximum concentration was measured at CC 4038).

The hazard quotients for the first scenario (using the US EPA default exposure values) show no likelihood of adverse health effects being experienced by any individuals in these homes, with the maximum HQ being 0.1 for an adult in CC 4038 (see Table 4.14).

Table 4.14: Hazard Quotients for 24-Hour Toluene Exposure using EPA Exposure Values

	CC0536	**CC4038**	**CC0548**	**CC0834**	**CC0823**	**CC1230**	**CC1635**
Adult	0.04	0.1	0.07	0.06	0.08	0.04	0.05
Child	0.02	0.05	0.03	0.03	0.03	0.02	0.02
Infant	0.03	0.09	0.05	0.04	0.06	0.03	0.04
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	0.09	0.05	0.06	0.02	0.07	0.09	0.09
Child	0.04	0.02	0.03	0.009	0.03	0.04	0.04
Infant	0.06	0.04	0.04	0.02	0.05	0.06	0.07

The next scenario is based on the same 24-hour toluene values as shown in Appendix 5, however, the exposure assessment was carried out using local exposure assessment values collected in the time-activity pattern questionnaire. The other exposure parameters remain the same as those in Table 4.1.

None of the hazard quotients calculated for this scenario were above 1.0 (see Table 4.15), indicating that it is unlikely that any individuals will experience adverse health effects due to toluene exposure. The hazard quotients did increase though, being higher than in the previous scenario using US EPA default exposure values. The highest HQ (0.6) was for the infant at CC 4038.

Table 4.15: Hazard Quotients for 24-Hour Toluene Exposure using Local Exposure Values

	CC0536	CC4038	CC0548	CC0834	CC0823	CC1230	CC1635
Adult	0.08	0.3	0.2	0.1	0.2	0.09	0.1
Child	0.4	0.3	0.2	0.2	0.2	0.1	0.1
Infant	0.2	0.6	0.4	0.3	0.4	0.2	0.3
	CC2316	**CC2909**	**CC2662**	**CC1843**	**CC1839**	**CC0797**	**CC0715**
Adult	0.2	0.1	0.1	0.04	0.2	0.2	0.2
Child	0.2	0.1	0.2	0.06	0.2	0.2	0.2
Infant	0.4	0.3	0.3	0.1	0.4	0.4	0.5

4.5 Uncertainty Analysis

The uncertainty associated with this health risk assessment is categorised under the headings of variable uncertainty, model uncertainty and decision-rule uncertainty (Finkel, 1990, in US EPA, 1998).

This study did not address adverse human health effects that might arise from:

- The possible synergistic effects between the pollutants listed in this study and the pollutants present in the atmosphere.
- The long-range transport of pollutants into and out of the study area.
- Occupational exposure of any individual in the study area to any of the pollutants listed here.
- Chemical pollutants resulting from activities unrelated to the domestic combustion of kerosene.

4.5.1 Variable Uncertainty

Variable uncertainty is associated with the variables or parameters that appear in the equations and models used in the risk assessment process. Variable uncertainty in this study includes:

- The measured pollutant levels may be inaccurate due to human error or equipment error. A medium level of confidence is placed on the air quality study results.
- The exact activity patterns and existing health status of the individuals may have been determined incorrectly. It is assumed that the population contains both healthy and sensitive individuals of the ages represented in the scenarios above. A medium level of confidence is placed on the determination of activity patterns.
- The local exposure values derived from the time-activity pattern questionnaires are assumed to be correct for the local population. A medium to low degree of confidence is placed on the questionnaire results as many of the questionnaires were incomplete.
- The US EPA default values for inhalation rate and body weight are assumed to be correct for the local population under study. A medium to high degree of confidence is placed on these default values. The average weight of the individuals who completed the questionnaires was very close to the default US EPA weight, and the inhalation rate is assumed to be fairly representative of any population.

4.5.2 Model Uncertainty

Model uncertainty is associated with the equations or models used in the risk assessment process. For this study these include:

- A medium to high degree of confidence is placed on the dose-response models used by the relevant Agency to determine each dose-response value. These benchmark values are subject to close scrutiny with thorough review processes. The benchmark values used in this study are taken from reliable Agencies.
- The US EPA equations used to quantify exposure and health risks have been subject to rigorous review and a high degree of confidence is placed on these equations.

4.5.3 Decision-Rule Uncertainty

Decision-rule uncertainty arises out of the decisions taken by the risk assessor throughout the risk assessment process. Decision-rule uncertainties in this study include:

- The exposure pathways used in the risk assessment were deemed the most significant pathways for the study. Domestic combustion of kerosene releases pollutants primarily into the air making inhalation of these pollutants the most important pathway for human exposure.
- The local exposure assessment values used from the time-activity pattern questionnaires are based on certain assumptions which have been made. A medium degree of confidence is placed on these assumptions based on local knowledge of the study area population and similar populations in other areas of South Africa.

4.6 Concluding Remarks

Eighty seven percent of the households taking part in the time-activity pattern questionnaire use kerosene for cooking. This represents a potential health risk to the individuals living in these homes who are daily exposed to many harmful pollutants because of the kerosene they use as a fuel. Nitrogen dioxide, benzene and toluene are three pollutants to which these individuals are exposed and their potential risk has been discussed above.

Nitrogen dioxide has the potential to impact on the respiratory and immune systems. Three scenarios were investigated for NO_2 exposure, namely a 1-hour exposure, a 24-hour exposure using US EPA default exposure values (US EPA, 1996) and a 24-hour exposure using local exposure values collected through the time-activity pattern questionnaire. The 1-hour exposure showed no risk of adverse health effects. The 24-hour exposure using US EPA default exposure values showed a slight risk to the sensitive adults living in two of the houses (the exposure period for adults was longer than that of infants and children for this

scenario). The 24-hour exposure scenario using local exposure values showed a potential risk to sensitive individuals living in all of these houses, as well as a potential risk to healthy individuals in some of the houses.

Benzene is a known carcinogen and also affects the blood and central nervous system. For benzene, only the 24-hour scenarios were considered. The 24-hour scenario using US EPA default exposure values revealed no risk to individuals in any of the houses. For the 24-hour exposure scenario using local exposure values a slight risk is present for some sensitive individuals, particularly infants.

Toluene has an effect on the central nervous system and is said to be 'not classifiable' as a carcinogen due to lack of evidence. The same 24-hour scenarios that were used for nitrogen dioxide and benzene exposure were also used to assess toluene exposure. Neither of the scenarios showed any risk to any individuals, sensitive or healthy, as a result of toluene exposure.

From these results it is evident that kerosene combustion products do pose a potential adverse health risk to individuals living in Cato Crest.

CHAPTER FIVE: CRITICAL EVALUATION OF THE US EPA HUMAN HEALTH RISK ASSESSMENT FRAMEWORK

The US EPA health risk assessment model is widely used in many countries. The concept of risk, and in some cases the actual health risk assessment framework, have been included into legislation around the world. At this point it is essential to ask the question – is the framework applicable to all communities around the world? Or is it specific to the community in which it was developed and the people on which it was based? One of the main objectives of this study was to evaluate the US EPA human health risk assessment framework and its suitability for assessing health risks in a typical informal South African community.

The process of conducting a health risk assessment requires the use of several resources in order to construct scenarios which are then used to analyse potential risk. One of these resources is the Exposure Factors Handbook (US EPA, 1996) which contains what are known as 'default options'.

Examples of default options include:

- Inhalation rate
- Dermal dose
- Surface area
- Body weight
- Lifetime
- Intake of various foodstuffs such as fruit, vegetables, fish, shellfish, meat, dairy products, home produced food, breast milk
- Occupational mobility
- Population mobility

- Activity patterns such as time spent showering, bathing, swimming, shopping
- Time spent in different micro-environments such as a garage, petrol station, night club, bar, school, playground, construction site, park, golf courses, farm

The default risk assessment options prescribed by the US EPA are also known as inference guidelines and have been described as 'generic approaches, based on general scientific knowledge and policy judgement, that are applied to various elements of the risk assessment process when the correct scientific model is unknown or uncertain'. The 1983 NRC report *Risk Assessment in the Federal Government: Managing the Process* defined a default option as 'the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary' (NRC, 1994).

These inference guidelines are not rules that bind the US EPA risk assessments, as risk assessors may depart from them when the US EPA judges it to be appropriate. The guidelines have been adopted in order to provide clarity and consistency in the risk assessment process, to ensure that risk assessments reflect the latest scientific understanding and that *ad hoc* decisions are not made in order to influence or manipulate the risk assessment. The US EPA allows departures from the inference guidelines when it decides that there is consensus among scientists that new evidence justifies a departure from the default option. Any potential departure from an inference guideline will be reviewed by the US EPA Science Advisory Board. The Science Advisory Board will then determine whether that departure is acceptable or not (NRC, 1994).

5.1 Evaluation of suitability of US EPA default exposure values

The default exposure assessment values provided by the US EPA Exposure Factors Handbook have been derived through extensive studies on North

American populations. The values presented in the Exposure Factors Handbook (EFH) represent typical values for the behaviour of a person living in North America. The behaviour patterns of South African individuals, particularly those in informal settlements, are expected to differ somewhat from the North American behaviour patterns.

The EFH was used to determine the proportion of time that an adult, child and infant would spend indoors (particularly in the kitchen). From the data presented in the EFH it was decided to use a 7-hour exposure period per day for the adult, and 2 hours exposure per day for the child and infant. The adult was assumed to spend approximately 1 hour per day cooking, 1 hour per day engaged in other activities in the kitchen, 2 hours per day doing chores and approximately 3 hours per day either eating or entertaining indoors. The child was assumed to spend approximately 2 hours per day in the kitchen either eating, doing chores or merely being present in the kitchen. The infant was assumed to be exposed for 2 hours per day, based on the theory that a 1-year old child would spend most of its day in a nursery or other room in the house.

The local exposure times were based on the data collected from the time-activity pattern study in Cato Crest. The exposure times used in each local health risk assessment were 14 hours for an adult, 12 hours for a child and 14 hours for an infant. The adult exposure time was based on the amount of time an individual living in a one-roomed house would spend indoors. The value is an average number of hours, calculated from Questions 47 and 48 shown in Appendix 4. The child is assumed to spend 12 hours inside the house either sleeping, helping with chores or doing school work. The infant was deemed to be exposed for the same time period as the adult, assuming that the infant would be carried on the mother's (or another adult's) back for most of the day, or would be sleeping indoors. The local exposure values were greater than the US EPA default values. The local adult exposure period was double that of the US EPA default of 7 hours

while the local child and infant exposure periods were six and seven times more than the US EPA default (2 hours), respectively.

The default values provided by the US EPA for an exposure assessment are not an accurate representation of the conditions found in Cato Manor, Durban. The community of Cato Manor is an informal urban area characterised by poverty and poor living conditions. This influences the behaviour patterns of the community and the life strategies they employ. The default values provided by the US EPA are based on communities which are probably more similar to many of the formal communities which can be found in South Africa. It is therefore likely that the US EPA default exposure values would be an accurate representation of formal communities in South Africa.

5.2 Differences in calculated risk values for HRA's using US EPA default exposure values and for HRA's using locally collected information as a substitute

The calculated risk value is affected when the risk assessment uses US EPA default exposure values as opposed to locally collected information on daily exposure periods. The reason for this is two-fold. Firstly, residents of Cato Crest tend to live in small, often one-roomed houses with inadequate ventilation. This results in higher pollutant concentrations inside the home. Secondly, the exposure time or time spent 'in the kitchen' is significantly greater than the US EPA default value. This is because local individuals often sleep (and perform other activities) in the same room in which the cooking is done.

The calculated risk value is increased when the risk assessment uses local exposure information as opposed to when US EPA default exposure information is used. The result is that a greater number of hazard quotient values are above one. More individuals are therefore likely to experience potential adverse health effects. The implication is that a health risk assessment conducted for a local

community using US EPA default exposure values is likely to underestimate the potential health risk to the community. In addition, many people living in informal settlements in South Africa are malnourished and could have diseases which result in suppression of the immune system. Their poor health status would result in a greater chance of them being affected by exposure to environmental pollutants, which, as we have just seen, can be underestimated when local conditions are not taken into consideration.

The solution to this problem lies in considering specific local conditions when conducting any health risk assessment in South Africa. This can be done through various methods such as conducting large-scale research into time-activity patterns throughout South Africa; making assumptions based on existing knowledge of a community as to what their time-activity patterns are likely to be; or conducting a small time-activity pattern study for each health risk assessment.

The most desirable of these solutions would be to undertake research throughout South Africa on time-activity patterns in different communities. However, this is also the most expensive and time-consuming option and is unlikely to occur considering the current financial constraints government departments are under. The second option is also not feasible. It is both undesirable and unscientific to base an entire health risk assessment on assumptions and 'best-guess' estimates, whether they are correct or not. The most feasible option would be the last option – to conduct small time-activity pattern studies for each health risk assessment. A study like this need not be complicated in design or very time-consuming. It could consist merely of an observer making notes on a few household's daily activities.

5.3 Applicability of the US EPA health risk assessment framework to South African populations

From the discussion above it is clear that the US EPA health risk assessment framework is applicable to South Africa. However, information on local conditions should be substituted into the model where necessary. Because of the high costs and time associated with the collection of new data, it is likely that South African risk assessments will be based on expert decisions where local information is required. This increases the decision-rule uncertainty of the risk assessment. Observation of local populations can also be used to infer exposure patterns and behaviour.

The health risk assessment framework is not able to evaluate exposure to pollutant mixtures and synergistic effects. This is not a problem that is unique to South Africa though. Most communities throughout the world face exposure to multiple pollutants through traffic, industry, dust, fires and so on. In addition, the synergy between pollutants in these mixtures will occur in both informal and formal settlements. Where those living in informal settlements may be at a disadvantage is their nutritional or health status. The US EPA health risk assessment framework does not allow one to assess the effects of a pollutant directly on an individual with pneumonia, for example, as opposed to an individual with tuberculosis. It only allows for sensitive individuals versus healthy individuals to be assessed.

CHAPTER SIX: CONCLUSION

6.1 Summary of results

The objective of this study was to investigate the application of the US EPA human health risk assessment framework for quantifying the adverse human health effects of exposure to inhaled kerosene pollutants in the South African context. The study focused on the informal settlement of Cato Crest within Cato Manor, Durban and was based on air quality data collected in the area as well as time-activity pattern data that was collected from the community through questionnaires. A total of 69 time-activity pattern questionnaires were collected from households in Cato Crest, with 14 houses being chosen from this group to participate in the air quality monitoring study. Nitrogen dioxide, benzene and toluene were monitored in each household over a 9-day period in September 2000.

Kerosene is widely used throughout South Africa as a domestic fuel. Cato Crest (one of the informal settlements in Cato Manor, Durban) is also known to have high kerosene use. The residents of Cato Crest rely heavily on kerosene for their cooking and lighting requirements. Kerosene is also sometimes used for heating, although this is less frequent.

The literature presented in Chapter 2 shows that there are many health effects associated with the use of kerosene as a domestic fuel. The pollutants emitted during the combustion of kerosene include, among others, carbon monoxide, carbon dioxide, nitrogen dioxide, sulphur dioxide, formaldehyde, particulates and various hydrocarbons such as 1,3 butadiene, benzene, toluene and styrene. Each of these pollutants has several health effects associated with either acute or prolonged exposure. These health effects range from irritation of mucous membranes to cancer.

The air quality data collected in September 2000 revealed that many of the products of kerosene combustion can be found indoors in the homes of Cato Crest. Nitrogen dioxide was measured indoors and found to be present in concentrations between 51.8 $\mu g/m^3$ (CC 0823) and 103.9 $\mu g/m^3$ (CC 0536) for the 1-hour exposure period. The Californian EPA 1-hour REL is 470 $\mu g/m^3$ and was therefore not exceeded at any of the houses. For a 24-hour exposure period the concentrations were between 1243.8 $\mu g/m^3$ at CC 0823 and 2493.8 $\mu g/m^3$ at CC 0536. The New Zealand REL for a 24-hour period of 100 $\mu g/m^3$ was exceeded at all the houses in the study group. The monitored benzene concentrations were between 31 $\mu g/m^3$ (CC 1230) and 1644 $\mu g/m^3$ (CC 0715). The ATSDR MRL for benzene of 161.3 $\mu g/m^3$ for a 24-hour period was exceeded at 12 of the 14 houses in the study group. Toluene concentrations ranged from 514 $\mu g/m^3$ (at CC 1843) to 3055 $\mu g/m^3$ (at CC 4038). The ATSDR 24-hour MRL of 1064 $\mu g/m^3$ for toluene was exceeded at 10 of the 14 houses in the study group.

The results of the time-activity pattern questionnaires revealed that the exposure period of individuals in Cato Crest was far greater than the US EPA default exposure periods given in the Exposure Factors Handbook. The adult exposure period doubled from 7 hours (US EPA default) to 14 hours (based on the time-activity pattern data which revealed that many people sleep in a multi-function room in which they also cook). The child exposure period was increased six-fold from 2 hours (US EPA default) to 12 hours (based on the time-activity pattern data in Appendix 4). The infant exposure period increased seven-fold from 2 hours to 14 hours. This large increase in exposure period from US EPA default values to the local values of Cato Crest results in a much greater dose of pollutant to the body resulting in a greater potential for adverse health effects to occur.

It is important to remember that the air quality and time-activity pattern samples are not valid statistical samplings of the total population. However, the consistency of the results provides some measure of confidence in the results of these studies.

The results of these two studies were then used in several human health risk assessments which were based on the US EPA model of health risk assessment. This was done in order to determine the potential risk of adverse health effects associated with inhalation of nitrogen dioxide, benzene and toluene.

Exposure to the 1-hour NO_2 concentrations is not likely to produce adverse health effects. Exposure to the 24-hour NO_2 concentrations using the US EPA default exposure periods will produce a slight potential health risk in sensitive individuals living in two of the households (CC 0536 and CC 2662), where the 24-hour NO_2 concentration is greater than 2000 ug/m^3. The same NO_2 concentration at local exposure periods has the potential to result in health effects being experienced by both healthy and sensitive individuals. The health effects include coughing, wheezing, chest tightness and increased airway resistance. Sensitive individuals are those with respiratory ailments such as asthma.

The 24-hour benzene concentrations at the US EPA default exposure periods is not likely to produce adverse acute health effects. However, when the local exposure periods are used, concentrations over 700 $\mu g/m^3$ for a 24-hour period may result in health effects in some sensitive individuals (those individuals with respiratory ailments or blood diseases/disorders).

The 24-hour toluene concentrations at both the US EPA default exposure periods and the local exposure periods are not likely to result in adverse health effects in any individuals.

This study has revealed that the US EPA default exposure values are not representative of local conditions in Cato Crest. However, the US EPA values may be useful for other communities in South Africa that are a closer representation of the North American communities on which the US EPA data are based. For future health risk assessments taking place in informal communities or in communities where the behaviour is known to differ from typical North American behaviour, it would be better for the risk assessor to make assumptions about local exposure patterns, provided that local data collection is not possible.

6.2 Recommendations for further studies

It is recognised that this study has shown certain limitations. Firstly, the time-activity pattern questionnaire was conducted on a sample of 69 households from approximately 4 500 households in Cato Crest. The questionnaire did not request the time-activity patterns of children and infants to be supplied, and no questions on other possible sources of exposure to pollutants (such as occupational exposure) were included. As the time-activity pattern study was not meant to be statistically representative of the population, it merely provides an indication of the lifestyles of Cato Crest residents. Future studies could be improved by increasing the sample size, by having people keep a diary of activities they do each day, by having observers staying with someone from the community to verify answers given, and by asking questions requiring more detailed answers than this study.

Secondly, the air quality study was also conducted on a limited sample of 14 households over a limited time period of 9 days. Future studies should perhaps enlarge the sample size and conduct monitoring over one or even several years in order to understand seasonal fluctuations in pollutant concentrations and to be able to assess long-term health risks of the pollutants. The spectrum of pollutants monitored should ideally be enlarged to include sulphur dioxide, particulates and

other VOCs. In addition, outdoor air quality monitoring of the same pollutants would be useful, as would studies on the ventilation of the houses of Cato Crest. Knowledge of the additive and synergistic effects between pollutants would be useful in determining potential health risks to populations. This is a field that is presently being studied by many individuals.

This study has shown that the use of kerosene as a domestic fuel in the informal settlement of Cato Crest in Cato Manor, Durban poses a potential health risk to individuals exposed to the combustion products of kerosene. Further research is required to understand exactly what health effects can be expected in which individuals, and what role disease, nutritional status and other exogenous factors play in determining the potential health effects likely to be experienced. In addition, research into housing design should be conducted to improve ventilation, for example, without increasing the costs of building an informal house in South Africa. A change from kerosene to a cleaner fuel is desirable, although many attempts have already been made at changing fuel use patterns in informal settlements without success.

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APPENDIX 1

TABLE SHOWING FUEL USE PER ACTIVITY IN SOUTH AFRICAN HOUSEHOLDS

	COOKING	HEATING	LIGHTING
Electricity	4 265 305	4 030 850	5 220 826
Gas	286 657	107 689	35 512
Kerosene	1 943 862	1 294 964	1 144 014
Wood	2 073 219	2 417 724	-
Coal	320 830	735 632	-
Dung	106 068	84 447	-
Candles	-	-	2 583 031
Other	63 629	388 266	76 190
	9 059 570	9 059 572	9 059 573

Source: Statistics South Africa, 2000

Note: Electricity includes both that obtained from the authority as well as that obtained from other sources.

Units: Number of households

APPENDIX 2

NUMBER:
INTERVIEWER:
OBSERVER:

Health Questionnaire

Chemicals in the environment can often make people sick or give them certain diseases. Researchers are sometimes able to find out what chemicals cause disease and can offer help to a community by telling them how to avoid coming into contact with those chemicals.

In your community paraffin is often used as a fuel for cooking, heating and lighting. Paraffin is a chemical that could cause disease or illness through the gases that are given off while it is burning. The amount of paraffin that you burn, the appliance that you use and the time you spend cooking and doing other things in your home can affect whether paraffin will make you sick or not. In order to find out if paraffin is likely to make you sick we need to ask you a few questions about the things you do around your home during the day. Would you mind if we asked you some questions about this? It will take approximately half an hour of your time and your answers will be kept confidential.

DETAILS OF THE PERSON BEING INTERVIEWED

Instruction to interviewer:

a) *Respondent must be 18 years old or older*

b) *Respondent must be ordinarily resident in the household*

1 Surname:_____

2 Name: _____

3 Physical Address: _____CC_____ (House number)

_____ (Area number)

_____

_____

4 Contact telephone no.: _____

5 Date: _____

Your confidentiality is ensured, **YOUR PERSONAL INFORMATION WILL NOT BE GIVEN OUT TO ANYONE** or any institution.

INTERVIEWER: PLEASE RING APPROPRIATE NUMBER

PERSONAL DETAILS

6 Sex: Male / Female

7 Date of Birth: _____ (YY/MM/DD)

8 What is your approximate weight? _____ kg

9 How long have you been living in this area (Cato Crest)?

a. Since birth
b. More than 5 years
c. From 2 to 5 years
d. Less than 2 years

10 How long have you been living in this present house?

a. Since birth
b. More than 5 years
c. From 2 to 5 years
d. Less than 2 years

11 Who is the head of the household? _____

12 What is your relationship to the head of the household?

a. I am the head of the household
b. Spouse
c. Brother
d. Sister
e. Daughter
f. Son
g. Grand-daughter
h. Grand-son
i. Parent
j. Niece
k. Nephew
l. Other _____

13 Are you currently:

a. Employed full-time
b. Employed part-time
c. Self-employed (Give details _____)
d. Retired, on pension
e. Retired, not on pension
f. Unemployed, not seeking work
g. Unemployed, seeking work
h. Attending school /technical college / university
i. Other _____

14 a How many people live in this household with you? _____

(Include people who are family members, those who are resident here on a daily basis and tenants or lodgers.)

14 b Complete their details here:

	Name	**Relationship to you**	**Approximate Age**	**Sex (M/F)**
Person 1				
Person 2				
Person 3				
Person 4				
Person 5				
Person 6				
Person 7				
Person 8				
Person 9				
Person 10				
Person 11				
Person 12				
Person 13				
Person 14				
Person 15				

FUEL USE

15 What type of fuel do you use mostly for cooking?

a. Electricity
b. Wood
c. Coal
d. Gas
e. Paraffin
f. Cattle manure
g. Grasses
h. Open fire
i. Other _____

16 **If the fuel use was (e) paraffin**, what type of stove is used?

a. Paraffin burner with sekeni
b. Paraffin primer and pump
c. Other _____

17 Approximately how long have you been using the stove? _____

18 What type of fuel do you use mostly for heating?

a. Electricity
b. Wood
c. Coal
d. Gas
e. Paraffin
f. Cattle manure
g. Grasses
h. Open fire
i. Other._____

19 What do you mostly use for a light source in the evenings?

a. Candles
b. Paraffin lamp
c. Gas lamp
d. Electricity
e. Fire
f. Combination of _____
g. Other (e.g. battery) _____

BUILDING STRUCTURE

20 What is your home constructed of?

a. Bricks, informal housing
b. Bricks, formal housing
c. Mud
d. "Wattle & Daub" / Gum tree and mud
e. Metal sheeting
f. Wooden boards
g. Plastic sheeting
h. Canvas sheeting
i. Squatter shack, (give details) :

_____

_____

j. Other _____

21 Please complete the following table for each room in your house.

Room (give a name, e.g. bedroom)	Number of windows present?

22 Do you open the windows or door while you are cooking?

In winter (daytime) Yes / No

In summer (daytime) Yes / No

In winter (at night) Yes / No

In summer (at night) Yes / No

23 If windows are opened in the room where cooking is done, how often are they opened?

a. Every day
b. 2-3 times in the week
c. 2-3 times in the month
d. Seldom
e. Never

24 How many doors are in your house (leading to the outside)?

_____

25 Is there a chimney? ('shaemula') _____

26 Is there a gap between the walls and the roof? _____

27 Do you ever light a fire inside your house?

a. Winter, yes
b. Winter, no
c. Summer, yes
d. Summer, no

If yes:

28 How often do you do this in summer? _____

29 How often do you do this in winter? _____

30 Do you ever open any windows or doors when the fire is burning or shortly afterwards? _____

COOKING & SMOKING

31 Who does most of the cooking in the home? _____

32 How many meals per day are cooked by that person?

a. One (1)
b. Two (2)
c. Three (3) or more

33 How many meals are cooked in the house per day? _____

34 Who else helps with the cooking or food preparation? _____

_____

35 Is the cooking done mostly indoors or outdoors during the day?

a. *Winter, indoors* Yes / No
b. *Winter, outdoors* Yes / No
c. *Summer, indoors* Yes / No
d. *Summer, outdoors* Yes / No

36 Is the cooking done mostly indoors or outdoors during the evening?

e. *Winter, indoors* Yes / No
f. *Winter, outdoors* Yes / No
g. *Summer, indoors* Yes / No
h. *Summer, outdoors* Yes / No

37 Approximately how long does it take to cook the main meal of the day?

a. Less than 1 hour
b. 1-2 hours
c. 2 or more hours

38 Does anyone sleep in the same room that the meals are cooked in?

Yes / No

39 How many people sleep in the same room that the meals are cooked in?

_____

40 Who are they? _____

_____

_____

_____

_____

41 Are there other people present indoors in the kitchen during the cooking of the meal? (i.e.: people who are not involved in the cooking)

a. Yes, all the time
b. Yes, frequently
c. Not very often
d. Never

42 If there are people present in the kitchen during cooking, who are they:_____

_____

43 Is there anybody who lives in the house that smokes?

a. Yes
b. No

44 Who are they? _____

45 Do they smoke inside? Yes / No

If yes:

46 How often do they smoke indoors at home?

a. Several times a day
b. Once or twice a day
c. Once or twice a week
d. Once or twice a month

TIME-ACTIVITY PATTERNS

IN WINTER:

47 How many hours do you usually spend each day doing the following:

a. Cooking inside your house _____
b. Inside your house doing other activities _____
c. Cooking outside your house _____
d. Outside your house but still on your property (other activities) _____
e. On or near a road (e.g. walking to work) _____
f. In a bus / car / taxi _____
g. Inside at work (away from home) _____
h. Outside at work (away from home) _____
i. Sleeping _____

IN SUMMER:

48 How many hours do you usually spend each day doing the following:

a. Cooking inside your house _____
b. Inside your house doing other activities _____
c. Cooking outside your house _____
d. Outside your house but still on your property (other activities) _____
e. On or near a road (e.g. walking to work) _____

f. In a bus / car / taxi _____

g. Inside at work (away from home) _____

h. Outside at work (away from home) _____

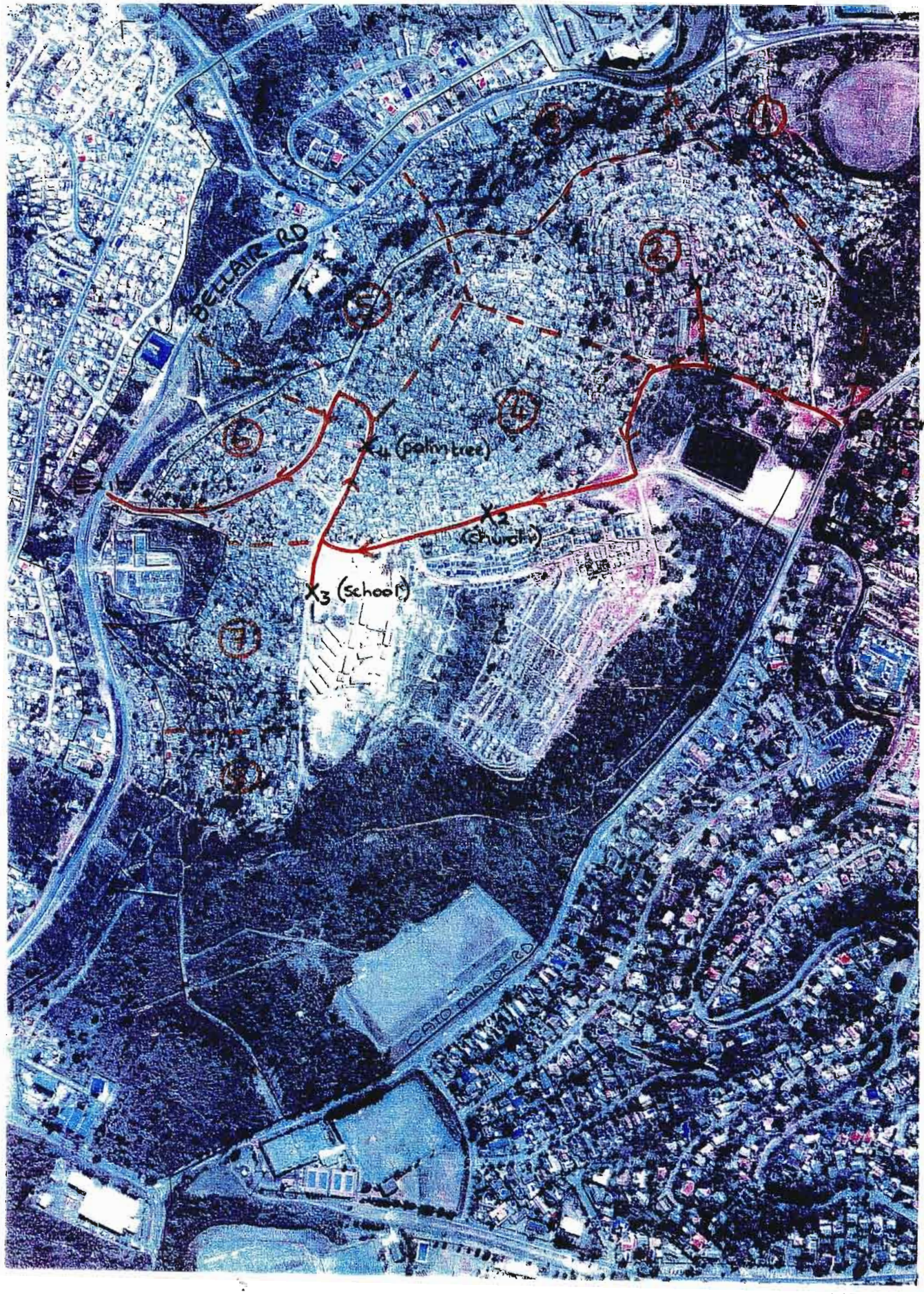
i. Sleeping _____

Thank you for taking the time to complete this questionnaire.

APPENDIX 3

AERIAL PHOTOGRAPH OF CATO CREST SHOWING THE ROUTE STUDENTS TOOK IN ORDER TO SAMPLE HOUSES FOR THE QUESTIONNAIRE.

Scale = 1 : 5 000 1cm = 50m



Road through Cato Crest

X Drop-off and collection points

O Areas

CATO CREST

N

APPENDIX 4

SELECTED RESULTS OF THE TIME-ACTIVITY PATTERN SURVEY

Question 6 – Gender of interviewee

Males	Females	Total
3	66	69

Question 7a – Average age of interviewee

34 years

Question 8b – Average weight of interviewee

68 kilograms

Question 9 – Length of time the interviewee has lived in the area

Since birth	More than 5 years	Two to 5 years	Less than 2 years
0	36	20	13

Question 10 – Length of time the interviewee has lived in this house

Since birth	More than 5 years	Two to 5 years	Less than 2 years
0	34	16	19

Question 12 – Who is the interviewee in relation to the head of the household?

Head	Spouse	Brother	Sister	Daughter	Son	Other
21	24	2	1	5	0	16

Question 13 – What is your current activity?

Employed full time	Employed part time	Self employed	Retired, on pension	Retired, not on pension	Unemployed, not looking for work	Unemployed, looking for work	Studying
10	8	6	3	1	5	34	2

Question 14 – Number of people living in this household?

1 to 5 people	6 to 10 people	11 to 15 people	16 to 20 people	21 to 25 people
45	19	2	0	1

Question 15 – What type of fuel do you use for cooking?

Electricity	Wood	Coal	Gas	Paraffin	Dung	Grasses	Fire	Other	Mixed W/P	Mixed G/P
0	0	0	1	60	0	0	0	0	3	5

Question 16 – For paraffin users, what type of stove is used?

Paraffin burner with wick / 'sekeni'	Paraffin primer and pump	Both	Unknown
60	6	2	1

Question 17 – Approximately how long has the stove been in use?

Less than 1 year	One to 2 years	More than 2 years	Unknown
24	24	18	3

Question 18 – What type of fuel do you use for heating?

Electricity	Wood	Coal	Gas	Paraffin	Dung	Grasses	Fire	Nothing	Blankets
0	13	0	0	22	0	0	8	12	14

Question 19 - What type of fuel do you use for lighting?

Candles	Paraffin	Gas	Electricity	Fire	Candles & Paraffin	Candles & Gas	Other
39	9	0	0	0	20	1	0

Question 20 – What is your home constructed of?

Brick, informal	Brick, formal	Mud	"Wattle & Daub"	Metal sheeting	Wooden boards	Plastic sheeting	Canvas sheeting	Combination of wood, mud, plastic & metal
0	3	9	13	5	10	0	0	29

Question 22 – Do you open the windows or door while you are cooking?

Winter, daytime		Winter, night		Summer, daytime		Summer, night	
Yes	No	Yes	No	Yes	No	Yes	No
30	30	5	64	50	19	7	62

Question 23 – How often are the windows opened?

Every day	2 to 3 times / wk	2 to 3 times / month	Seldom	Never
41	5	0	5	18

Question 24 – How many doors in your house lead outside?

1 to 2	3 to 4	5 to 6	7 to 8
62	7	0	0

Question 25 – Does your house have a chimney?

Yes	No
0	69

Question 26 – Is there a gap between the walls and the roof of your house?

Yes	No	No answer
20	47	2

Question 27 – Do you ever light a fire inside your house?

Winter		Summer	
Yes	No	Yes	No
10	53	3	60

Question 28 – How often do you do this in summer?

Daily	Weekly	Monthly
3	0	0

Question 29 – How often do you do this in winter?

Daily	Weekly	Monthly
10	0	0

Question 30 – Do you ever open any windows or doors when the fire is burning or shortly afterwards?

Yes	No
3	7

Question 33 – How many meals are cooked indoors per day?

One	Two	Three
22	33	14

Question 35 – Is the cooking done indoors or outdoors during the day?

Winter, indoors		Winter, outdoors		Summer, indoors		Summer, outdoors	
Yes	No	Yes	No	Yes	No	Yes	No
67	2	0	69	67	2	1	68

Question 36 – Is the cooking done indoors or outdoors during the night?

Winter, indoors		Winter, outdoors		Summer, indoors		Summer, outdoors	
Yes	No	Yes	No	Yes	No	Yes	No
67	2	0	69	67	2	0	69

Question 37 – Approximately how long does it take to cook the main meal?

Less than 1 hour	1 to 2 hours	More than 2 hours	No answer
8	45	11	5

Question 38 – Does anyone sleep in the same room that the meals are cooked in?

Yes	No
50	19

Question 39 – How many people sleep in the same room that the meals are cooked in?

1 to 3	4 to 6	7 to 9	10 to 13
40	8	1	0

Question 41 – Are there other people present in the kitchen during the cooking of the meal?

All the time	Frequently	Not often	Never
13	11	26	19

Question 43 – Is there anybody who lives in the house that smokes?

Yes	No
36	33

Question 45 – Do they smoke indoors?

Yes	No
26	10

Question 46 – How often do they smoke indoors at home?

Several times per day	Once or twice a day	Once or twice a week	Once or twice a month
21	4	1	2

Question 47 – Average hours spent per activity in winter

Cooking indoors	Indoors, other	Cooking outdoors	Outdoors, other	Near a road	In transport	Indoors, at work	Outdoors, at work	Sleeping
2	2.6	2	2	1	1.5	8.2	2.8	10

Question 48 – Average hours spent per activity in summer

Cooking indoors	Indoors, other	Cooking outdoors	Outdoors, other	Near a road	In transport	Indoors, at work	Outdoors, at work	Sleeping
2	2.4	2	4.3	1	1.5	8.3	2.4	9

APPENDIX 5
AIR QUALITY STUDY RESULTS

Nitrogen Dioxide, Benzene and Toluene Concentrations Measured by Passive Sampling (ug/m^3)

House number	Average 1-Hour Nitrogen Dioxide	Cumulative 24-Hour Nitrogen Dioxide	Cumulative 24-Hour Benzene	Cumulative 24-Hour Toluene
CC 0536	103.9	2493.8	552	958
CC 4038	55.5	1330.9	1087	3055
CC 0548	60.9	1461.4	914	1789
CC 0834	Incomplete	Incomplete	543	1467
CC 0823	51.8	1243.8	743	1844
CC 1230	67.6	1623.1	31	989
CC 2316	60.4	1449	943	2067
CC 2909	57.9	1389.9	376	1200
CC 2662	93.3	2238.8	1026	1433
CC 1843	55.9	1343.3	37	514
CC 1635	69.8	1675.9	382	1289
CC 1839	61.2	1467.7	226	1722
CC 0797	67.4	1616.9	1026	2089
CC 0715	64	1536	1644	2233