# The Grande Prairie and Area Community Exposure and Health Effects Assessment Program

FINAL REPORT

Alberta
HEALTH AND WELLNESS

MISTAHIA
HEALTH REGION

March 2002

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The Grande Prairie and Area Community Exposure and Health Effects Assessment Program is the second in a series of reports published by the Health Surveillance Branch at Alberta Health and Wellness.

Other publications in this series include:

The Alberta Oil Sands Community Exposure and Health Effects Assessment Program: Pilot Study Report, 1997.
The Alberta Oil Sands Community Exposure and Health Effects Assessment Program: Summary Report, 2000.
The Alberta Oil Sands Community Exposure and Health Effects Assessment Program: Technical Report, 2000.
The Alberta Oil Sands Community Exposure and Health Effects Assessment Program: Methods Report, 2000.

For more information or copies of any of these documents contact:

Health Surveillance
Alberta Health and Wellness
P.O. Box 1360
10025 – Jasper Avenue
Edmonton, Alberta
T5J 2P4

Phone: (780) 427-4518
Fax: (780) 427-1470
Internet: www.health.gov.ab.ca

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This report provides the results of a community exposure and health effects assessment conducted in Grande Prairie during the summer and fall of 2000. The assessment used a protocol developed in Alberta to assess personal exposure and the potential health impact of airborne contaminants.

This report would not have been possible without the valuable contributions made by:

### *Science Team*

| | |
|---|---|
| Dr. Stephan Gabos | Alberta Health and Wellness |
| Alexander MacKenzie | Alberta Health and Wellness |
| Jonathan Robb | Alberta Health and Wellness |
| Dr. Donald Schopflocher | Alberta Health and Wellness |
| Susan Shaw | Alberta Health and Wellness |
| Fu-Lin Wang | Alberta Health and Wellness |

### *Health Surveillance Staff*

Kendra Doyle
Regena Wiseman

### *Consultants*

Erik Ellehoj
Irwin Huberman
Dennis Prince

### *Mistahia Health Authority Staff*

Sharon Mill
Dr. Hilary Wynters

### *Field Coordinators*

Tricia Lowrey
Jaime Pinzon

### *Field Monitoring Team Members*

Jim Meagher
Michael Melanson
Terry Scott
Raymond De Smit
Jeremy Spencer
Elmer Spilchen
Merry Turtiak

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# 1.0 Executive Summary

## 1.1 Objectives

This report summarizes the results of a community exposure and health effects assessment undertaken in Grande Prairie to assess the impact of airborne contaminants on the health of the population. The report describes the population and personal distribution of exposure to airborne chemicals and particulates in the city of Grande Prairie and surrounding region. Using a personal exposure model, the relative contribution of various exposure sources and pathways to airborne chemicals is estimated and associations between exposure to airborne chemicals and human health effects are described.

## 1.2 Methods and Analysis

The data used for the analysis was collected over a 28 week period (March 24 to October 4, 2000), using volunteers from the city of Grande Prairie as well as surrounding areas such as Sexsmith, Beaverlodge, and Debolt. Data was evaluated and, where applicable, additional comparisons were made to the scientific literature or to comparable data collected elsewhere in Alberta. The study collected a variety of measures for each participant, including personal, indoor, and outdoor levels of selected contaminants (sulfur dioxide, nitrogen dioxide, ozone, a group of volatile organic compounds, and particulate matter), measures of other sources of exposure, diet and health behaviors, and selected health outcomes.

## 1.3 Significant Findings

Despite ongoing recruitment activities, the field co-ordinator encountered difficulties obtaining the targeted number of volunteers. This suggests that exposure to contaminants from air-borne sources may not be an issue of primary concern to most residents of Grande Prairie and the surrounding areas. It seems inconsistent with expectations in view of the notoriety given to concerns with air quality in the region.

The sample, although smaller than anticipated, provided measures of exposure from all areas of the city of Grande Prairie, as well as a number of communities in the surrounding region. The sample generally represented the rest of the population in gender and level of education, but had a larger proportion of high-income households. A significantly smaller proportion of study participants were smokers compared to an independent assessment of the area.

Analysis of the individual measures of exposure indicated that:

- Nitrogen dioxide levels were low compared to existing guidelines and were comparable to other similar studies.
- Levels of sulfur dioxide measured in Grande Prairie were very low compared to existing guidelines.
- Ozone indoor and personal levels were very low. Outdoor levels were an order of magnitude higher, which suggests that ambient measures are an inadequate measure of personal exposure.
- Indoor concentrations were the predominant factor affecting personal exposure to VOCs. Other factors were of only minor relative importance, which suggests that exposure to VOCs was predominantly from sources affecting indoor levels.

- $PM_{2.5}$ outdoor concentrations measured in Grande Prairie were lower than other communities and well below guidelines. They were not important as either a driver or a pathway of personal exposure.

An exposure model was developed to describe variation in personal exposure. Nine general factors were examined as potential causes of exposure variation: 1) gender; 2) urban-rural location; 3) housing characteristics; 4) ownership of a garage; 5) job status; 6) smoking characteristics; 7) time activity pattern; 8) outdoor concentration levels; and 9) indoor concentration levels.

The major findings were:

- Indoor variation accounted for over one-half of the variation in personal $NO_2$ exposure described by the model. Time activity was also an important driver of personal exposure while smoking and housing characteristics had minor effects. The most important factor within time activity appears to be the amount of time spent indoors at work; higher exposure is associated with more indoor work time.
- Overall, variations of outdoor levels accounted for roughly one-half of the variation in personal $SO_2$ exposure explained by the model. Time activity was also an important factor affecting personal exposure.
- The variation in personal $O_3$ exposure described by the model was due to outdoor levels and time activity acting directly and indirectly through indoor levels. Indoor concentrations were also an important factor and housing characteristics were found to be of relatively minor importance.
- Variation in indoor concentrations are the predominant factor affecting personal VOCs exposure (except benzene), while other factors were of minor relative importance. Outdoor concentrations did not have a significant direct effect on personal exposure but had a small indirect effect through indoor air.
- In contrast, benzene exposure was influenced by time activity patterns. Specifically, time spent outdoors at home and at work were important influences.
- Indoor concentration levels were predictive of particulate matter exposure. In an analysis of indoor concentration level, the only variable that emerged as predictive was the number of cigarettes smoked.

In addition to measuring exposure, the study collected a variety of indicators of health status. These included lifestyle behaviors, previous diagnoses and contacts with the health care system, in addition to objective measures of neurocognitive functioning and biomarkers of exposure and effect.

The major findings were:

- Participants indicated that they consumed less than the recommended servings of grain products and that they consumed an average of 2-3 servings of sweets or other non-nutritious foods each day. The average body mass index (BMI) for the sample was 27.3, higher than the estimated Canadian average of 25.4, indicating a higher level of obesity. The sample also reported an average amount of physical activity that barely met minimum recommendations established by Health Canada.
- Biomarkers for benzene, toluene, and nicotine were measurable (i.e., above laboratory detection limits), but all levels were unassociated with measures of exposure.
- No statistically significant differences in neurocognitive functioning were found between the study sample and reference populations.

- The most common self-reported diagnosis of chronic diseases in the sample were back problems (35%) and allergies (34%).

## 1.4 Recommendations

**1. Establish ongoing monitoring of personal exposure levels to air contaminants.**

This study did not find evidence of significantly elevated personal exposure to airborne contaminants. A long-term program is recommended that would monitor personal exposure to contaminants in order to detect any changes over time.

**2. Participate in the implementation of an organized approach to community exposure and health effects assessment in the province in support of long-term comparisons with other areas across the province.**

Strategic information gathering on community exposure and health across the province is key to evidence based decision making, on managing health risks, and the development of health promotion, disease prevention, and exposure control strategies. Such information is also important to public concerns about air contaminants and health and for the development of health based air quality guidelines at a local, regional, and provincial level. Therefore, in collaboration with other agencies and organizations such as Alberta Health and Wellness, regional health authorities, the Clean Air Strategic Alliance, Health Canada, and Alberta Environment, a co-ordinated system should be developed for the ongoing collection, analysis, and interpretation of air quality and health information. Such a system should be sustainable, cost-efficient, and should build on already existing resources without adding significant new costs.

**3. Adopt and promote the use of innovative methods and technologies such as personal exposure monitoring to further our understanding of the relationship between air quality and human health.**

The results of this study indicate that the ambient concentration of contaminants measured at monitoring stations is not a good predictor of individual exposure (i.e., personal exposure). In the study of health and air quality and in the development of human health-based air quality guidelines, it is important to go beyond traditional emission inventories and ambient air quality monitoring. Personal exposure monitoring is a method that can complement existing methods.

## 2.0 Introduction

Human health concerns related to air quality have been raised by various stakeholder groups throughout Alberta including First Nations, environmental associations, governments, and the Clean Air Strategic Alliance (CASA). In response, a long-term, systematic approach to data gathering has been implemented in Alberta that will improve our knowledge about the link between the environment and human health. The approach combines two broad concepts in an integrated population-based environmental health framework: (1) the direct measurement of personal and population exposure to environmental factors, and (2) the epidemiologic surveillance of health outcomes in the population.

The major industries within the Grande Prairie region are: Alberta Energy Corporation, Amoco Canada, Anadarko Canada, Apache Canada, Burlington Resources, Canadian Natural Resources Ltd., Devin, Husky Oil, Pan Canadian Resources, RioAlto, Suncor Energy, and Talisman Energy (oil and gas); the Canadian Forest Products Ltd. (Canfor; sawmill); Weyerhaeuser Canada Ltd. (bleached kraft pulp mill); Ainsworth Lumber Co. Ltd. (oriented strand board plant); Canadian Agra Foods Inc. (canola crushing plant); Risley Manufacturing (builds forestry-related attachments); and Sterling Pulp Chemicals (sodium chlorate plant). In addition to the industries mentioned, there are a number of smaller industries in the surrounding area and the region is currently experiencing rapid growth and development.

The Grande Prairie and Area Community Exposure and Health Effects Assessment Program is part of an ongoing effort by public health officials in Alberta to collect information on airborne contaminants and health concerns across the province. The information gathered in the Grande Prairie region will become part of the province wide database and will allow comparisons of human exposure and levels of airborne contaminants across among various communities in Alberta. A previous study has examined Fort McMurray and Lethbridge. A study is currently being conducted in the Fort Saskatchewan region.

Alberta Health and Wellness and the Mistahia Regional Health Authority recognize that there are significant gaps in information that limit our understanding of the relationship between air quality and human health outcomes. These include:

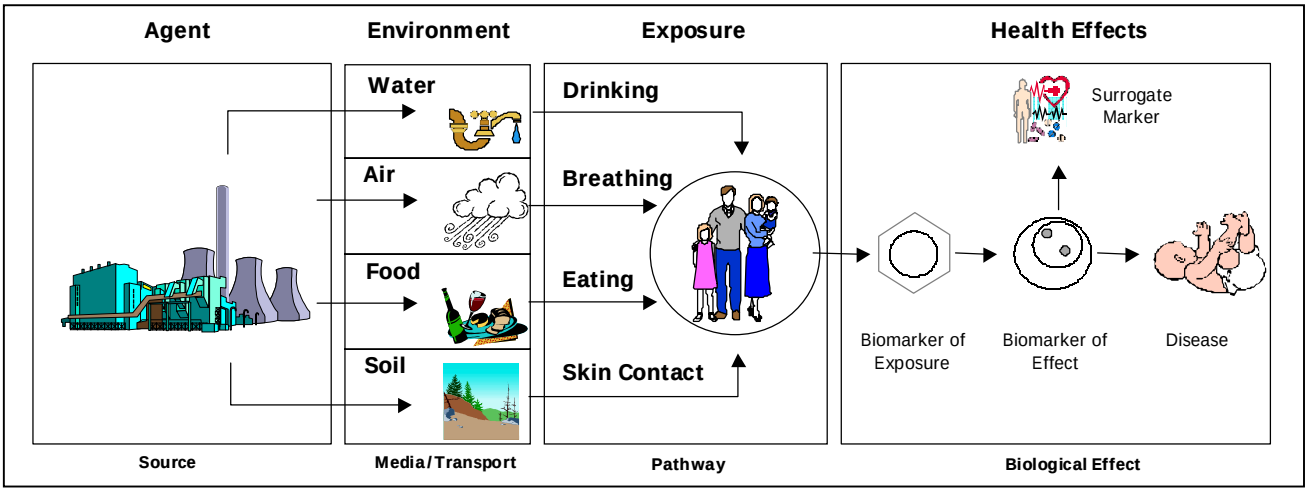
- An understanding of the population and personal distribution of exposure to airborne chemicals and particulates; and
- An understanding of the relative contribution of various exposure sources and pathways to airborne chemicals (i.e., the relative contribution of outdoor and indoor air to the total exposure).

The Grande Prairie and Area Community Exposure and Health Effects Assessment Program was implemented using a scientific methodology and protocol that has evolved over many years and has been proven effective in previous assessment programs.[1]

## 3.0 Background and Rationale

In general, exposure can be defined as any contact between a substance, biological agent, or radiation and an individual or community. We are all exposed to low levels of contamination in the air we breathe, the food we eat, the water we drink, and the consumer products we use. Contaminants can interfere with the normal biological functions, causing effects ranging from subtle biochemical changes to clinical disease and even death. The concept of a continuum from source of contamination to the final health effect is a basic feature of all contemporary risk models.

**Figure 1: Continuum of Exposure**




Determining the risk posed by environmental contaminants to populations requires knowledge about the following fundamental components:

- source(s) of contaminants;
- transport of agents in the environment;
- exposure of individuals and communities to chemicals;
- dose received by those exposed (biological markers of exposure);
- early biological effects resulting from the dose (biological markers of effect); and
- overt health effects (clinical disease, death).

The output of each component in the chain of events serves as input to the next. The lack of information on any one component thus impairs our ability to make accurate assessments of the associated population health risks. Our knowledge about the source and transport of chemicals and other agents in the ambient environment is increasing as the result of environmental monitoring programs, however, there is a need to integrate these data with information on population exposure, biological markers, and health effects. This is very important in achieving new health-based protection levels.

In dealing with population health outcomes, which may be attributable to long-standing exposures to low-levels of contaminants, we are confronted with the difficult and complex problem of chronic health effects. A number of conditions, such as cancers, disorders of the cardiovascular system, neurological disease, chronic respiratory ailments, and many other diseases, have important environmental, behavioural, social, and genetic links. The causes of these conditions are multi-factorial in nature. Other characteristics, such as multistage development, long induction time, and the absence of information on individual and population exposure, make progress in chronic disease prevention slow and tenuous. In order to be able to address these issues, more than ever, there is a need to look beyond one-time epidemiologic studies.

Environmental health surveillance is a tool which can be used to gather data and information on the health of people for the purpose of tracking and detecting trends and associations among a broad range of environmental and health related variables. The process consists of an on-going, systematic collection, analysis, and interpretation of selected data on health outcomes, environmental quality parameters, and population exposure. In addition, data on behavioural, lifestyle, social, economic, and other confounding variables are also considered.

The Alberta Community Exposure and Health Effects Assessment Program protocol was developed to obtain measures of exposure across the continuum of exposure, including measures of contaminants in the environment, the quantity of contaminants to which an individual is exposed through these sources, and finally biological measures of exposure, effect and disease. Further details regarding the study protocol can be found in the Alberta Oil Sands Community Exposure and Health Effects Assessment Program: Methods Report.

## 4.0 Program Objectives

The Grande Prairie Community Exposure and Health Effects Assessment Program's primary objectives were to:

- Describe the population and personal distribution of exposure to airborne chemicals and particulates through:
  - estimation of the population distribution of selected airborne chemicals and particulates; and
  - characterization of the personal variation of exposure as a function of individual activity patterns.
- Quantify the relative contribution of indoor and outdoor air on personal exposure.
- Describe associations between exposure to airborne chemicals and human health effects by analyzing the occurrence of relationships between selected exposures, biomarkers and health outcomes.

## 5.0 Study Method and Protocol

### 5.1 Sample Selection and Recruitment

Data were collected during a period of approximately 28 weeks (March 24 to October 4, 2000) with an average rate of six individuals per week, for a total sample of approximately 130 individuals. Unpaid volunteers between the ages of 18 and 65 years were recruited from the community of Grande Prairie and surrounding area in the Mistahia Regional Health Authority.

Participation in the study places significant demands on the participants. As a result, the protocol recommends that recruitment of volunteers is preferable to a complex sampling design that would require participants to be solicited for participation. Considerations included the following:

- Participation rates would be expected to be so low as to defeat the purpose of a complex sampling design;
- Self-selection biases are not likely to affect exposure rates; and
- Cost would be substantially reduced.

Participants were recruited through advertising in various local media and through active recruitment at various industries, educational institutions, recreational facilities, and other public venues.

Children (<18 yr.) were excluded from the study sample for the following reasons:

- very young children cannot carry the personal exposure air monitors;
- children might not be able to provide reliable time-activity data;

- ingestion may be an important route of exposure to particulates for children that could not be evaluated within the parameters of the study;
- children are likely to have higher exposures to particles and chemical constituents than adults because of their activity patterns; and
- older children who could carry the monitor might be less likely than adults to wear it because it would interfere with normal activities.

## 5.2 Study Design

Several countries as well as the World Health Organization (WHO) are implementing exposure and health effects assessment approaches to address human health concerns related to environmental and other (e.g., occupational) factors. The Alberta Community Exposure and Health Effects Assessment Program is a complete study protocol that was designed to ensure that the results of exposure assessments conducted in Alberta are comparable. This approach provides information for comparison purposes and contributes toward a province-wide source of information on personal exposure measures. The protocol is modeled after an approach to exposure assessment developed by the US Environmental Protection Agency known as the TEAM approach.[2] The Program was designed to produce baseline population exposure and health outcome data through a population exposure assessment conducted in conjunction with a population health assessment. Previous studies have been completed to develop and test data collection methods for exposure assessment, develop and test data collection methods for the collection of additional data, and examine study logistics. The results of these studies are described in separate reports.[3-8] This report provides the results of the implementation of the Program protocol in the Grande Prairie city and surrounding area.

### *Contaminants Measured*

Data were gathered on the following contaminants:

- Nitrogen dioxide ($NO_2$) – a gas that results from combustion; sources include vehicular exhaust, gas stoves, tobacco smoke, kerosene heaters, wood-burning stoves and fireplaces, and gas pilot lights.
- Sulfur dioxide ($SO_2$) – a gas produced by several industrial processes; sources include vehicles, outdoor air, unvented kerosene heaters, and wood-burning heaters and stoves.
- Ozone ($O_3$) – a gas created through the interaction of hydrocarbons, nitrogen oxides, and sunlight; ozone is primarily found in outdoor air, although sources may also include residential electronic air cleaners, negative ion generators, photocopy machines, deodorizers, germicides, and some aerosol sprays.
- Volatile organic compounds (VOCs) – a number of compounds that are carbon-based vapours and gases, many of which are produced from chemical reactions; sources include air fresheners, moth balls, polyurethane floor finish, synthetic fabrics, furniture polish, latex paint, floor wax and wax strippers, shoe polish, solvents, particle board, floor and carpet adhesives, fluorescent lighting, and tobacco smoke.
- Inhalable particulates – microscopic particles that remain floating in the air and can enter the respiratory system; sources include tobacco smoke, kerosene heaters, home renovations, fabric lint, wood stoves or fireplaces, humidifier deposits, and dander.
- Polycyclic aromatic hydrocarbons (PAHs) – compounds that can be formed by incomplete combustion, some of which exhibit carcinogenic effects in humans; sources include gas flaring, teepee burners, automobile exhaust, and any type of natural (e.g., forest fires) or unnatural

burning; indoor sources of may include fireplaces, tobacco smoke, and any other household smoke sources (e.g., burnt toast).

### *Passive Air Sampling*

All volunteers were required to wear passive sampling air monitors in their personal breathing zone continuously for a 7-day period.[1] The air sampling monitors were analyzed for levels of nitrogen dioxide ($NO_2$), sulfur dioxide ($SO_2$), ozone ($O_3$), and a wide range of volatile organic compounds (VOCs) such as benzene and toluene. Similar air samplers were located inside and outside of participant's homes to provide measures of contaminants in and around their personal living space.

### *Polycyclic Aromatic Hydrocarbon (PAH) Air Sampling*

Approximately 15% (1 in 7 volunteers) of the sample were requested to have polycyclic aromatic hydrocarbon (PAH) monitoring equipment located inside and outside of their homes continuously for a 7-day period to gather data on the levels of these contaminants in and around their personal living space. The monitoring of PAHs is at the pilot stage and is not part of the program. The results of the PAH monitoring is presented in Appendix A: Measuring Exposure to Polycyclic Aromatic Hydrocarbons, A Pilot Study.

### *Additional Data Sources*

In addition to the exposure sampling listed above, all volunteers were requested to complete the following:

- review and sign a consent form outlining the participant's involvement in the study;
- a series of neurocognitive tests;
- two health and exposure related questionnaires, provided to the participants to complete at their convenience during the 7-day period of participation;
- one sample of blood and one 12-hour composite sample of urine; and
- a diary of personal activities throughout the 7-day period of participation.

Table 1 shows the various components and sources of data used for the study.

[1] In the original study protocol, four consecutive 24-hour samples were collected from each volunteer. This was modified in the Grande Prairie study to one continuous, 7-day sample to lower the method detection limit and to accommodate field logistics. A 7-day sample also provided a more representative exposure measure as it spanned both weekday and weekend activities for each volunteer.

**Table 1: Components of the Study**

| Component | Media or Source of Data | Purpose |
|---|---|---|
| Characteristics of the Sample | Vital Statistics<br>Other Demographics | General information to characterize the samples and populations. |
| | Lifestyle behaviours | Sections of the questionnaire identified individual smoking habits, weight, height, nutritional intake, and physical activity levels. |
| | Time Activity Diary | The time activity diary identified potential routes of exposure in daily activities. |
| Exposure Measurement | Personal Exposure Monitors:<br>Passive samplers<br>Particulate/PAH samplers | Measures of the actual exposure levels of each participant during a regular week, using personal, indoor, and outdoor air monitors. Measures of exposure for particulate matter and polycyclic aromatic hydrocarbons (PAHs) were collected for a sub-sample. |
| | Other Sources of Exposure:<br>Household sources<br>Work sources<br>Dietary sources | Sections of the questionnaire identify potential sources in the home and work environments, and identification of potential dietary sources of exposure. |
| Biomarkers of Exposure | Blood | Analysis included measures of cotinine (a metabolite of nicotine), pesticides, and phytoestrogens. |
| | Urine | Analysis included measures of metabolites of the BTEX compounds benzene, toluene, ethylbenzene, m-, p-xylene, and o-xylene). |
| Biomarkers of Effect | Neurocognitive measurement | Neurocognitive tests to determine the potential impact of chronic exposure on neurocognitive functioning. |
| Measures of Health Outcome | Health Care System Records | Records of participant contacts with the health care system in the recent past identify health conditions not captured by the questionnaires. Diagnosis rates were compared to control communities. |
| | Questionnaires | Sections of the questionnaire identified general, occupational, emotional, and psychological health. |
| | | Sections of the questionnaire identified previously diagnosed health problems. |

## 5.3 Study Logistics

### *Science Team*

A science team was established to lead the design and implementation of the program protocol. The science team was responsible for:

- the development of schedules for deployment and collection of samples;
- training field staff including the field co-ordinator and field monitoring teams;
- defining any alterations to the original protocol to address issues unique to the Grande Prairie area; and
- statistical analysis of the data and preparation of the final report.

### *Field Staff*

The field co-ordinator was responsible for selecting and screening participants, booking appointments for the field monitoring teams, maintaining the sampler inventory, and co-ordinating the flow of samplers to the laboratory for analysis. In addition, the field co-ordinator was responsible for co-ordinating the flow of sampling time information and respondent data, ensuring that all aspects of the study are administered to each of the participants, and entering all data electronically into various databases.

The field monitoring teams consisted of two trained personnel who were responsible for placing the samplers in an appropriate location in each participant's home, collecting spent samplers, and recording various sources of data. A multi-day training session was held for the field monitoring teams. Classroom training consisted of a review of the study and the requirements for successful completion. Each team member was required to practice and demonstrate the ability to correctly handle and locate samplers in a participant's home.

### *Field Operations*

Each participant was requested to complete a standard protocol which included participation in all aspects of the program. The protocol requested each volunteer to visit the study office for initial testing. Each participant was required to sign a consent form and requested to provide their Personal Health Number (PHN) before beginning. Additional screening criteria included:

- availability for an interview at the study office to provide the required preliminary information and complete a set of neurocognitive tests; and
- availability that week to allow field monitoring teams to deploy and retrieve the air monitoring equipment at the beginning and end of the 7-day period.

The field co-ordinator explained the study in detail, stressing the requirements of complete participation. Photographs of the monitoring equipment and typical placements were used as part of the explanation. After answering any questions about the study, the co-ordinator gave the participant time to read the consent form. If necessary, the co-ordinator read the consent form to the participant. At the completion of the data collection period, consent forms were separated from the other documents, sorted by identification number, and filed in secured storage. Since these forms contain names and linkages to other data, they were kept separate from other information to assure the confidentiality of respondent information.

After the initial screening was completed, the monitoring team appointment booked, and all forms signed, the participant was required to complete tests of visual acuity and colour-blindness, a respiratory health survey, and a variety of tests of neurocognitive functioning. Two questionnaires that request information about the individual's home, lifestyle, diet, and health were provided to each participant to complete during their participation in the study. The individual was also required to schedule an appointment at the Queen Elizabeth II Hospital laboratory to provide a blood sample and a 12-hour urine sample.

The field monitoring teams deployed air-sampling devices at the participant's home remained in place for the 7-day sampling period. The field monitoring teams retrieved the air monitoring equipment at the end of the sampling period. Each participant was fitted with a minimum of four samplers.

Field monitoring teams operated in pairs to ensure safety and improve accuracy. Each team received a list of participants who had completed the initial testing phase described above and the appointment times. The teams were responsible for contacting the participant at the previously arranged appointment time to place the samplers in the home and on the individual. On arrival, the monitoring teams provided details about the equipment being placed in the home and explained what to do if there were problems with the

equipment. The monitoring teams also provided additional details about the time activity diary that the participant were requested to complete: participants were asked to record their activities throughout the 7-day sampling period. At the conclusion of the 7-day period, the field monitoring teams reviewed and collected the diaries and answered any final questions.

### *Data Entry and Analysis*

All information collected by the field staff was returned to the study office at the end of the day. The field co-ordinator reviewed it to verify completeness and, if necessary, follow-up with the participant to complete any missing information. Data was entered by the field co-ordinator. The field co-ordinator then sent the electronic and paper files to Alberta Health and Wellness where a database co-ordinator verified data entry and cleaned records. Once data entry was completely verified, the electronic files were compiled and merged as necessary into a database for analysis. All data components were made identifiable by the arbitrarily assigned participant identification number only; other identifiable information was stripped from the records to ensure confidentiality of the results. Data analysis was then conducted by the science team at Alberta Health and Wellness offices using SAS and SPSS statistical packages.

## 5.4 Exposure Monitoring Procedures

The field-monitoring protocol was designed to collect sufficient samples to characterize the exposure of a representative population to nitrogen dioxide ($NO_2$), sulfur dioxide ($SO_2$), ozone ($O_3$), volatile organic compounds (VOCs), and inhalable particulates (up to 2.5µm in aerodynamic diameter). Each compound of interest was monitored for a 7-day period in three locations: personal (in the participant's breathing zone), indoors (in an appropriate location inside the participant's home), and outdoors (in an appropriate location outside the participant's home). To enhance quality assurance and quality control procedures the field teams also deployed "blanks", or unexposed samplers. Blanks were handled and analyzed in an identical manner as the other air monitors, but, unlike the other monitors, they were not exposed to the environment.

Meteorological data was obtained from Environment Canada. The measurements regularly taken included wind speed, wind direction, temperature, and relative humidity.

### *Monitoring Equipment*

#### *Passive Air Monitors*

Nitrogen Dioxide ($NO_2$): A passive air monitor was used for measuring nitrogen dioxide. The clip-on air monitor contains a chemical adsorbent that collects nitrogen dioxide indicators by passive diffusion.

Sulfur Dioxide ($SO_2$): A passive air monitor was used for measuring sulfur dioxide. The clip-on air monitor contains a chemical adsorbent that collects sulfur dioxide indicators by passive diffusion.

Ozone ($O_3$): A passive air monitor was used for measuring ozone. The clip-on air monitor contains a chemical adsorbent that collects ozone indicators by passive diffusion.

Volatile Organic Compounds (VOCs): A passive air monitor was used for measuring a variety of VOCs. The clip-on air monitor contains a chemical adsorbent that collects various VOCs by passive diffusion.

All four passive air monitors were designed to be worn in the participant's breathing zone to measure personal exposure. The participants were encouraged to continue normal activities while wearing the monitor. During activities such as sleeping or showering, however, the sampler was to be kept as near to the person as practical while protecting the sampler from damage and high humidity environments.

One of each type of sampler was deployed inside and outside the participant's home using a stationary stands constructed to house and shelter the monitors during the 7-day exposure period. The air monitors were attached to identically constructed indoor and outdoor stationary stands approximately one (1) metre above the floor or ground. The outdoor passive air monitoring stand has a rain shield approximately 30cm in diameter for shelter.

The method detection limits (MDL) of the passive samplers were based on field blanks and the limit of quantitation of the laboratory analysis. The detection limits for VOCs were based on the laboratory limit of quantitation (150 ng/sampler) when more than 90% of the field banks were less than the limit of quantitation and are indicated by an asterisk in the table. For the other compounds, quantifiable field blanks the detection limit was based on three standard deviations of the field blank levels and may vary slightly between the batches of samplers through the study. The average detection limits over the study for the compounds investigated (assuming a seven-day sample) are listed in the third column of Table 2. Columns 4 to 6 in the table show the fraction of the measurements that were above the detection limit. Measurements below the detection limits remain useful in characterizing community exposures.

**Table 2: Summary of Passive Sampler Detection Limits**

| Sampler Compound | Sample Rate (ml/min) | Detection Limit ($ug/m^3$) | Fraction of samples less than MDL | | |
|---|---|---|---|---|---|
| | | | Personal | Indoor | Outdoor |
| $NO_2$ | 120 | 2.1 | 0% | 0% | 25% |
| $SO_2$ | 218 | 1.1 | 80% | 90% | 60% |
| $O_3$ | 24.5 | 0.82 | 10% | 25% | 0% |
| HEXANE | 32 | 2.2 | 25% | 60% | 100% |
| BUTANONE | 36.3 | 8.5 | 85% | 85% | 100% |
| METHYLHEXANE | 28.9 | 0.51 * | 10% | 40% | 90% |
| BENZENE | 35.5 | 0.42 * | 5% | 15% | 35% |
| HEPTANE | 28.9 | 0.51 * | 5% | 10% | 85% |
| TOLUENE | 31.4 | 2.6 | 5% | 5% | 40% |
| OCTANE | 26.6 | 0.56 * | 30% | 50% | 100% |
| ETHYLBENZENE | 27.3 | 0.55 * | 10% | 25% | 90% |
| M-P-XYLENE | 27.3 | 1.0 | 0% | 10% | 50% |
| O-XYLENE | 27.3 | 0.55 * | 10% | 20% | 75% |
| NONANE | 24.6 | 0.60 * | 25% | 55% | 95% |
| DECANE | 23.1 | 0.64 * | 15% | 30% | 95% |
| LIMONENE | 30 | 0.50 * | 0% | 0% | 95% |

* Detection limit based on laboratory limit of quantitation (150 ng/sampler) assuming 7-day sample period.

*Active Air Monitors*

Particulates/PAHs: For measurement of respirable particulates from indoor and outdoor environments at the participant's home, the stationary indoor and outdoor air particulate pumps were used to house particulate sampling heads and filters. The particulate sampling heads were oriented in a position that avoided particle deposition due to gravity and were attached to the particulate pumps approximately one (1) metre above the floor or ground. Before and after exposure monitoring, the particulate filters were weighed, and the information was recorded along with the filter identification numbers for analysis purposes after the 7-day exposure period.

***Sampler Placement Strategy***

All sampler locations were determined during the initial visit to each home. Locations were selected after carefully determining the layout of the home, based on the daily habits of the participant, the type of dwelling (home, apartment, etc.), and the outside layout of the yard or grounds. Samplers were placed in the main living area of the participant (the room in which the participant spends the most time while awake), ensuring that the samplers were at least two metres away from exterior doors, windows, and ventilation registers.

The protocol specifies that the participant's backyard is the preferred location for outdoor sampling and that the monitors should not be located within one metre of trees and bushes or within five metres of any type of air vent. For second floor apartments, a "yardarm" was deployed from a window or balcony to support the sampling devices. If a yardarm was not possible, the protocol considers collection of samples at ground level acceptable for second floor apartments. Non-ideal situations required some reasonable compromises, but were identified by the field teams for consideration during data analysis.

## 5.5 Neurocognitive Functioning

Participants were requested to complete a series of computerized neurobehavioral tests using the Neurobehavioral Evaluation System (NES2)[9] software installed on an IBM compatible computer. Participants were informed that they could stop and ask questions or, if absolutely necessary, leave the premises at any time, and they should not feel pressured to continue to respond. Prior to completing the series of neurobehavioral tests, subjects were given a brief explanation of how they were expected to respond (e.g., what keys to use) and were introduced to the joystick (required for some of the tests). Subjects were also given visual tests to ensure normal visual acuity and colour vision (required for the colour-word test). A pre-test questionnaire was completed to identify the subject's general well-being and current health status.

The NES2 tests administered included: finger tapping test; hand-eye co-ordination task; simple reaction time test; continuous performance test; pattern comparison test; symbol-digit substitution test; pattern memory test; serial digit learning test; associate learning test; associate learning delayed recognition test; vocabulary test; switching attention test; colour-word test; and mood test. The auditory digit span from the Wechsler Memory Scale (WMS-R) and the Neuropsychological Impairment Scale were added to provide a non-automated and non-visual activity. These activities were administered by a trained interviewer.

## 5.6 Questionnaires

Two questionnaires were given to each participant following the completion of the neurocognitive functioning tests for completion at their convenience during the 7-day testing period. The first questionnaire, the Demographic and Exposure Questionnaire, was designed to collect information about

participant demographics, occupational health, and their work and home environments, including potential sources of contaminants. This questionnaire also includes all of the questions included on the Basic Standard Environmental Inventory Questionnaire, designed to help classify relative concentration estimates.[10]

The second questionnaire, The Health and Nutrition Questionnaire, was designed to collect a variety of health indicators, including mental and physical health, physical activity levels, and nutritional intake. The questions on nutrition attempt to characterize actual nutrition levels using the amounts dictated by the Canadian Food Guidelines. Two standardized scales of general health were included in The Health and Nutrition Survey: the General Health Questionnaire (GHQ) and the Short-Form-60 Health Survey (SF-60). Both questionnaires are well validated and documented tools for assessing health. The GHQ assesses psychological well-being, and the SF-60 assesses physical functioning, role limitations, bodily pain, social functioning, general mental health, vitality, and general perceptions. Additional measures from the National Population Health Survey, conducted by Statistics Canada, were also included to provide information about physical activity level.

### 5.7 Biological Tests

A laboratory technologist from the Queen Elizabeth II Hospital extracted a sample of each participant's blood for testing by the laboratory to identify biomarkers of exposure. The participant was also requested to submit a 12-hour urine sample. Biological samples were generally obtained during the final day of the sampling period.

### 5.8 Health Records Analysis

All participants were requested to provide a Personal Health Number, and give written consent for its use in retrieving administrative information for use in the evaluation. The two primary data sources used for analysis were hospital discharge summaries and physician billing claims. This data was used to identify health status variables to compare with the rates of diagnosis for other communities.

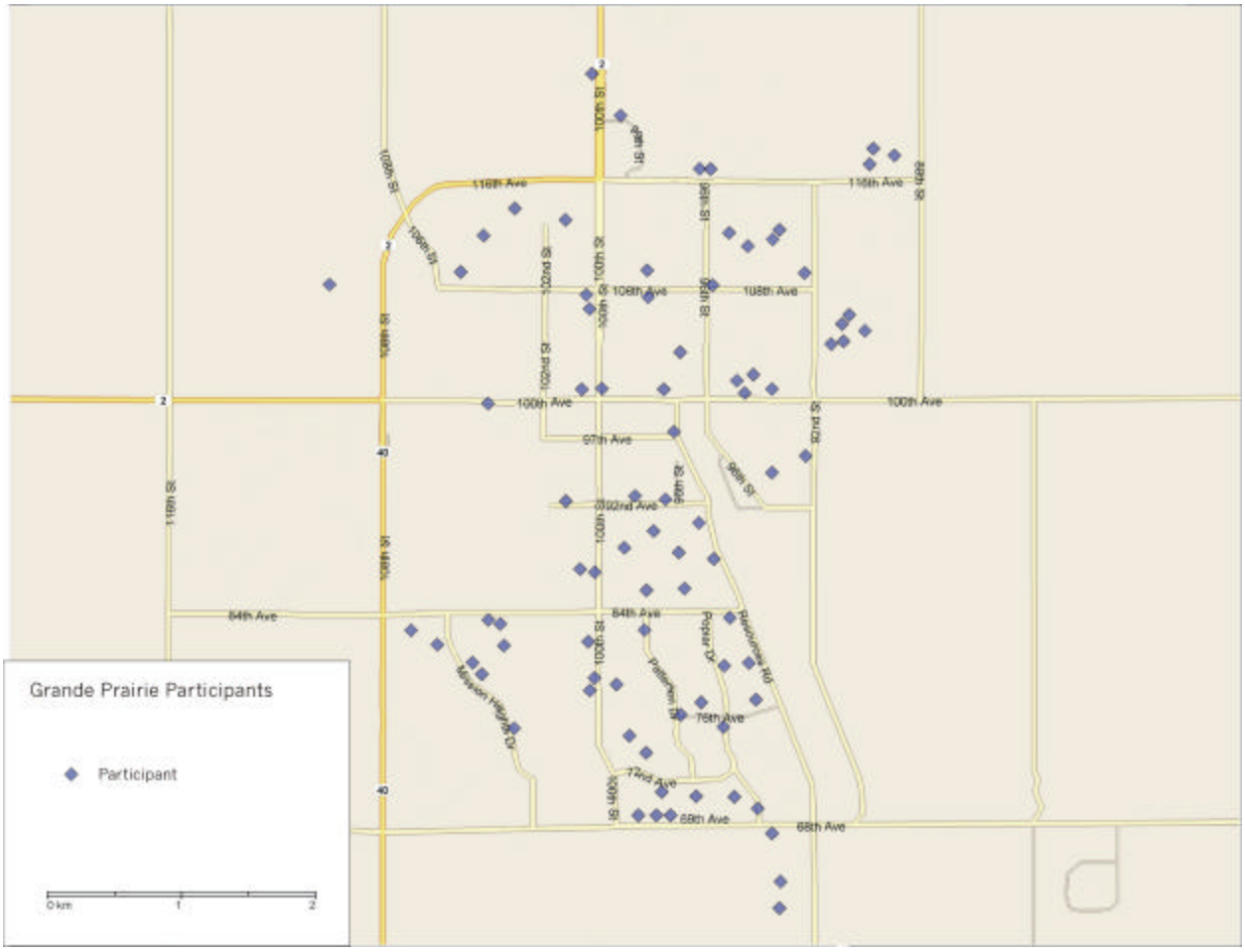
Age-adjusted incidence rate ratios were computed to compare the observed rate for the area (i.e., enumeration area) with a baseline rate (i.e., overall rate for the community). Disease incidence rate ratios use a categorical scale: higher, high, average, low, and lower.

Disease rates were computed for the Grande Prairie community and surrounding areas. Graphs of age adjusted incidence rates are used to represent disease trends over the time period, because a pattern often becomes evident when disease rates are calculated over longer periods of time, after adjusting for various confounding factors such as changes in age structure.

## 6.0 Characteristics of the Sample

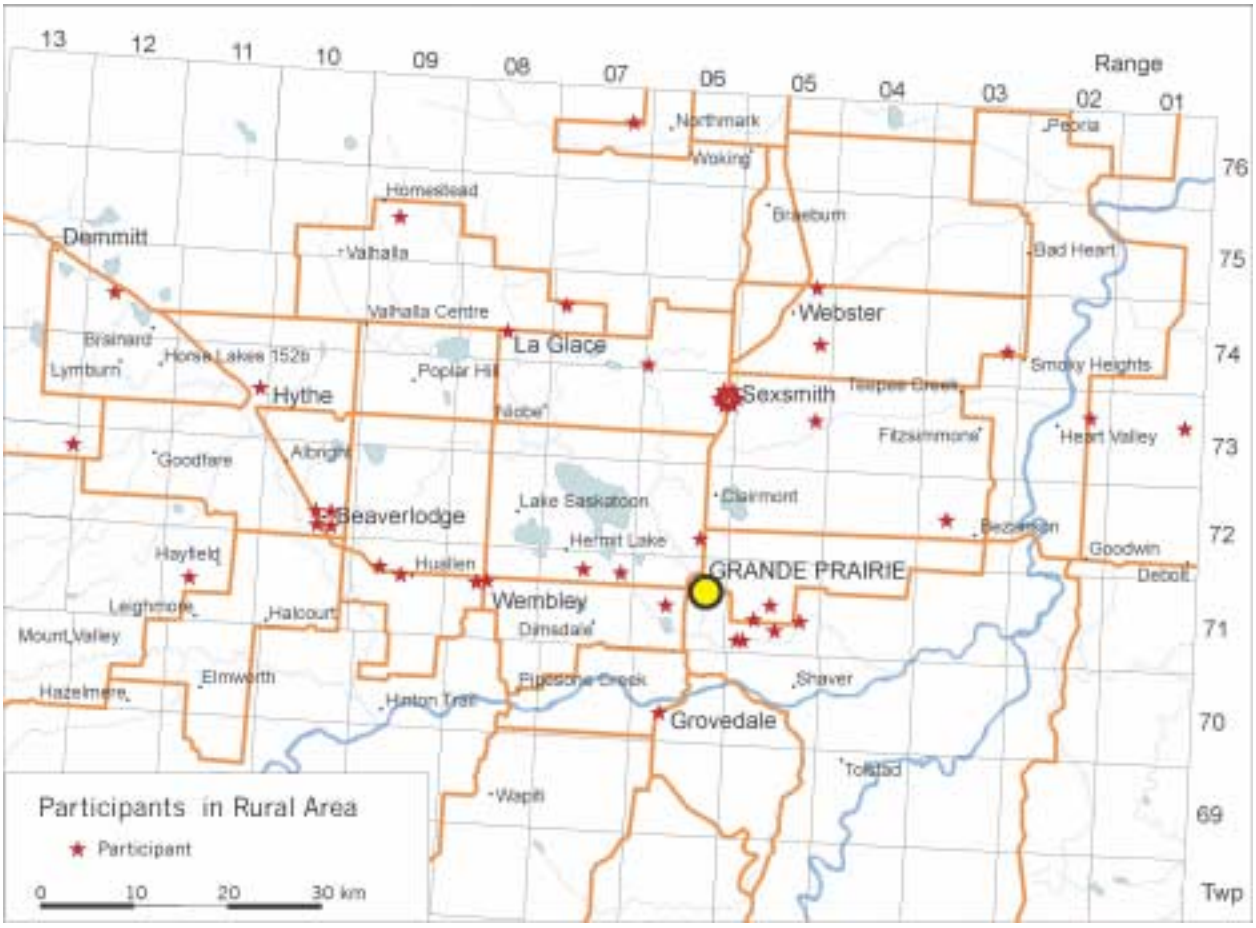
The Grande Prairie Community Exposure and Health Effects Assessment Program included 140 residents of Grande Prairie and the surrounding area. All participants were at least 18 years of age. Map 1 shows the distribution of the participants in the city of Grande Prairie, and Map 2 shows the distribution of the participants living outside the city limits. Samples of exposure were obtained from all areas of the city.

**Figure 2: Distribution of Urban Participants**




*Note: Locations of residences have been slightly randomized to protect confidentiality of participants.*

**Figure 3: Distribution of Rural Participants**




## 6.1 Sample Size

The protocol recommended a minimum sample size of 150 participants. Obtaining volunteers was difficult and the optimum sample size was not obtained, despite an aggressive recruitment campaign. A total of 140 people volunteered to participate in the assessment, but only 121 participants provided enough of the required information to be included in all analyses. Table 3 shows the number of participants who completed various components of the study.

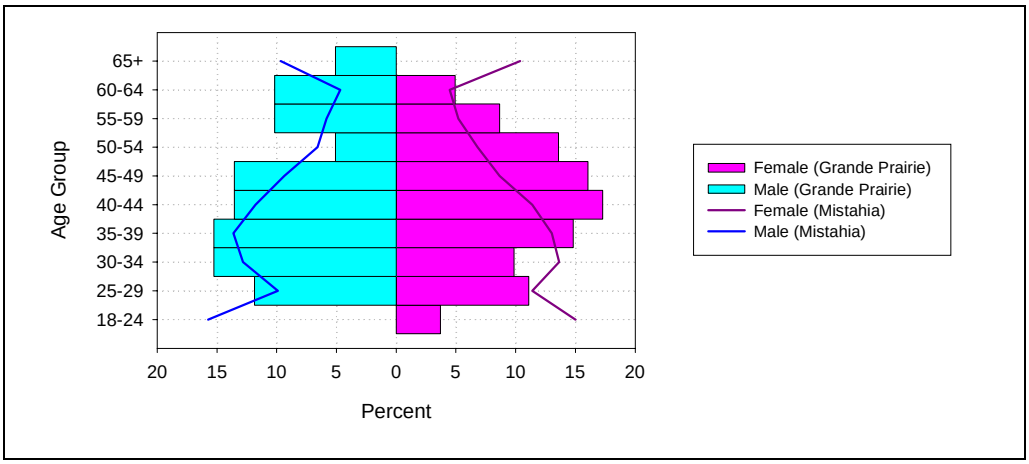
**Table 3: Number of Participants Completing Each Study Component**

| Study Component | Number of Participants |
|---|---|
| Total number in study | 140 |
| Passive exposure assessment | 132 |
| Particulate exposure assessment | 39 |
| Completed demographic questionnaire | 125 |
| Completed health questionnaire | 121 |
| Completed neurocognitive assessment | 135 |
| Completed time-activity diary | 133 |
| **Total with Complete Data** | **121** |

## 6.2 Age and Gender

The average age of the sample was 43.2 years (N = 140; SD = 11.37). More than half of the sample were female (57.9%) and the average age of the women (42 years) was younger than the average age of the men (44 years) included in the sample. Figure 4 shows the age and gender distribution of the sample compared to the age and gender distribution of the population of the Mistahia Health Region.

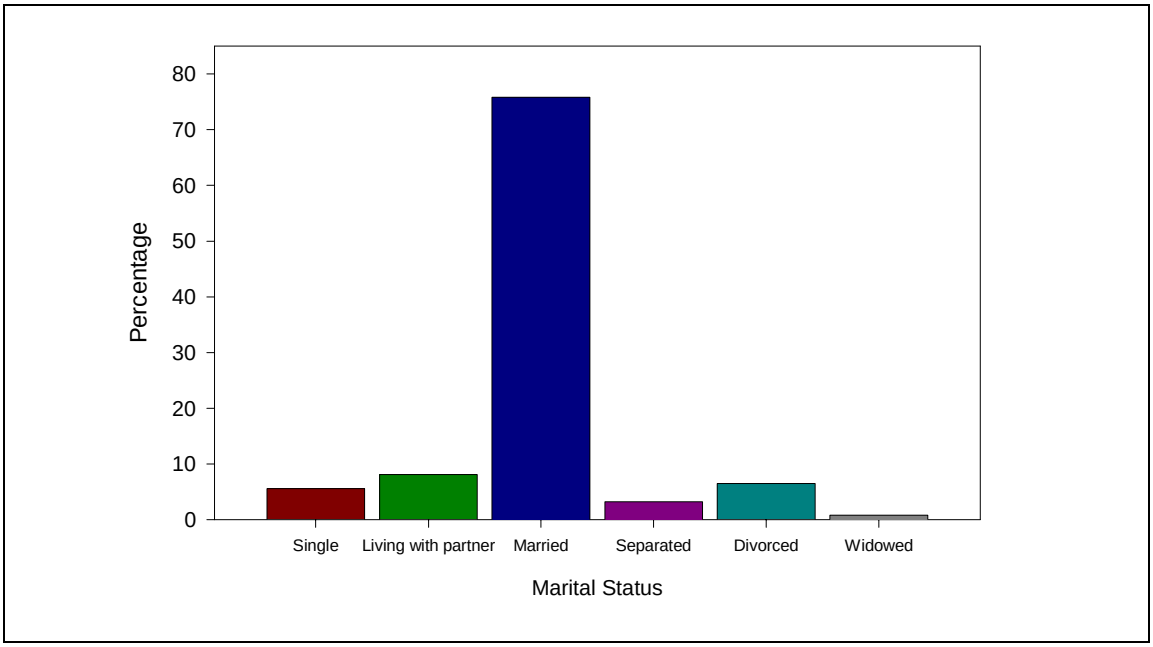
**Figure 4: Age and Gender Distribution**



### 6.3 Marital Status

Participants in the study were asked about their marital status. The majority of participants were either currently married (75.8%) or living with a partner (8.1%). Only 6.5% of the sample were divorced, 5.6% were single, 3.2% were separated, and 0.8% were widowed.

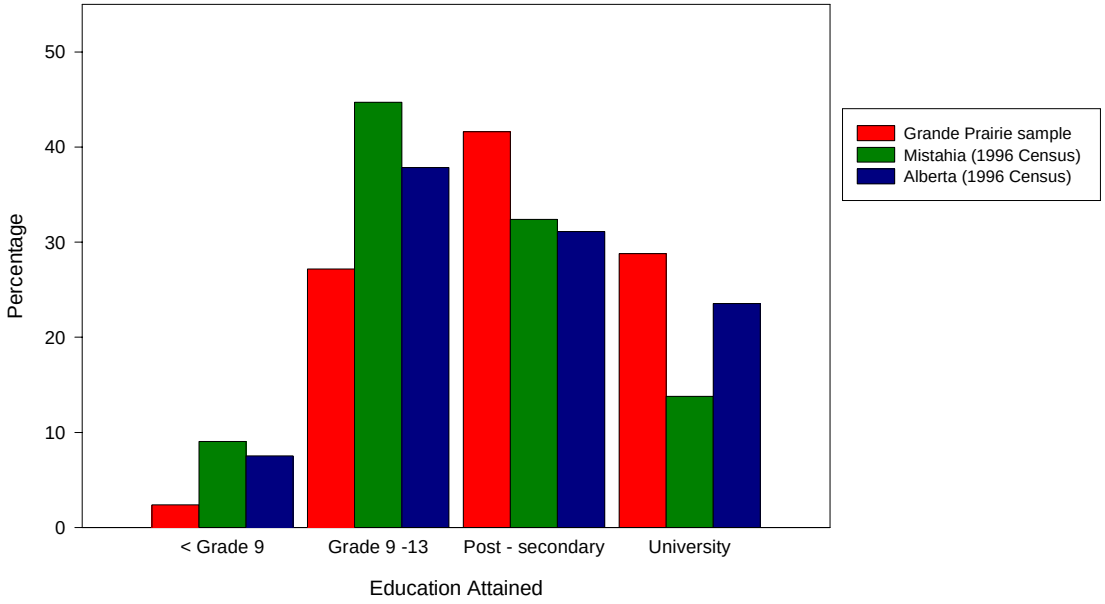
**Figure 5: Marital Status**




### 6.4 Education

Figure 6 compares the levels of education for the Grande Prairie sample population with the population living in the Mistahia Health Region area and with the province of Alberta. The average number of years of education reported by the Grande Prairie sample was 15.7 years (N = 125; SD = 4.63). Over half of the Grande Prairie sample had completed at least one year of education at the university. The Grande Prairie sample population had a higher level of education compared to the population living in the Mistahia Health Region and compared to the overall Alberta population estimated level of education.

**Figure 6: Education Level**




## 6.5 Language

English was indicated as the native language of 88.6% of the Grande Prairie sample. In the 1996 census, 88.0% of the Grande Prairie inhabitants specified English as their mother tongue, which is equivalent to our Grande Prairie sample. The Grande Prairie rate of 88.6% was also quite comparable to the 1996 census rate of 87.7% for the Mistahia Health Region.

## 6.6 Occupation

A majority of the participants indicated that they were currently employed by a health organization (22.6%). The next greatest number were employed by an educational organization (14.0%). Table 4 shows the participants' primary employment status and whether this employment was full- or part-time.

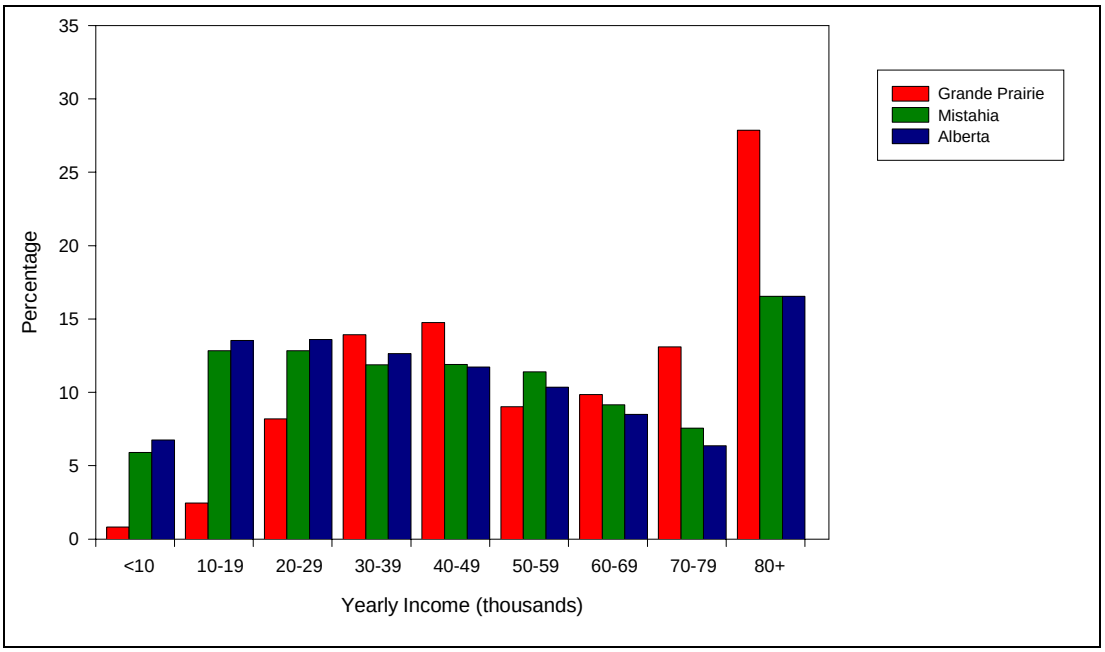
**Table 4: Primary Work or Employment Status**

| | Percentage of Sample (N = 120) | |
|---|---|---|
| Have a paid job outside of home | 72.4% | 79.5% full time |
| | | 19.3% part time |
| Self-employed in home | 8.9% | 11.1% full time |
| | | 66.7% part time |
| Student | 0.8% | 100% full time |
| | | 0.0% part time |
| Full-time homemaker | 8.1% | |
| Currently unemployed | 0.8% | |
| Retired or disabled | 8.1% | |
| Other | 0.8% | |

## 6.7 Income

Over half (59.0%) of the participants indicated their annual household income to be less than $70,000. Figure 7 displays the distribution of household income for the Grande Prairie sample population as well as for the Mistahia Regional Health Authority (RHA) and for the province of Alberta. As is confirmed by the Grande Prairie sample population data, the percentage of households making at least $80,000 annual income is about twice that of both the Mistahia Health Region (RHA #13) and Alberta as a whole. The RHA average is similar to the provincial average.

**Figure 7: Distribution of Household Income**



### 6.8 Smoking

Of the Grande Prairie respondents, 42.4% indicated they had smoked as much as one cigarette a day for as long as one year. Whether the participants currently smoked or not, when they did smoke they smoked between one (1) to over forty (>40) cigarettes per day. At the time of the study, 6.4% of the respondents indicated that they currently smoke.

The majority of Grande Prairie (74.2%) respondents indicated that they worked in a non-smoking environment; however, the average daily exposure to cigarette smoke (second-hand smoke) varied greatly across participants, from no exposure to as much as 960 minutes (or 16 hours) per day.

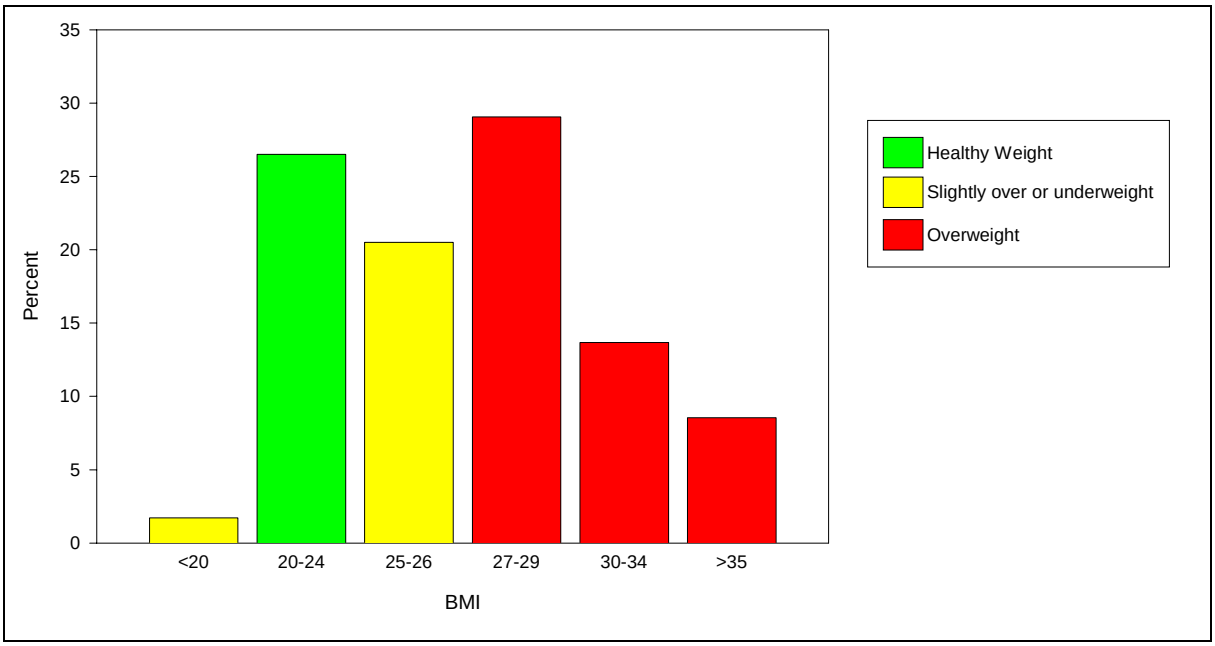
The Mistahia Regional Health Needs Assessment found that smoking prevalence in the Mistahia Health Region was higher than the Canadian average for females in the youngest age group (15 to 19), and for males between the ages of 20 and 29 and between the ages of 40 to 59.[11] A prevalence study showed that only 27% of regional residents currently smoked daily, almost the same as the Canadian average of 25%.[12] Smokers were less likely to volunteer for the study as evidenced by the low smoking rate in the study sample compared to the rate found in this independent study of the general population.

### 6.9 Body Mass Index

A body mass index (BMI) was calculated from reported height and weight for each participant. The BMI is considered a valid measure of obesity because it correlates well with skinfold and body density measures, and has been adopted in the *Canadian Guidelines for Health Weights.*[13] A BMI of less than 20 indicates that the individual is underweight for their height, and there may be some associated health problems. A BMI between 20 and 24 is considered a healthy range. A BMI of between 25 and 27 indicates that the individual is slightly overweight, which may lead to health problems for some people, while a BMI over 27 indicates an increased risk of health problems associated with weight.

Figure 8 shows the distribution of BMI for the sample population. The average BMI for the Grande Prairie participants was 27.3. Twenty percent (20.5%) of the Grande Prairie participants were slightly overweight (BMI of 25 to 26); 51.3% had a BMI greater than 27. The estimated average BMI for the Canadian population is 25.4, lower than the study population.[14] Fewer study participants had a BMI in the lower or healthy range compared to the Canadian estimates. A larger percentage of study participants (22.2%) had a BMI greater than 30 compared to the Canadian estimates (14.0%).

**Figure 8: Distribution of Body Mass Index**




## 6.10 Nutritional Intake

Participants were asked about their usual dietary habits. The participants indicated that they ate less than the recommended 5 to 12 servings of grain products each day, and that they ate approximately five servings of fruits and vegetables each day, which is the minimum (5-10) recommended number of servings. The average number of servings of milk products corresponded to the recommended number (2 to 3), and the number of servings of meat and alternatives also corresponded to the minimum number of servings recommended by the Canada Food Guide (2 to 3). Respondents indicated that they consumed an average of 2 to 3 servings of sweets or other non-nutritious foods each day. Participants drank an average of two cups of coffee per day, and less than one drink per day of cola or alcohol. Participants also estimated that they consume an average of 8 cups (1.89 L) per day.

## 6.11 Local Wild Food Sources

The frequency of consumption of local wild food sources was recorded because this can indicate whether there are other sources of contaminants or pathways of exposure that are unique to the local population. Eighty-five percent (85.7%) of the Grande Prairie participants indicated that they eat locally grown fruits and vegetables when available. Seventy-four percent (74.8%) of the participants indicated that they ate local wild berries. Consumption of local wild game was not as common as consumption of wild fruits, although 36.4% of the sample population stated that they ate local moose, and 28.1% stated they ate local deer. Grouse (11.6%) was another game animal consumed by a relatively large portion of the Grande Prairie study population. A number of participants (45%) indicated that they ate locally caught fish. Trout was the most frequently mentioned fish in Grande Prairie (33.1%), followed by Pike (19.0%) and Walleye (19.0%).

### 6.12 Sources of Drinking Water

Data was collected on characteristics of household drinking water and personal drinking water habits. Most Grande Prairie respondents indicated their source of tap water as the city water treatment facility (80.0%). Other sources of drinking water included wells (18.4%) and surface water from a dug out (1.6%). Tap water was used for drinking and drink mixes by 79.8% of participants. When drinking water from the tap, only 51.6% indicated that they run the water for a period of time before filling their glass and 22.6% indicated that they sometimes do. About one-third (34.4%) of respondents indicated that they have a filter of some type that purifies the water, most of which were the activated carbon type (e.g., Brita, Amway). Bottled water was used by 27.0% of respondents, and another 34.4% indicated that they sometimes used bottled water. Of those that used bottled water, 44.7% indicated they use it for all drinking, while others limited their use of bottled water to travelling (67.1%), at work or school (48.7%), cooking (7.9%), or other uses (13.2%).

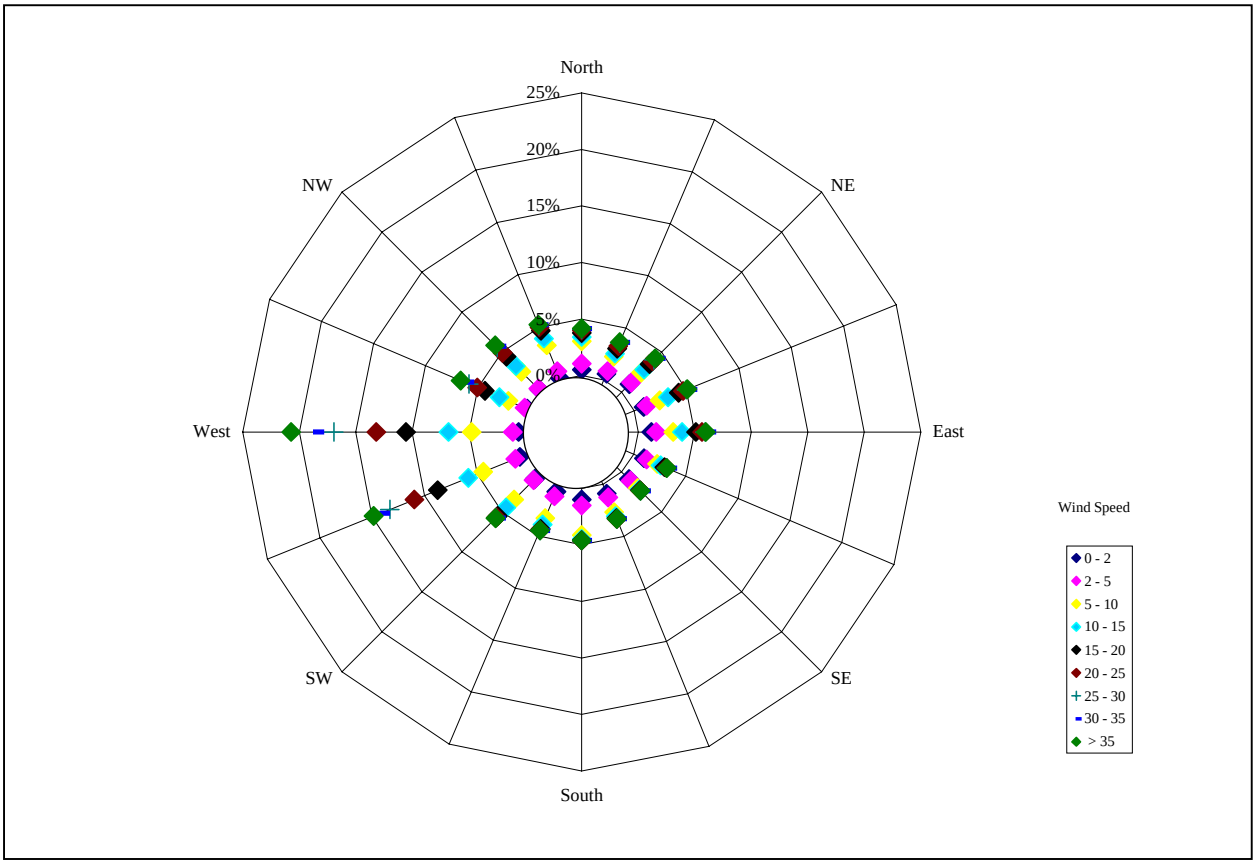
### 6.13 Physical Activity Level

The physical activity section of the Health Habits and Diet Survey assessed participants' involvement in a variety of physical activities. Health Canada recommends at least 20-30 minutes of vigorous activity, or 60 minutes of light effort, every day, to maintain good health.[15] The mean time spent in physical activity per week in the Grande Prairie sample was 3.9 hours indicating that many participants did not meet Health Canada's minimum requirements for physical activity. This also corresponds with the large proportion of participants with a BMI greater than the healthy range.

### 6.14 Meteorological Data

The wind diagram in Figure 9 describes the percent of time the wind blows from various directions and speeds. As the wind diagram shows, during the study period, 40% of the time the wind blew from the west. The figure also shows that winds from the west were usually above 10 km/hr.

**Figure 9: Wind Rose Diagram Showing Wind Characteristics During the Study**




## 6.15 Time Activity Diaries

Participants were asked to record the time spent in activities at various general locations for the duration of their participation. Figure 10 shows the average levels for the participant group as a whole.

There were trade-off relationships among the relative mixes of general activities across different individuals. The primary trade-off involved time spent indoor at home versus time spent in other indoor or outdoor activities; and independently time spent indoors at home versus time spent indoors at work.

Table 5 shows that gender and job status are also a major determinant of the relative activity mix between home and work.

**Figure 10: Average Proportion of Time in a Day**

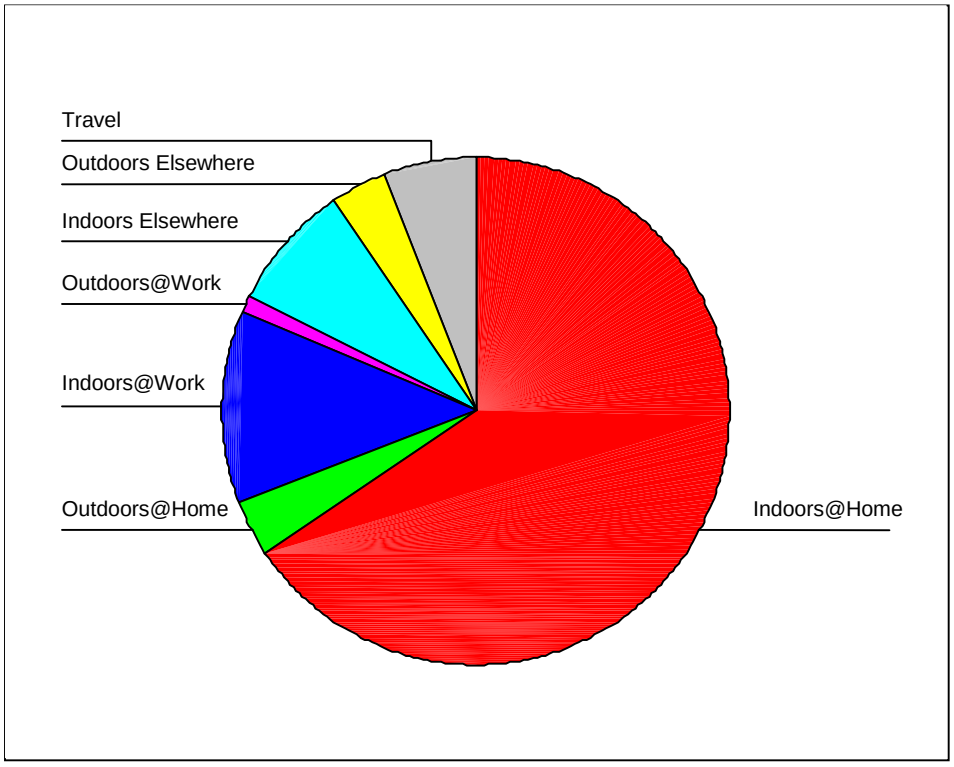



**Table 5: Activity Mix by Gender and Job Status**

| | Indoors at Home (proportion) | Indoors at Work (proportion) |
|---|---|---|
| **Female** | | |
| Not employed | .72 | .01 |
| Part time Job | .71 | .08 |
| Full time Job | .61 | .17 |
| **Male** | | |
| Not employed | .77 | .00 |
| Part time Job | .45 | .10 |
| Full time Job | .54 | .16 |

# 7.0 Air-Borne Contaminants

## 7.1 Passive Samplers

Passive air quality measurements were taken with four separate samplers, each deployed for a one week period. Each participant carried samplers around their neck hanging in their breathing zone (Personal sample), had a sampler deployed inside their home (Indoor sample), and had a sampler deployed in the environment immediately outside their home (Outdoor sample). Table 6 shows the sampler types and the chemicals monitored by each sampler.

**Table 6: Samplers and Chemical Concentrations Measured**

| Sampler | Chemical Concentrations Measured |
|---|---|
| $NO_2$ | Nitrogen Dioxide |
| $SO_2$ | Sulfur Dioxide |
| $O_3$ | Ozone |
| Volatile Organic Compounds | Hexane |
| | 2-butanone |
| | 3-methylhexane |
| | Benzene |
| | Heptane |
| | Toluene |
| | Octane |
| | Ethylbenzene |
| | m-, p-xylene |
| | o-xylene |
| | Nonane |
| | Decane |
| | Limonene |

The Field Teams successfully deployed 2,192 personal exposure monitors (PEMs) throughout the course of the study. Of these, only 28 PEMs had missing data due to lost or missing monitors. Table 7 shows how the remaining 2,164 PEMs were distributed.

**Table 7: Distribution of Personal Exposure Monitors (PEMs)**

| Number by Location | Number by Type |
|---|---|
| 539 Personal | 136 $NO_2$ |
| | 136 $SO_2$ |
| | 135 $O_3$ |
| | 132 VOCs |
| 542 Indoor | 136 $NO_2$ |
| | 137 $SO_2$ |
| | 137 $O_3$ |
| | 132 VOCs |
| 540 Outdoor | 136 $NO_2$ |
| | 136 $SO_2$ |
| | 136 $O_3$ |
| | 132 VOCs |
| 543 Blank | 137 $NO_2$ |
| | 137 $SO_2$ |
| | 137 $O_3$ |
| | 132 VOCs |
| Total | 2,164 |

Calculation of the concentrations of each chemical from the amount of material detected on each sampler filter involved formulae relating sampling rates to concentration levels. In addition, a time correction was applied to correct for the precise amount of time (in minutes) that the samplers were exposed to air. A correction for blank levels (levels measured on unexposed sampler filters) was also applied. This correction itself involved an examination of the variability of the blank values over the course of the study, and for some chemicals resulted in a complex time dependent correction.

In the sections that follow, three graphs are presented to describe the study results for each chemical.

The first graph shows the distribution of all measures taken through the study from the Grande Prairie location for each of the sample types: personal, indoor, and outdoor. The graph plots the calculated average concentration in the air to which the sampler was exposed plotted against the percentile of this exposure level in the particular sample type across all samples collected. The median exposure level is located at the point where a vertical line drawn from the 50th percentile mark on the horizontal axis intersects with the curve. The concentration level at that point is read from the vertical axis by drawing a horizontal line from that point on the curve to the vertical axis. The vertical axis is presented as a logarithmic scale that reflects the general finding of positive skew in distributions of chemicals in air. If the line deviates from a straight line and especially if the curvature is marked at either end (usually the end indicating higher exposure levels), this indicates a skewed distribution of exposure to that chemical more marked than the log normal distribution. The degree of slope in the linear section of the curve is

related to the overall variability of the sample such that steeper slopes indicate more variable distributions. Curves that do not appear to start at percentile 0 indicate that a proportion of cases fell below the blank level for the sampler for that chemical. The proportion of samples for which this is true is determined by noting the percentile level at which the curve begins.

The second graph represents a line of best fit derived by locally weighted regression methods to show the temporal trend in the sampled concentrations for each sample type. The lines appear smooth, but they typically represent a very weak relationship between season and concentration. To illustrate that this relationship is weak, the individual concentrations are plotted on this graph as points. As well, the duration of the sampling (approximately 6 months) restricts the ability to fully determine the shape of any yearly cycle that might be present in the data.

The third graph was designed to give an indication of the degree of relationship between levels of personal exposure and levels of indoor and outdoor concentrations respectively. It is created as follows: first, personal exposure values are ranked from highest to lowest; second, a graph is created which orders the data from highest to lowest (where the concentration is given on the vertical axis, and the order values for each participant are presented along the horizontal axis); third, the values for outdoor and indoor concentrations are plotted at the horizontal point in the graph at which the point indicating the personal concentration for that participant had previously been plotted; fourth, a locally weighted regression line is produced to help visualize the association between personal exposure and indoor exposure and between personal exposure and outdoor exposure. For strong relationships, the interpolated lines for the associated sampler sites will mimic the general downward trend of the line for personal exposure (and at the same time the points will cluster closely around this line). The stronger the relationship, the closer the curves will be to being parallel to each other. Weak or non-existent relationships will be characterized by interpolated lines that are parallel or close to parallel to the horizontal axis. In general, even strong apparent relationships had only moderate correlations (0.4-0.5) between personal exposure and either indoor or outdoor exposure.

### *Nitrogen Dioxide ($NO_2$)*

Figure 11 shows the cumulative distribution of $NO_2$ concentrations for the three types of samplers (personal, indoor, and outdoor). Concentrations measured on the personal samplers were greater than the other sampler locations, but the differences were not large.

The indoor and personal samples were above the method detection limit (MDL) of 2.1 $\mu g/m^3$ while 25% of the outdoor samples were below the detection limit. While the imprecision associated with individual outdoor samples increases dramatically when measures fall below the detection limit, the data provides a prediction of overall community exposure.

The median and $95^{th}$ percentile $NO_2$ levels ($\mu g/m^3$) for the different locations are summarized and compared to guidelines and levels in other communities in Table 8. In addition, the relative levels of $NO_2$ at the locations are compared by the ratios of personal to indoor (P/I), personal to outdoor (P/O), and indoor to outdoor (I/O). The indoor and outdoor levels of $NO_2$ were an order of magnitude below guideline levels and were similar to levels found in other relevant studies.

**Figure 11: Distribution of Nitrogen Dioxide**

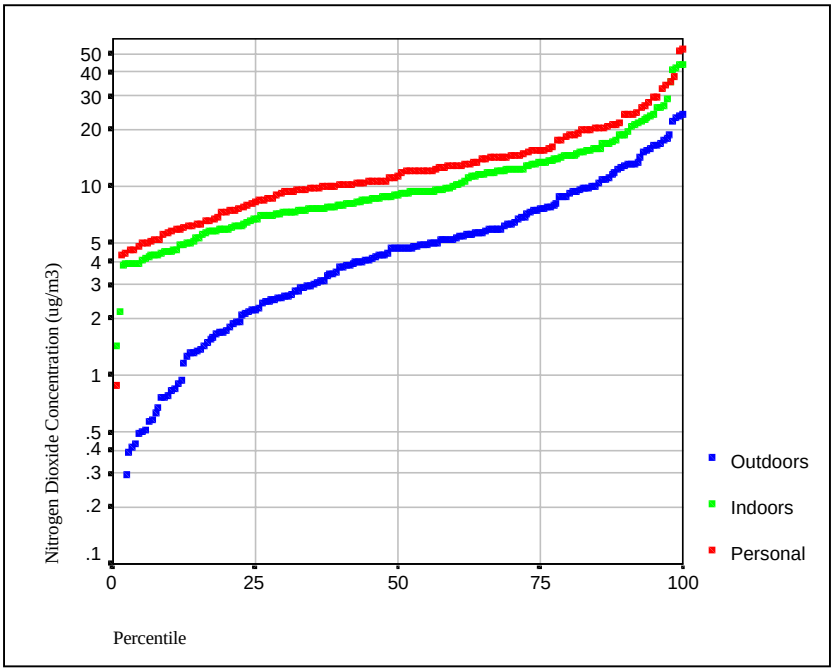



**Table 8: Comparison of $NO_2$ Levels in µg/m$^3$ with Guidelines and Other Studies[16, 17, 18]**

| Parameter | G.P. Median | G.P. 95th | Ft. Mc. Median | Ft. Mc. 95th | Leth. Median | Leth. 95th | Relevant Studies | Guideline/ Reference Level |
|---|---|---|---|---|---|---|---|---|
| Personal | 11.6 | 30.2 | 15.9 | 53.2 | 17.7 | 41.6 | N/A | N/A |
| Indoor | 9.1 | 25.8 | 8.6 | 30.0 | 9.8 | 30.3 | 6* | 100 (long term) 480 (hour)*** |
| Outdoor | 4.7 | 16.5 | 9.5 | 38.5 | 13.8 | 42.8 | 12* | 200 (day) AENV |
| P/I ratio | 1.3 | 1.1 | 10.8 | 36 | N/A | N/A | N/A | N/A |
| P/O ratio | 2.5 | 1.8 | 1.9 | 1.8 | 1.8 | 1.4 | N/A | N/A |
| I/O ratio | 1.9 | 1.5 | 1.7 | 1.4 | 1.3 | 1.0 | 0.65* | N/A |
| | | | 0.90 | 0.78 | 0.71 | 0.71 | | |

* Hagenbjork-Gustafsson et al., 1996.
** Spengler et al., 1983.
*** Health Canada, 1989.

Figure 12 shows smoothed curves (produced by locally weighted regression) to represent the temporal trend in $NO_2$ concentrations. Outdoor concentrations appear to increase in the fall months. Personal concentration measures were greater than both indoor and outdoor levels of $NO_2$.

**Figure 12: Temporal trend in Nitrogen Dioxide Concentration**

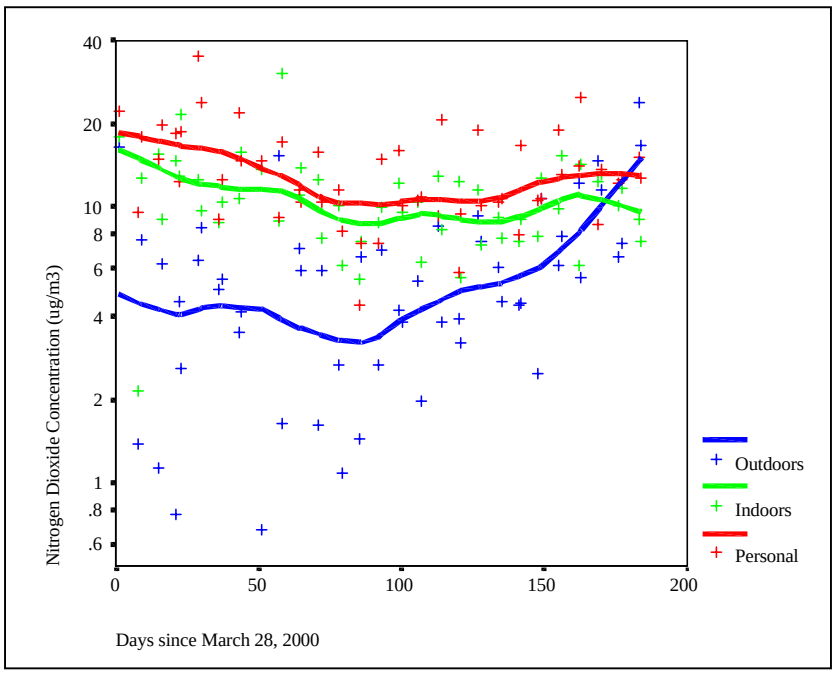
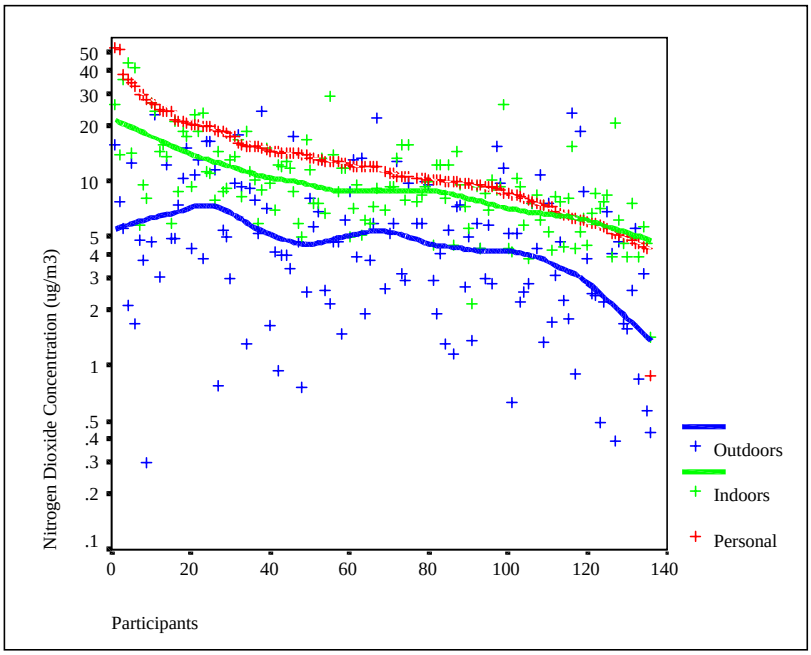



Figure 13 shows the relationship between the $NO_2$ concentrations monitored personally, indoors and outdoors. The graph shows the ordered personal exposure levels for each subject in the study, and their corresponding levels of indoor and outdoor concentration levels. A locally weighted regression curve has been added for indoor and outdoor concentration levels to give an indication of the strength of the association between personal levels and indoor and outdoor levels respectively. A horizontal line would show no relationship, while positive associations would be shown by sloped lines (and particularly by the relative degree of scatter of the individual points around those lines). This graph shows high relationships between measures of indoor and personal concentrations, and moderate relationships between measures of outdoor and personal concentrations.

**Figure 13: Relationship between Exposures to Nitrogen Dioxide by Sampler Site**




### *Sulfur Dioxide ($SO_2$)*

Figure 14 shows the cumulative distribution of $SO_2$ concentrations for the three types of samplers (personal, indoor, and outdoor). The median outdoor concentrations were approximately double the personal concentrations.

Levels of $SO_2$ were highest in outdoor air and lowest in home indoor environments. Overall, levels were low with between 60% to 80% of the samples collected falling below the MDL of 1.1 $\mu g/m^3$.

The median and 95$^{th}$ percentile $SO_2$ levels ($\mu g/m^3$) for the different locations are summarized in Table 9 and compared to guidelines and levels in other communities. The levels of $SO_2$ measured in Grande Prairie were much lower than guidelines.

**Figure 14: Distribution of Sulfur Dioxide**

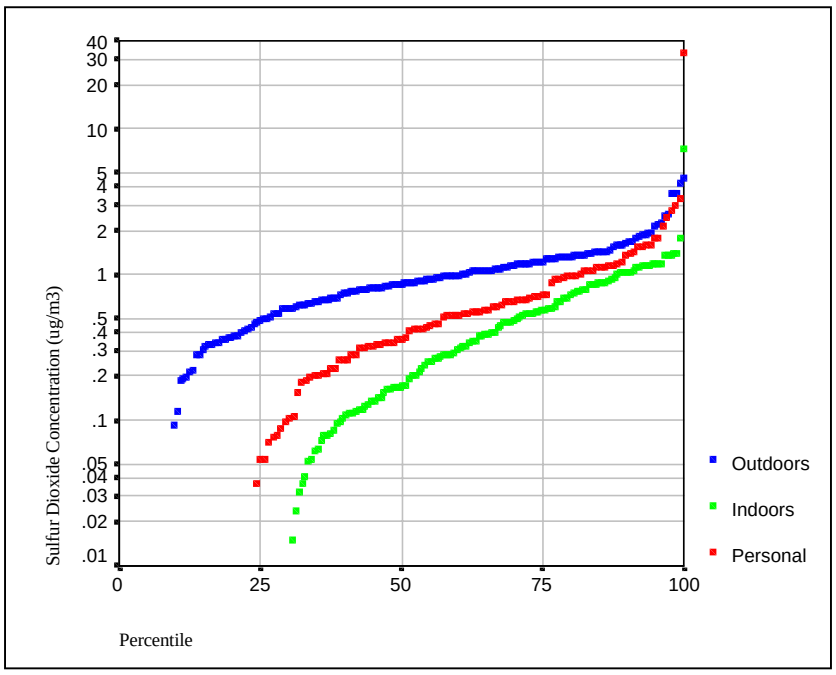



**Table 9: Comparison of $SO_2$ Levels in µg/m$^3$ with Guidelines and Other Studies[19]**

| Parameter | G.P. Median | G.P. 95th | Ft. Mc. Median | Ft. Mc. 95th | Leth. Median | Leth. 95th | Relevant Studies | Guideline/ Reference Level |
|---|---|---|---|---|---|---|---|---|
| Personal | 0.37 | 1.83 | 0.87 | 5.6 | 0.21 | 3.1 | N/A | N/A |
| Indoor | 0.17 | 1.18 | 0.41 | 4.1 | 0.16 | 2.9 | N/A | 50 (long term)<br>1000 (5 minutes)* |
| Outdoor | 0.86 | 2.23 | 1.6 | 8.0 | 1.1 | 5.2 | N/A | 150 (day) AENV |
| Ambient Station | | | 2 | 6.5 | N/A | N/A | N/A | 445 (hour), 157 (day), 26 (year) AEP<br>39-60 (year) EC<br>340 (hour) WHO |
| P/I ratio | 2.14 | 1.54 | 2.1 | 1.4 | 1.3 | 1.1 | N/A | N/A |
| P/O ratio | 0.42 | 0.82 | 0.53 | 0.70 | 0.19 | 0.59 | N/A | N/A |
| I/O ratio | 0.20 | 0.53 | 0.25 | 0.52 | 0.15 | 0.56 | N/A | N/A |

* Health Canada, 1989.

Figure 15 shows smoothed curves (produced by locally weighted regression) to represent the temporal trend in $SO_2$ concentrations.

**Figure 15: Temporal Trend in Sulfur Dioxide Concentration**

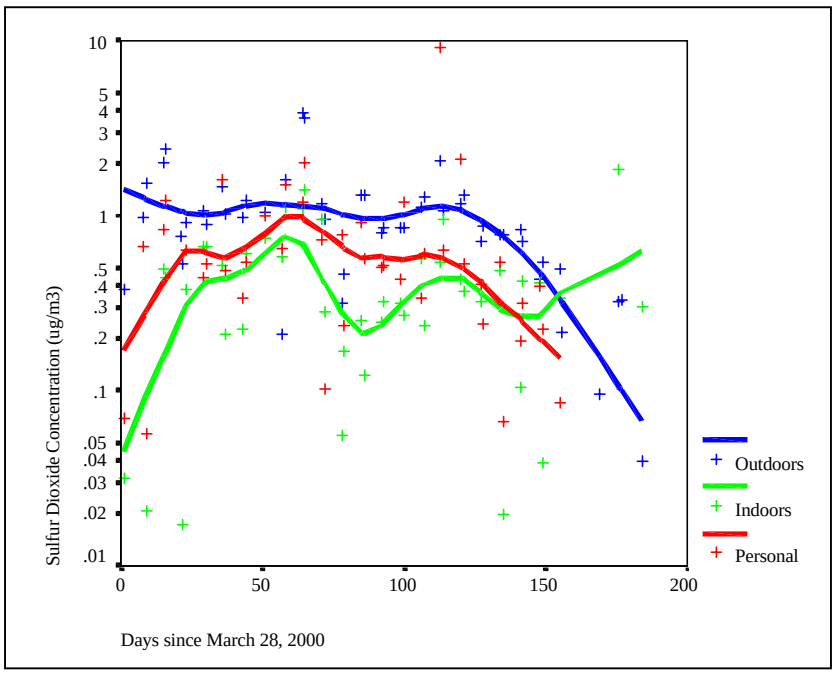
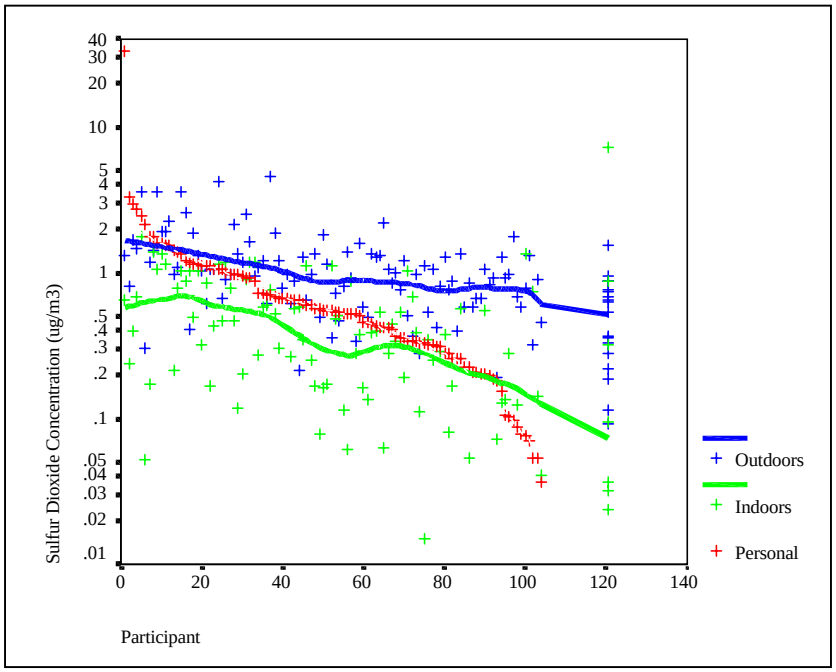



Figure 16 shows the relationship between the concentrations monitored personally, indoors and outdoors. This graph shows a small relationship between personal and indoor concentrations and a slightly larger relationship between personal and outdoor concentrations.

**Figure 16: Relationship between Exposures to Sulfur Dioxide by Sampler Site**



### *Ozone*

Figure 17 shows the cumulative distribution of ozone concentrations for the three types of samplers (personal, indoor, and outdoor). The median outdoor concentrations were approximately one order of magnitude higher than the personal and indoor concentrations. Other researchers[20] have also reported that ambient and outdoor concentrations are considerably above personal exposure levels in other locales, though less dramatically than was seen here. This finding speaks to the inherent inaccuracy of using ambient concentration levels as a proxy for personal exposure. While all distributions are positively skewed, none of the concentrations for personal exposures exceeded 50 ug/m3 while over half of the concentration measures for the outdoor sample exceeded that level.

As the figure demonstrates, median indoor and personal levels of $O_3$ were less than 10% of outdoor levels. All the outdoor samples were above the MDL of 0.82 µg/m$^3$ while 25% of the indoor and 10% of the personal samples were below the MDL. The MDL achieved in this study was low compared to other studies using passive samplers.[21, 22]

The median and 95$^{th}$ percentile $O_3$ levels (µg/m$^3$) for the different locations are summarized in Table 10 and compared to guidelines and levels in other communities. Levels of $O_3$ measured in this study were comparable to Lethbridge and Fort McMurray values. The indoor and personal levels in Grande Prairie were lower than other studies and much lower than the guideline.

Based on measures taken, the current ambient $O_3$ guideline for daily average concentrations (50 µg/m$^3$) was exceeded during the study. This guideline is often exceeded in rural areas of the province and is under review. Guidelines from other jurisdictions are considerably higher (157 U.S.EPA, 100 UK for 8 hr period). This guideline is currently under review and will be replaced by a Canadian wide standard of 125µg/m$^3$ over an 8-hour period by 2010.

**Figure 17: Distribution of Ozone**

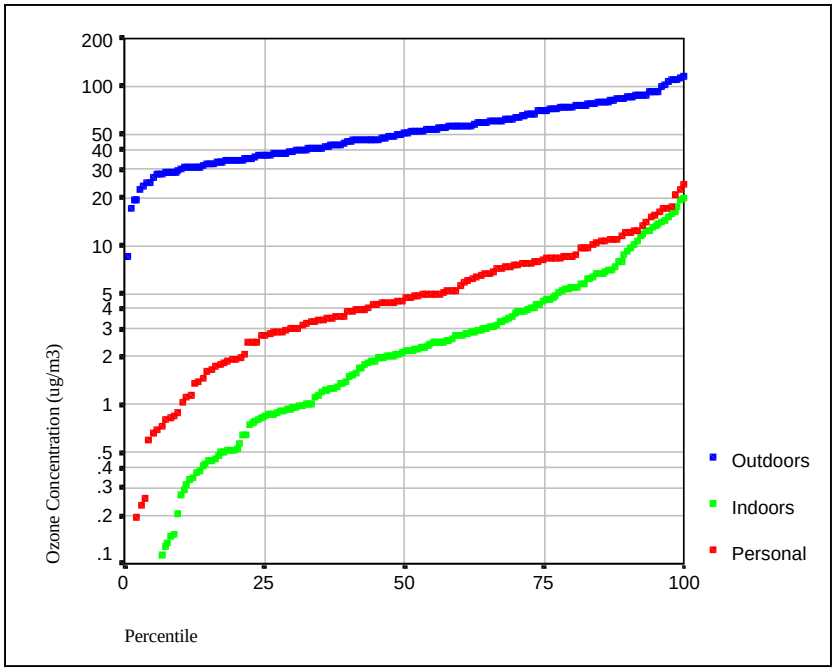


**Table 10: Comparison of $O_3$ Levels in µg/m$^3$ with Guidelines and Other Studies[23,24, 25]**

| Parameter | G.P. Median | G.P. 95th | Ft. Mc. Median | Ft. Mc. 95th | Leth. Median | Leth. 95th | Relevant Studies | Guideline/ Reference Level |
|---|---|---|---|---|---|---|---|---|
| Personal | 4.7 | 16.5 | 3.3 | 18 | 4.9 | 20 | 16 (summer)* 2.6 (winter)* | N/A |
| Indoor | 2.2 | 13.6 | 2.4 | 15 | 2.4 | 11 | 14 (summer)* 3.1 (winter)* | 240 (hour)*** |
| Outdoor | 51.2 | 94.3 | 39 | 91 | 57 | 140 | 37 (summer)* 30 (winter)* | 160 (hour) AENV |
| Ambient Station | | | 50 | 100 | N/A | N/A | N/A | 160 (hour) AENV |
| P/I ratio | 2.15 | 1.21 | 1.3 | 1.2 | 2.0 | 1.8 | 1.2 (summer)* 0.81 (winter)* | N/A |
| P/O ratio | 0.09 | 0.17 | 0.08 | 0.20 | 0.09 | 0.15 | 0.43 (summer)* 0.08 (winter)* | N/A |
| I/O ratio | 0.04 | 0.14 | 0.06 | 0.16 | 0.04 | 0.08 | 0.41**; 0.37 (summer)* 0.10 (winter)* | N/A |

* Lui, et al., 1995.
** Bernard et al., 1999.
*** Health Canada, 1989.

Figure 18 shows smoothed curves (produced by locally weighted regression) to represent the temporal trend in ozone exposures. Outdoor concentration levels peak in the spring at levels approximately double the summer and fall lows. Indoor levels appear to peak in the summer.

**Figure 18: Temporal Trend in Ozone Concentration**

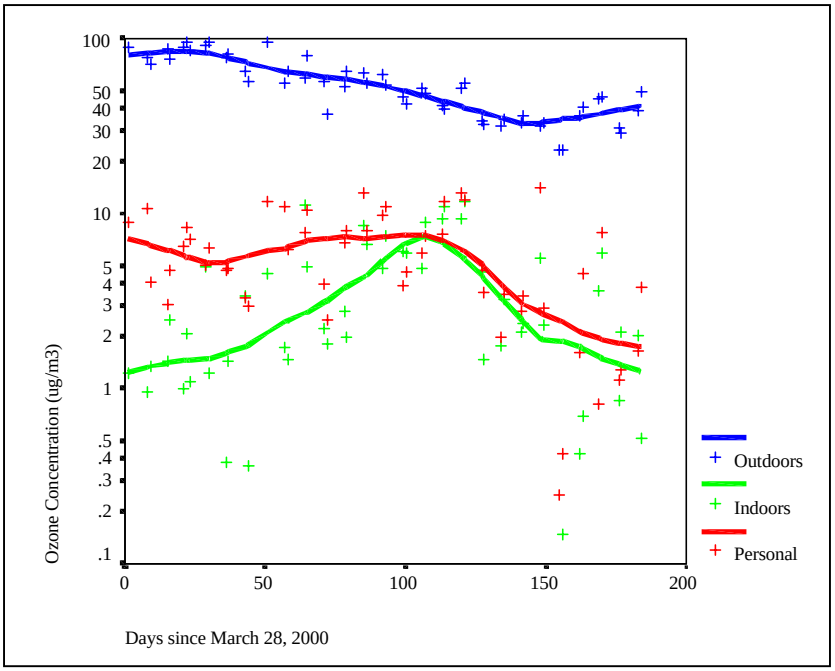
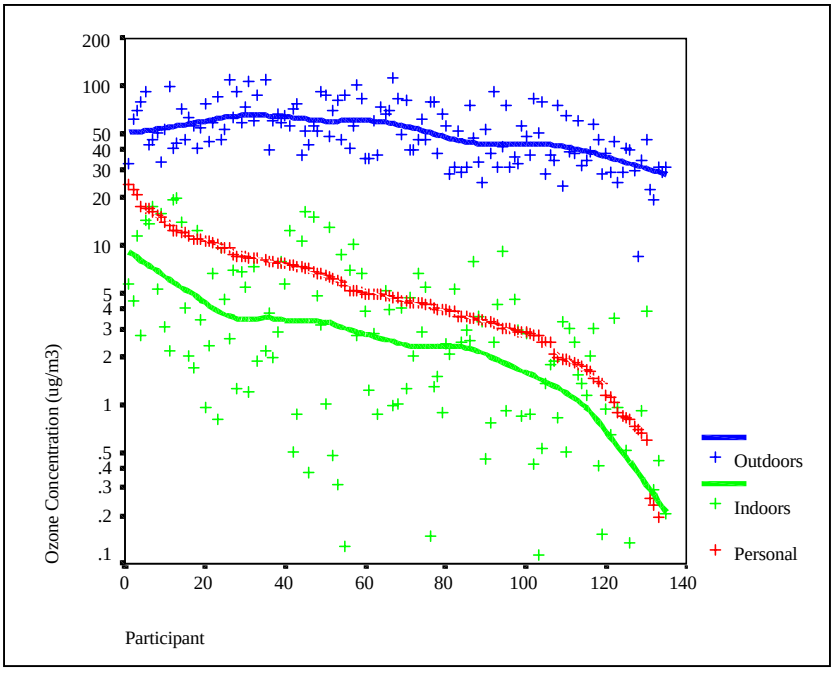


Figure 19 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows the ordered personal exposure levels for each subject in the study, and their corresponding levels of indoor and outdoor concentration levels. The current figure shows a strong relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. The relationship between outdoor exposures and personal exposures is considerably weaker, but positive nonetheless. The relative levels of the three exposures is strongly suggestive of a model of ozone diffusion which moves from outdoors to indoors and then to the person, who also moves outdoors often enough to raise personal exposure levels above the indoor concentration levels. A more detailed analysis is presented in a later section.

**Figure 19: Relationship between Exposures to Ozone by Sampler Site**




### *Volatile Organic Compounds*

The analyses of the volatile organic compounds (VOCs) detailed in the following pages share several general features: 1) there were generally many measurements that were below detection limits; 2) personal exposure levels were generally higher than indoor and outdoor levels; and 3) the strongest relationships occurred between personal and indoor levels of concentration, suggesting indoor sources of exposure for most of these chemicals.

#### *Hexane*

Figure 20 shows the cumulative distribution of hexane concentrations for the three types of samplers (personal, indoor, and outdoor). At the 50th percentile, personal and indoor concentrations were much higher than outdoor concentrations.

**Figure 20: Distribution of Hexane**

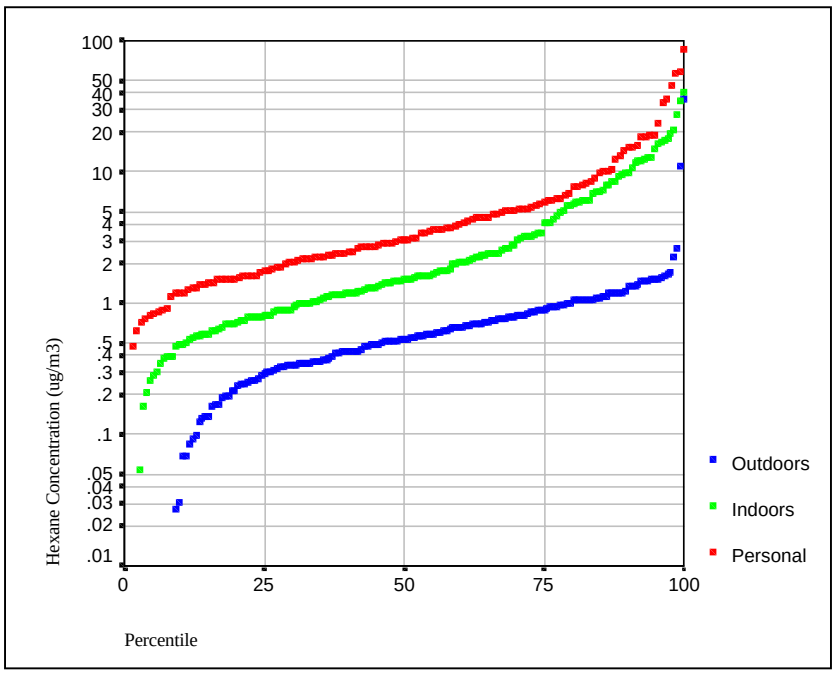



Figure 21 shows the temporal trend in hexane concentrations. There is little evidence to support a seasonal cycle.

**Figure 21: Temporal Trend in Hexane Concentration**

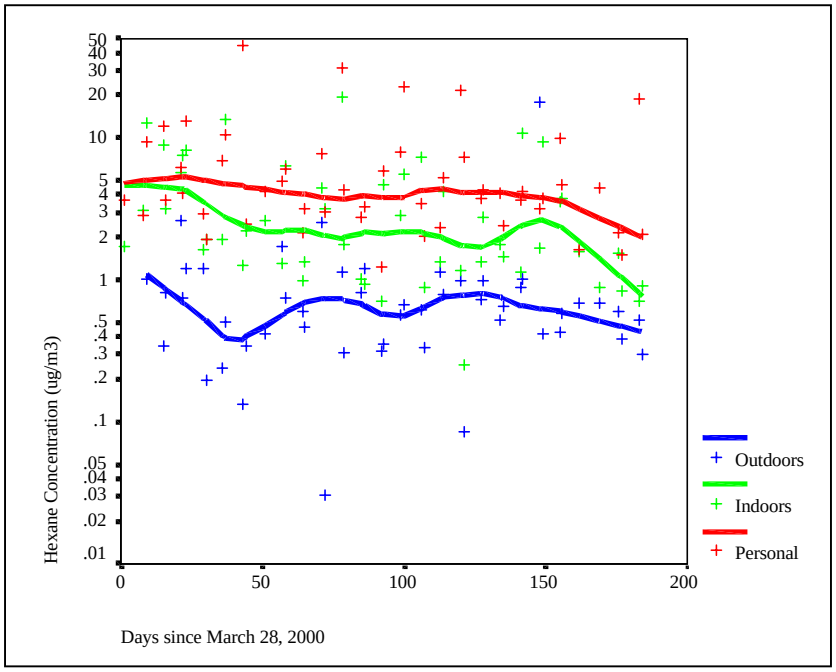
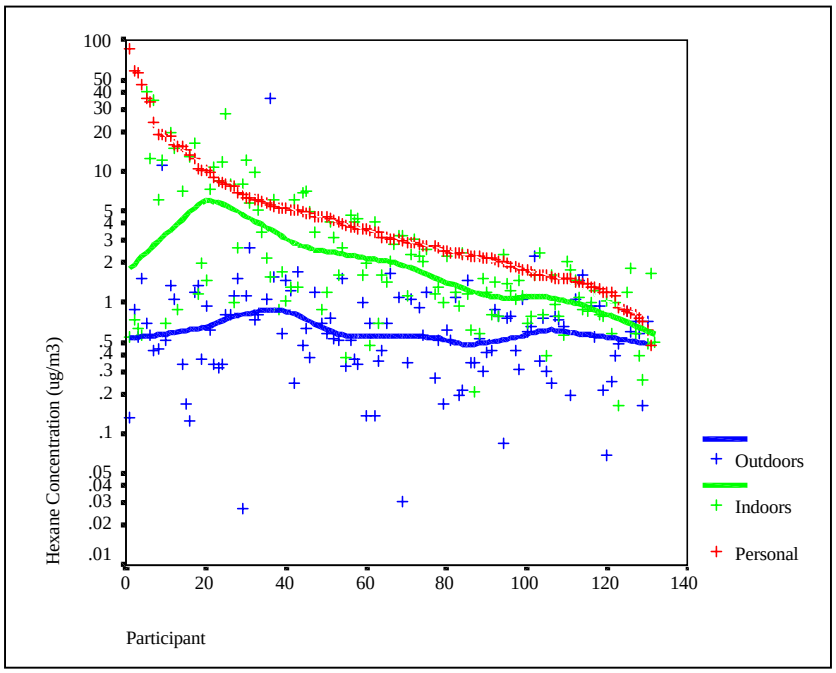


Figure 22 shows the relationship between the concentration obtained from the personal, indoor and outdoor samplers. There is a moderate relationship between measures of indoor and personal concentration.

**Figure 22: Relationship between Exposures to Hexane by Sampler Site**




*2-Butanone*

Figure 23 shows the cumulative distribution of 2-butanone concentrations for the three types of samplers. This contaminant was not detectable on a large minority of samplers at any location: more than 85% of the personal and indoor samplers did not have detectable concentrations, and more than 100% of outdoor samplers did not have detectable concentrations of 2-butanone. Of the few samplers that had detectable concentrations, indoor and personal samplers were significantly greater than outdoor measures.

**Figure 23: Distribution of 2-butanone**

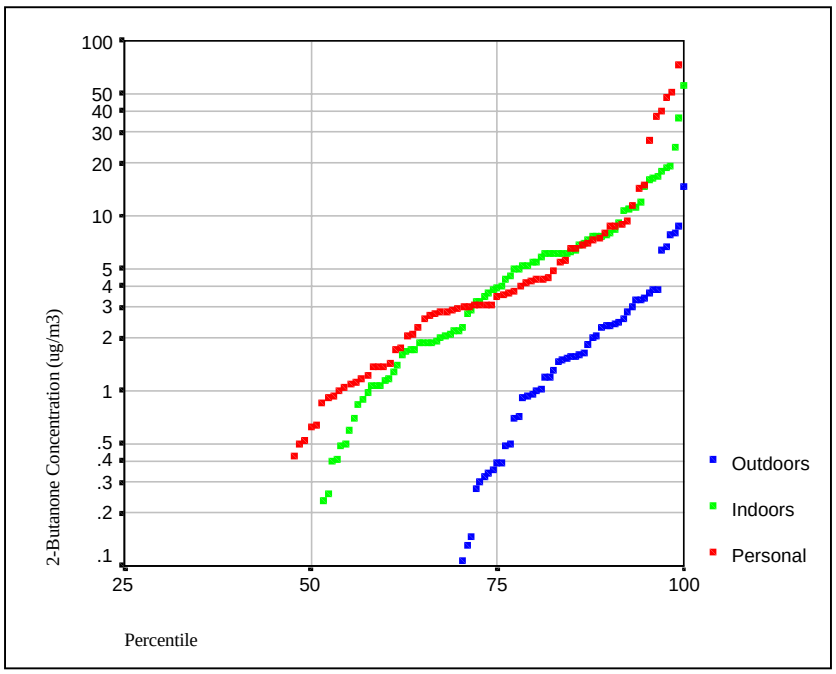



Figure 24 shows the temporal trend in 2-butanone concentrations. There are too few measurements to determine whether a temporal trend exists.

**Figure 24: Temporal Trend in 2-butanone Concentration**

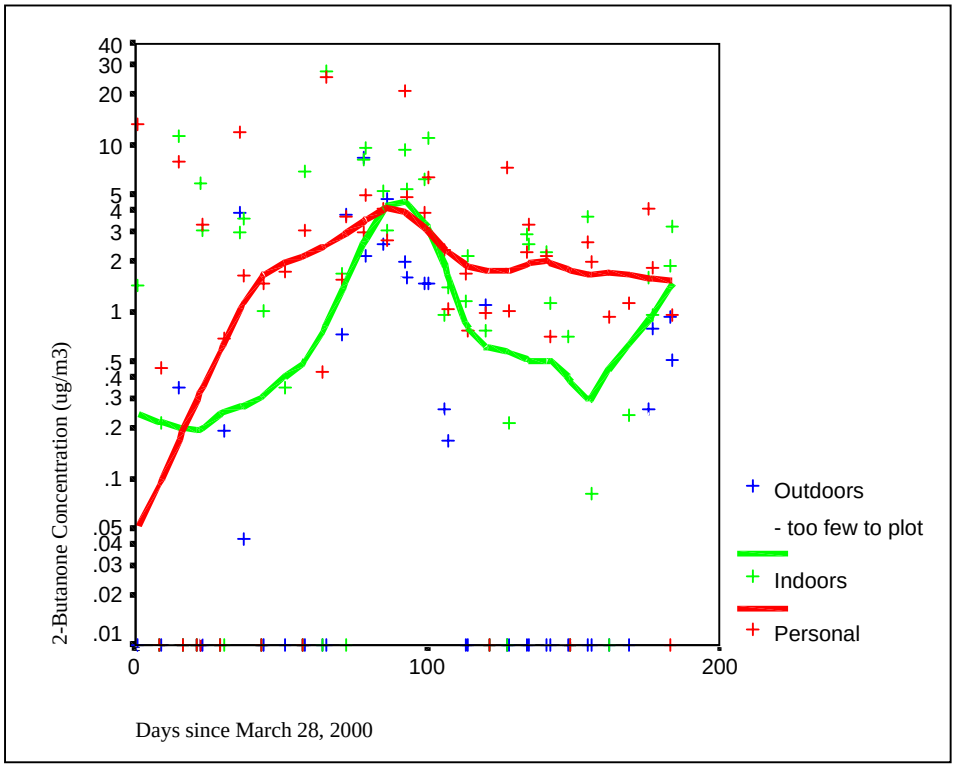
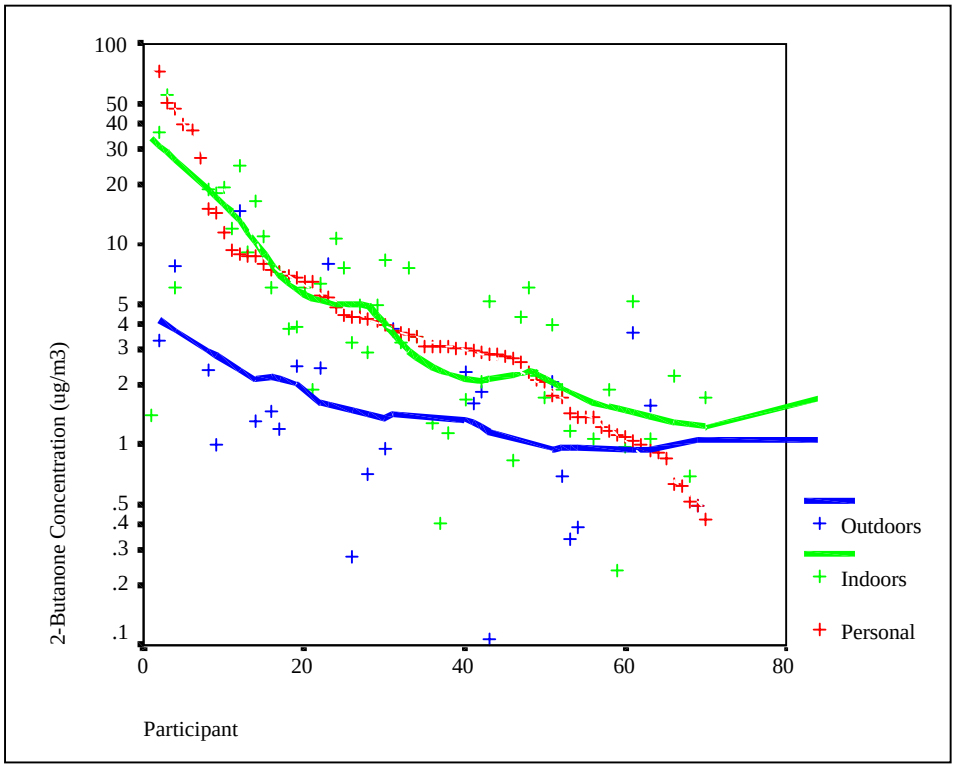


Figure 25 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations.

**Figure 25: Relationship between Exposures to 2-butanone by Sampler Site**




*3-Methylhexane*

Figure 26 shows the cumulative distribution of 3-methylhexane concentrations for the three types of samplers (personal, indoor, and outdoor). About 40% of the indoor samplers and more than 90% of the outdoor samplers had concentrations of 3-methylhexane below detectable limits. Personal and indoor concentrations were much higher than outdoor concentrations.

**Figure 26: Distribution of 3-methylhexane**

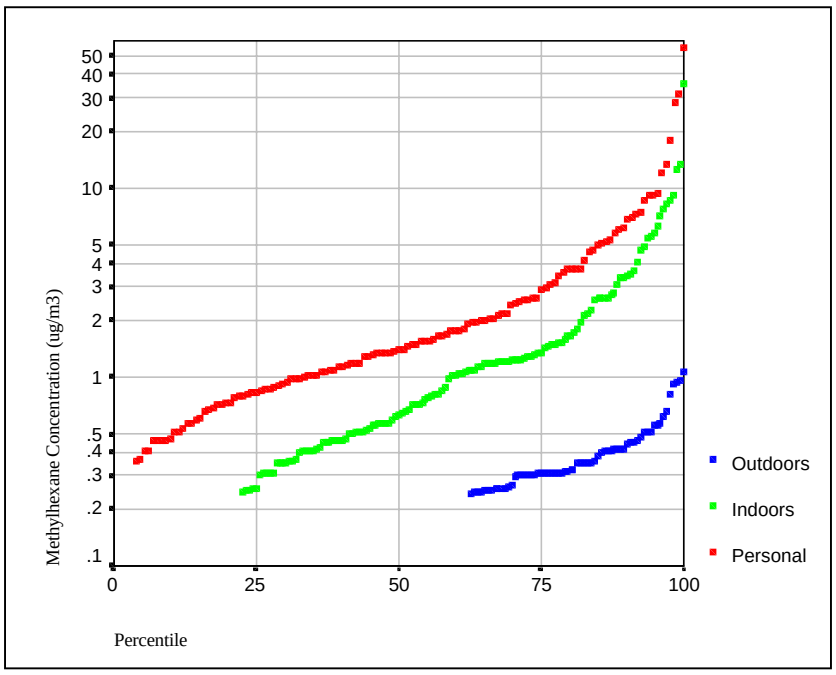



Figure 27 shows the temporal trend in 3-methylhexane concentrations. There is some suggestion that spring levels of exposure may be higher than fall levels.

**Figure 27: Temporal Trend in 3-methylhexane Concentration**

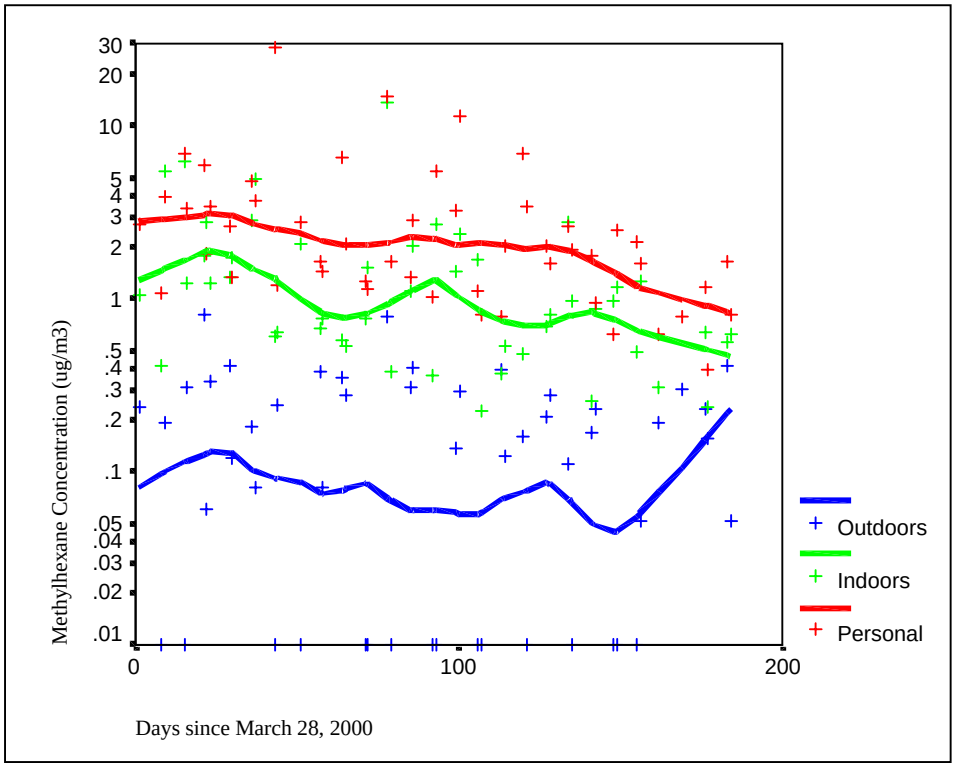
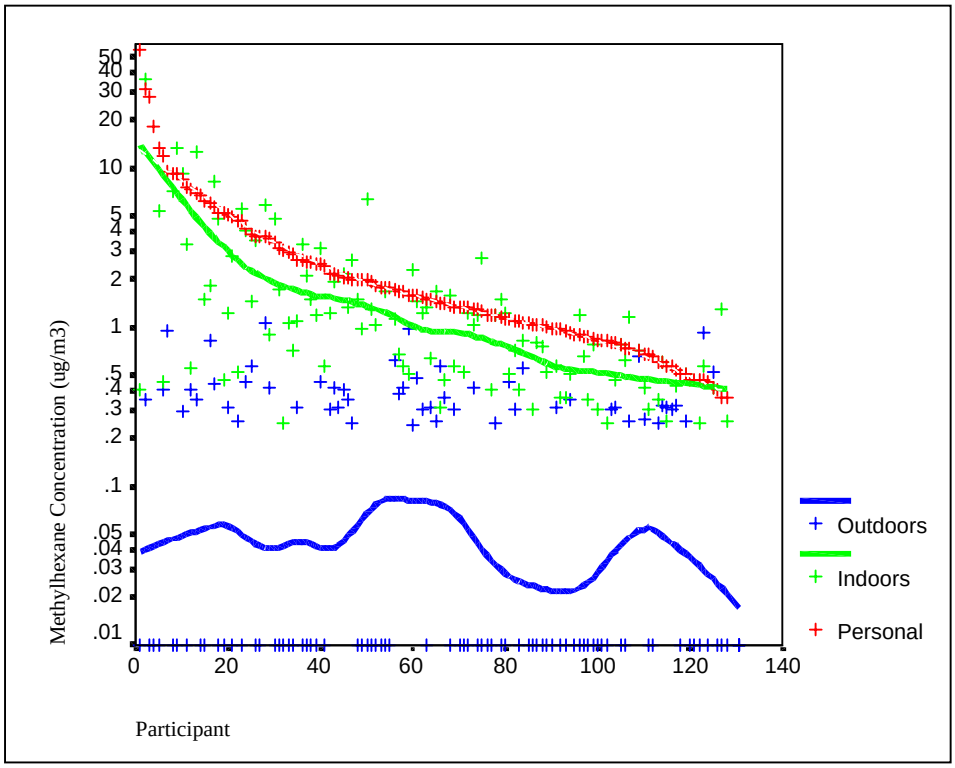


Figure 28 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. There is no relationship between outdoor exposures and personal exposures.

**Figure 28: Relationship between Exposures to 3-methylhexane by Sampler Site**




*Benzene*

Figure 29 shows the cumulative distribution of benzene concentrations for the three types of samplers (personal, indoor, and outdoor).

Table 11 contains a summary of the benzene measures taken during the study showing the median and 95$^{th}$ percentile levels (µg/m$^3$) compared to guidelines and levels at other relevant communities. With fewer samples taken in Lethbridge during the study, estimates of median indoor and outdoor levels were not reliable and are not included. The outdoor benzene levels were low and comparable to levels reported for rural areas in Canada. The median personal levels were roughly 2.5 times the outdoor levels and roughly 10% of the levels reported in the TEAM study. The TEAM study also found that the highest levels of benzene were from the personal samplers, followed by the indoor sampler levels, while the outdoor samplers contained the lowest levels of benzene.[26]

**Figure 29: Distribution of Benzene**

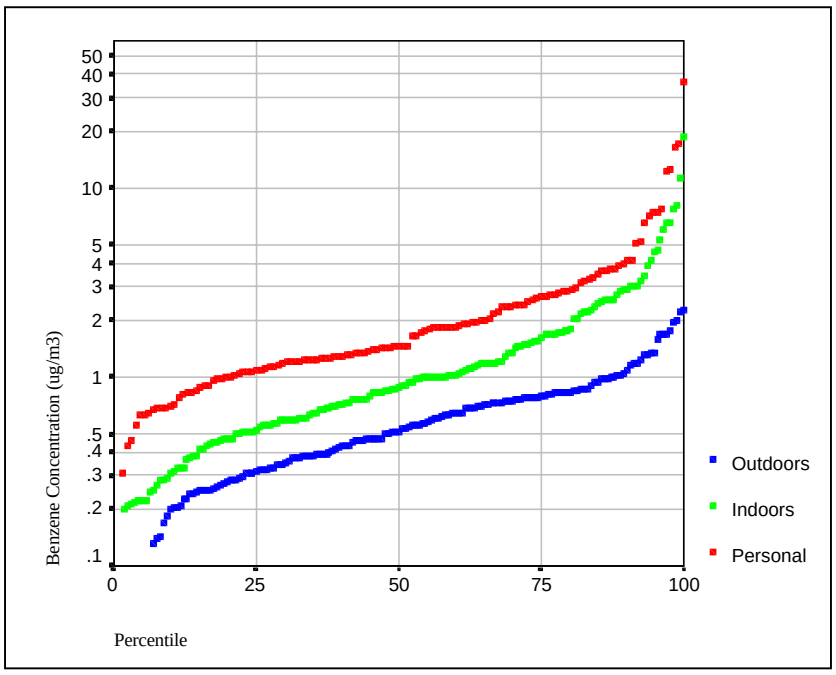



**Table 11: Comparison of Benzene Levels in µg/m$^3$ with Guidelines and Other Studies[27, 28]**

| Parameter | G.P. Median | G.P. 95th | Ft. Mc. Median | Ft. Mc. 95th | Leth. Median | Leth. 95th | Relevant Studies | Guideline/ Reference Level |
|---|---|---|---|---|---|---|---|---|
| Personal | 1.45 | 7.53 | 2.8 | 10.0 | 2.1 | 6.7 | 15 (TEAM)** | N/A |
| Indoor | 0.89 | 4.89 | 1.7 | 6.6 | * | 4.8 | 10 (TEAM)** | N/A |
| Outdoor | 0.52 | 1.61 | 1.3 | 5.5 | * | 3.6 | 2.6*** | 30 (hour) AENV |
| Ambient Station | | | 1.2 | 3.1 | N/A | N/A | 4.4 (urban)<br>0.6 to 1.2 (rural)** | 30 (hour) AENV<br>16 UK current<br>3.2 UK future |
| P/I ratio | 1.63 | 1.54 | 1.7 | 1.5 | N/A | 1.4 | 1.5** | N/A |
| P/O ratio | 2.79 | 4.67 | 2.05 | 1.82 | N/A | 1.90 | 2.5** | N/A |
| I/O ratio | 1.71 | 3.03 | 1.23 | 1.20 | N/A | 1.34 | 1.7** | N/A |

* Estimate not available due to small number of Lethbridge samples.
** Wallace, 1996.
*** Median value from monitoring across Canada (Dann et al., 1995).

Figure 30 shows the temporal trend in benzene concentrations. Exposure to benzene appears to vary across the seasons.

**Figure 30: Temporal Trend in Benzene Concentration**

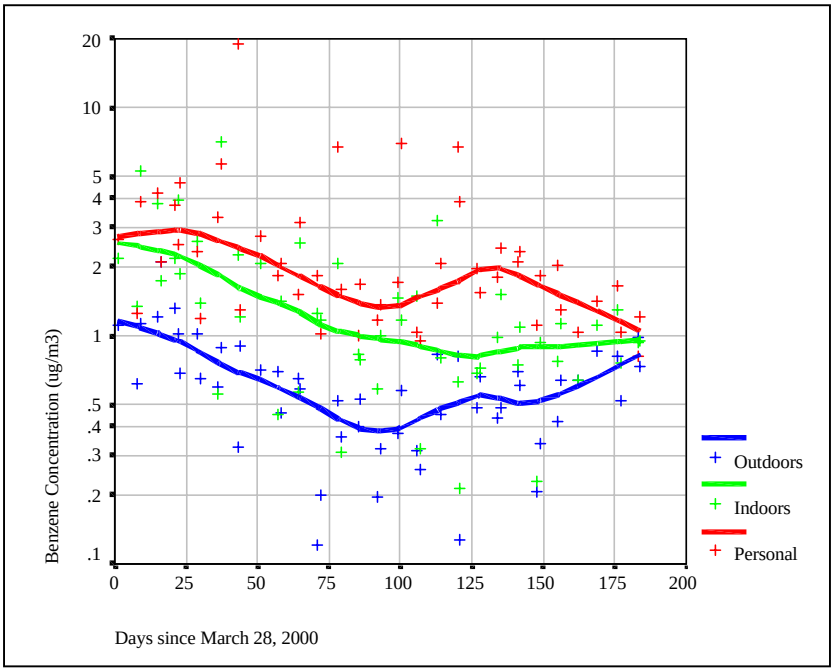
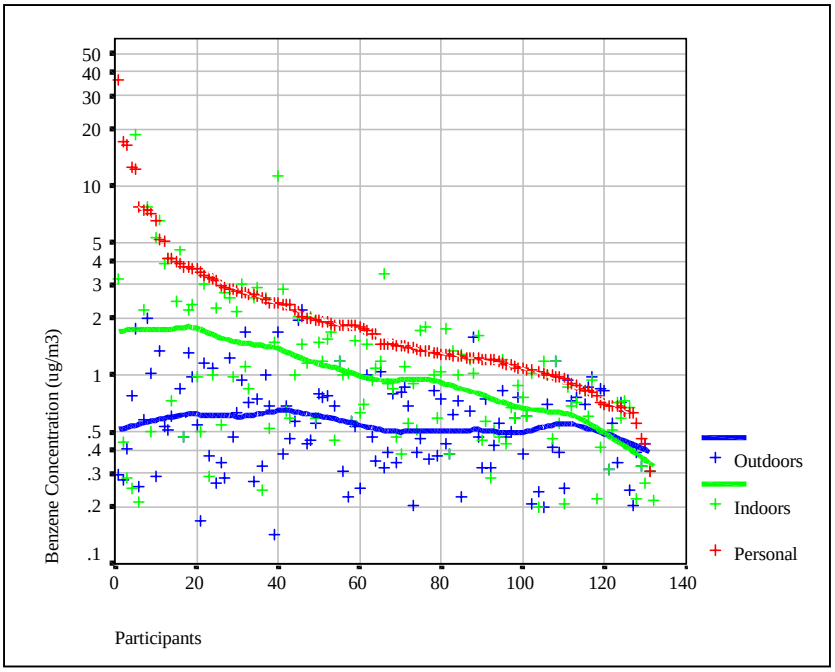



Figure 31 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a moderate relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. There appears to be no relationship between personal exposure and outdoor.

**Figure 31: Relationship between Exposures to Benzene by Sampler Site**



*Heptane*

Figure 32 shows the cumulative distribution of heptane concentrations for the three types of samplers (personal, indoor, and outdoor). More than 80% of the outdoor samplers had concentrations of heptane below detectable limits. Personal and indoor concentrations were much higher than outdoor concentrations.

**Figure 32: Distribution of Heptane**

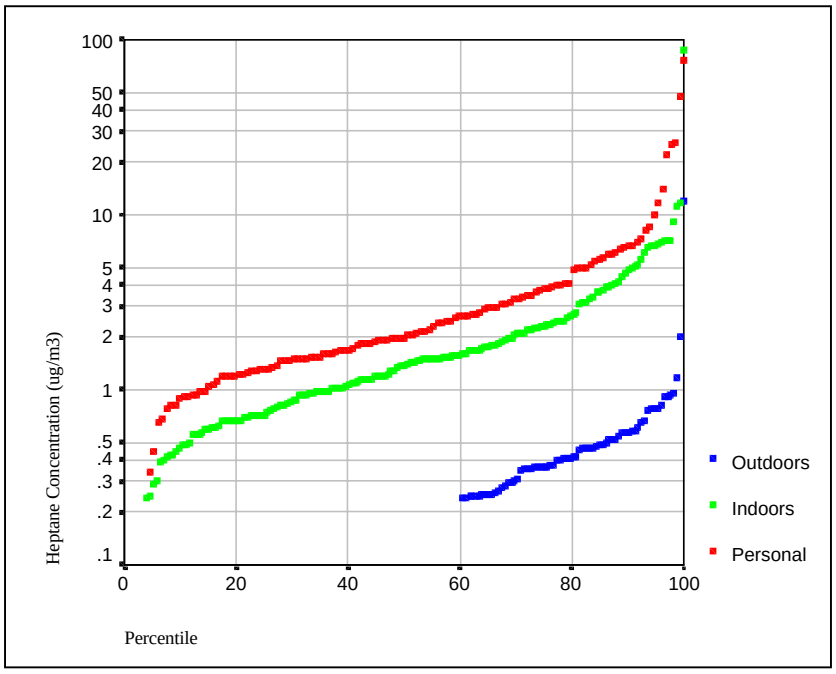



Figure 33 shows the temporal trend in heptane concentrations. There is insufficient evidence to conclude that heptane concentrations differ across the seasons.

**Figure 33: Temporal Trend in Heptane Concentration**

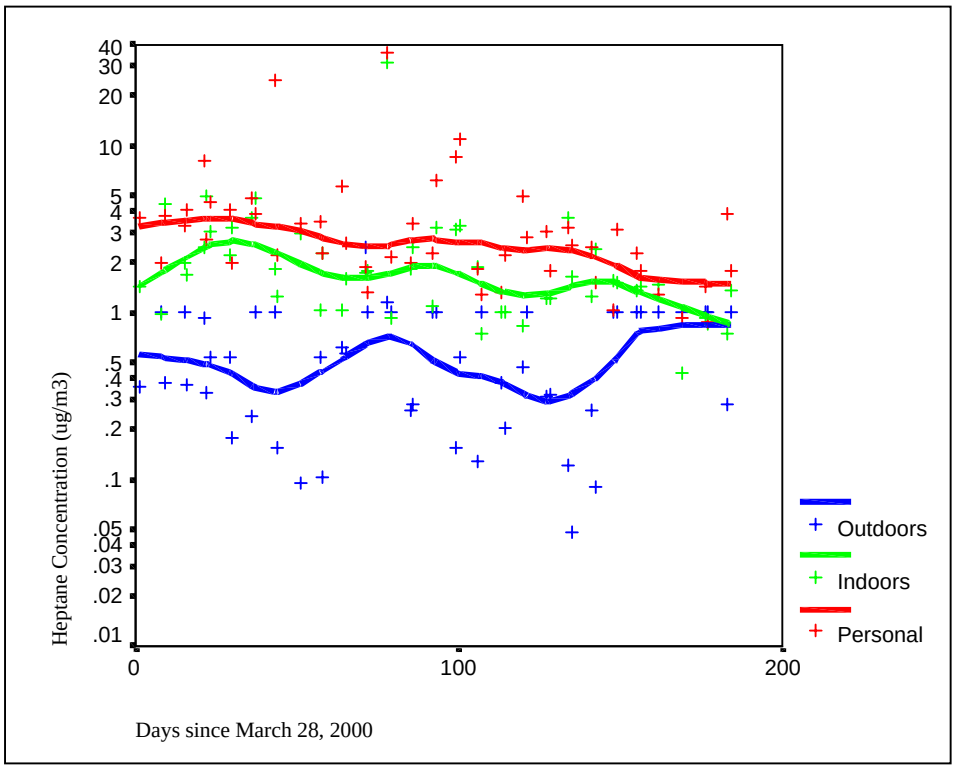
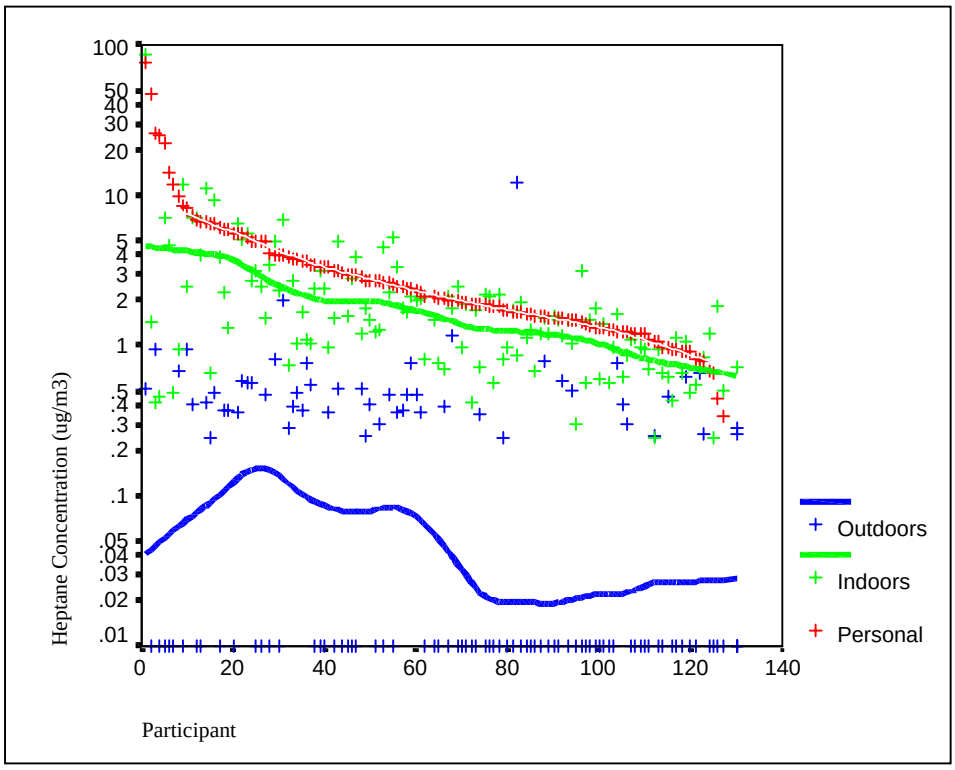



Figure 34 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a relatively strong relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. There is no apparent relationship between outdoor exposures and personal exposures.

**Figure 34: Relationship between Exposures to Heptane by Sampler Site**




*Toluene*

Figure 35 shows the cumulative distribution of toluene concentrations for the three types of samplers (personal, indoor, and outdoor). At the 50th percentile, personal and indoor concentrations were almost an order of magnitude higher than outdoor concentrations.

**Figure 35: Distribution of Toluene**

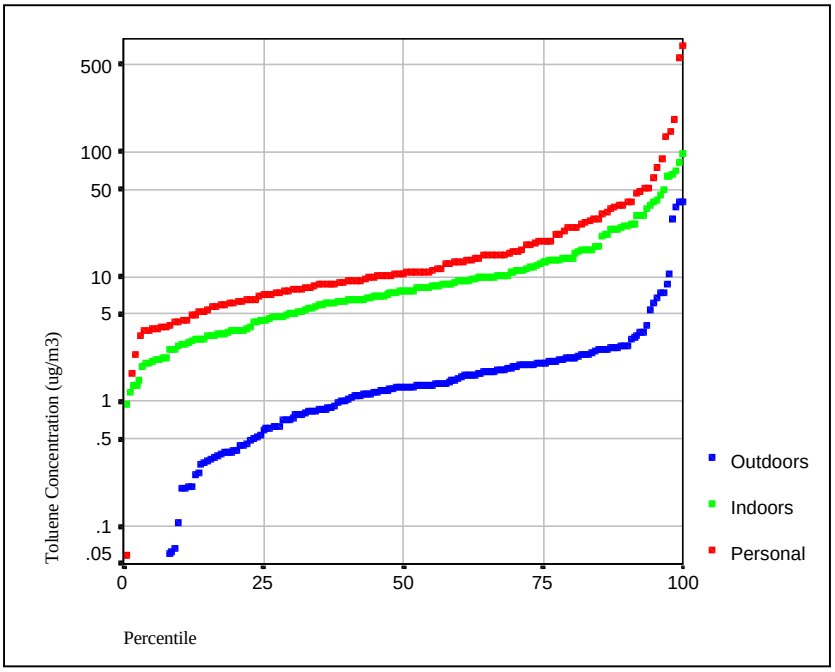



Figure 36 shows the temporal trend in toluene concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons.

**Figure 36: Temporal Trend in Toluene Concentration**

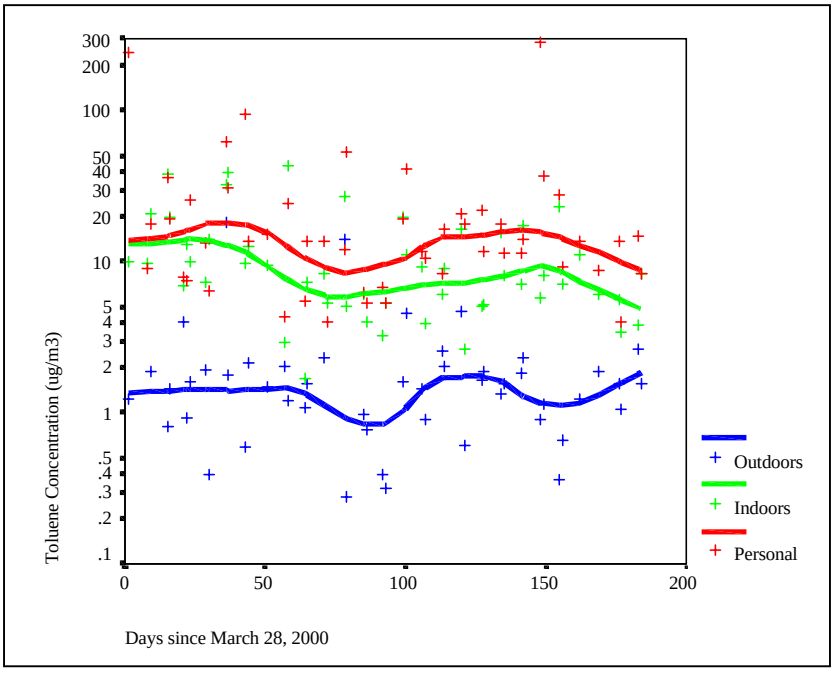
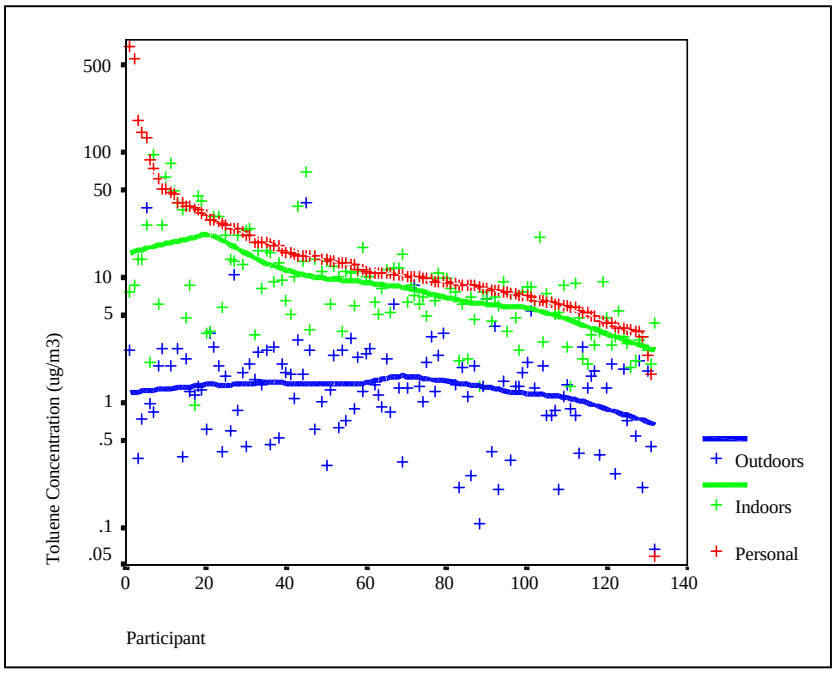


Figure 37 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal exposure concentrations and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. There appears to be no relationship between personal and outdoor exposures.

**Figure 37: Relationship between Exposures to Toluene by Sampler Site**




*Octane*

Figure 38 shows the cumulative distribution of octane concentrations for the three types of samplers (personal, indoor, and outdoor). Very few outdoor samplers had detectable concentrations of octane. Personal levels are higher than indoor levels.

**Figure 38: Distribution of Octane**

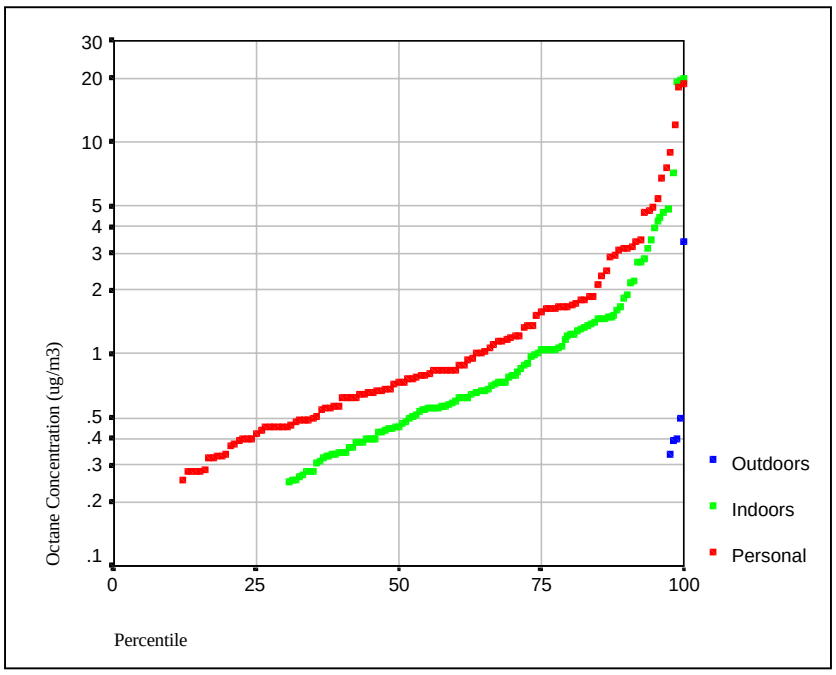



Figure 39 shows the temporal trend in octane concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons.

**Figure 39: Temporal Trend in Octane Concentration**

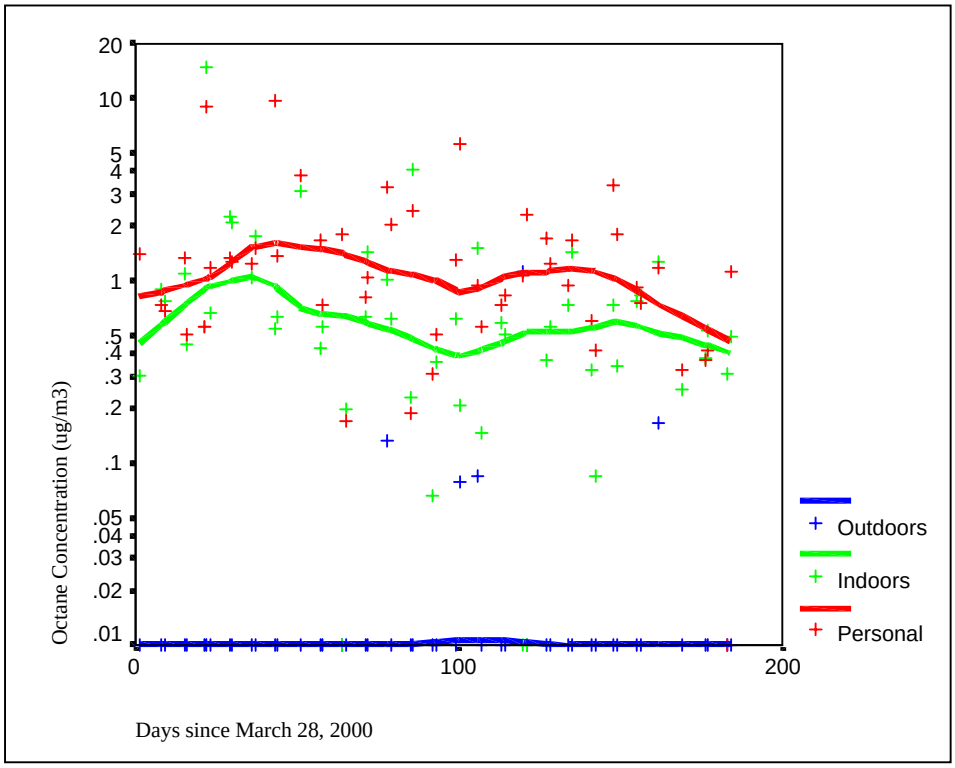
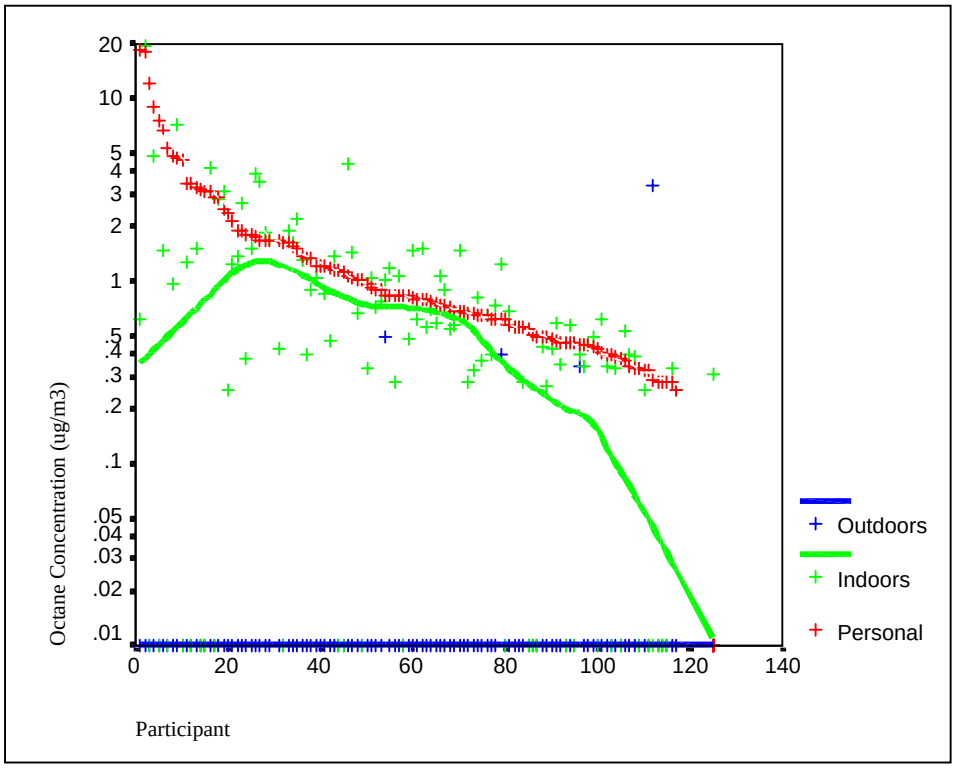


Figure 40 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations, but no relationship with outdoor concentrations.

**Figure 40: Relationship between Exposures to Octane by Sampler Site**




*Ethylbenzene*

Figure 41 shows the cumulative distribution of ethylbenzene concentrations for the three types of samplers (personal, indoor, and outdoor). More than 90% of the outdoor samplers had concentrations of ethylbenzene below detectable limits. Once again, personal concentrations were greater than indoor concentrations.

**Figure 41: Distribution of Ethylbenzene**

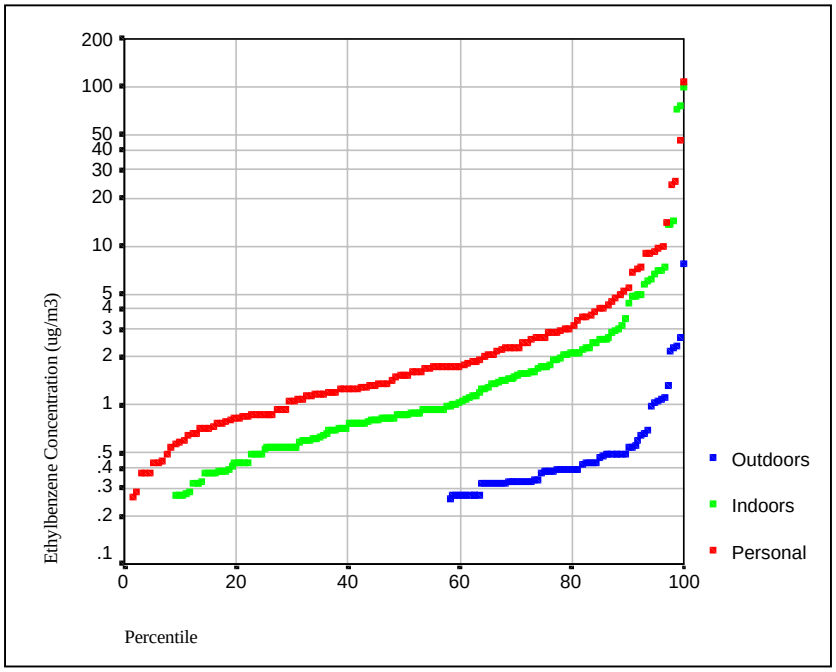



Figure 42 shows the temporal trend in ethylbenzene concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons.

**Figure 42: Temporal Trend in Ethylbenzene Concentration**

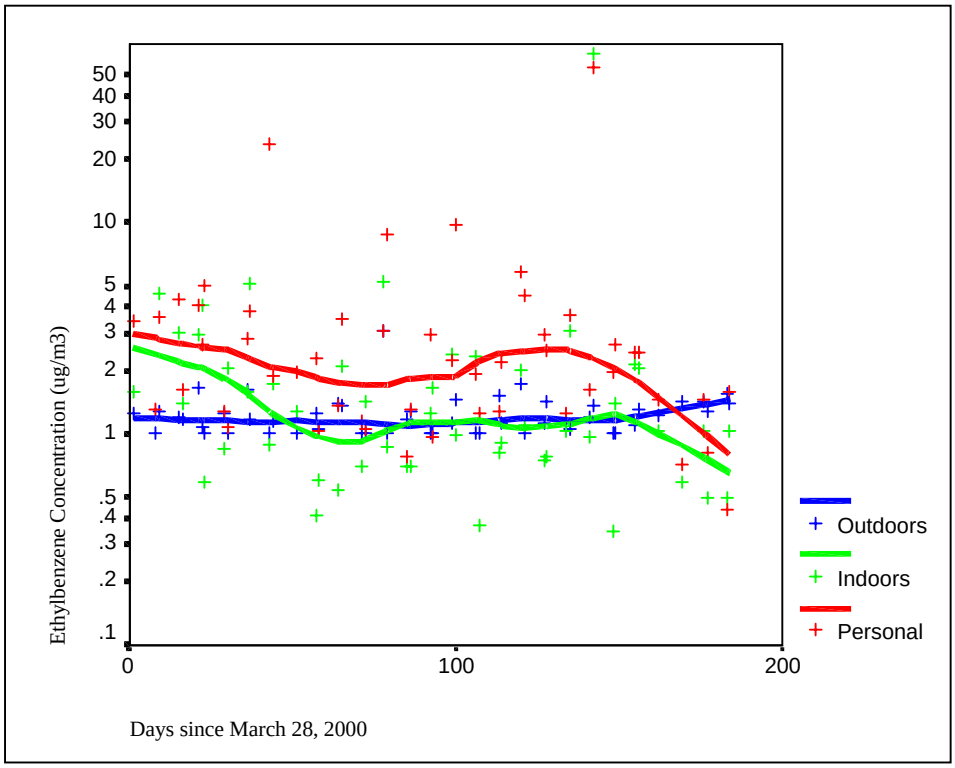
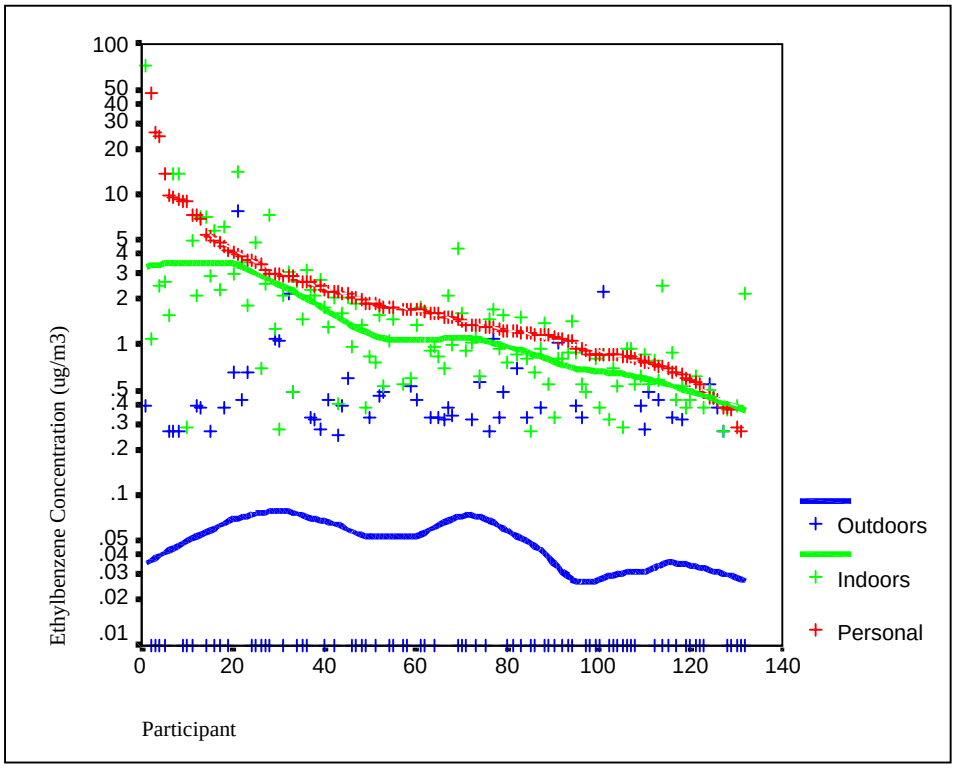


Figure 43 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations but no relationship between personal and outdoor exposures concentrations.

**Figure 43: Relationship between Exposures to Ethylbenzene by Sampler Site**




*M-, P-xylene*

Figure 44 shows the cumulative distribution of m-, p-xylene concentrations for the three types of samplers (personal, indoor, and outdoor). At the 50$^{th}$ percentile, personal and indoor concentrations were more than triple outdoor concentrations.

**Figure 44: Distribution of m-, p-xylene**

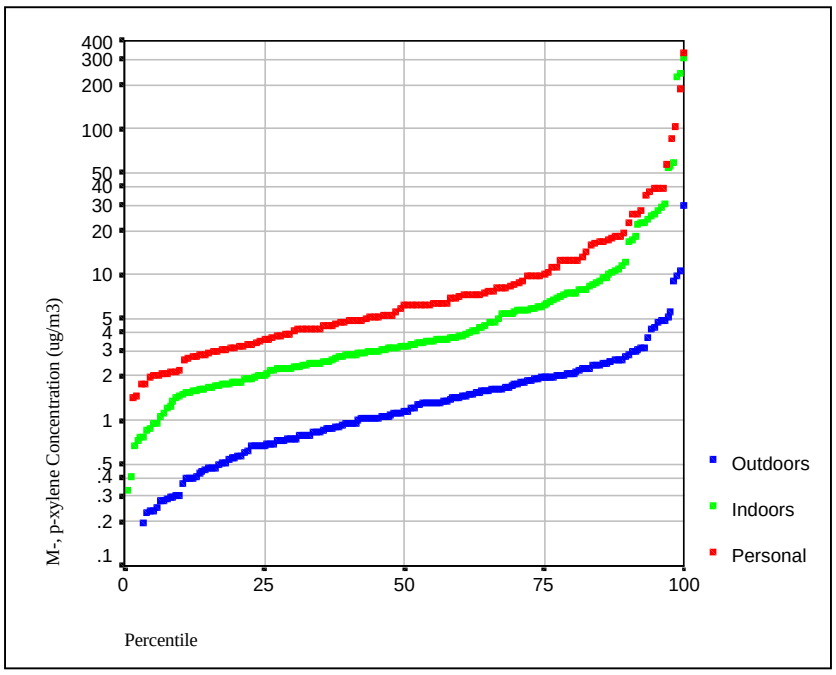



Figure 45 shows the temporal trend in m-, p-xylene concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons.

**Figure 45: Temporal Trend in m-, p-xylene Concentration**

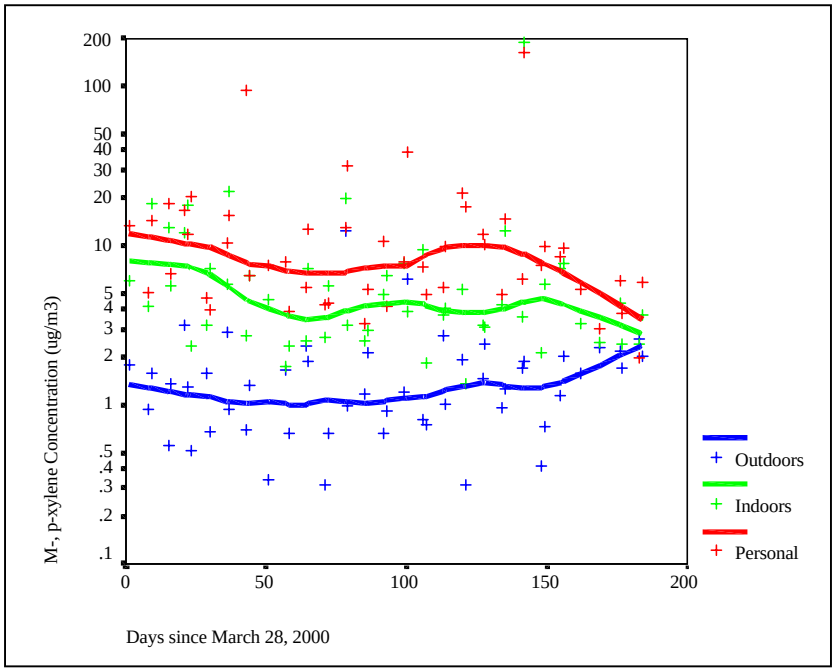
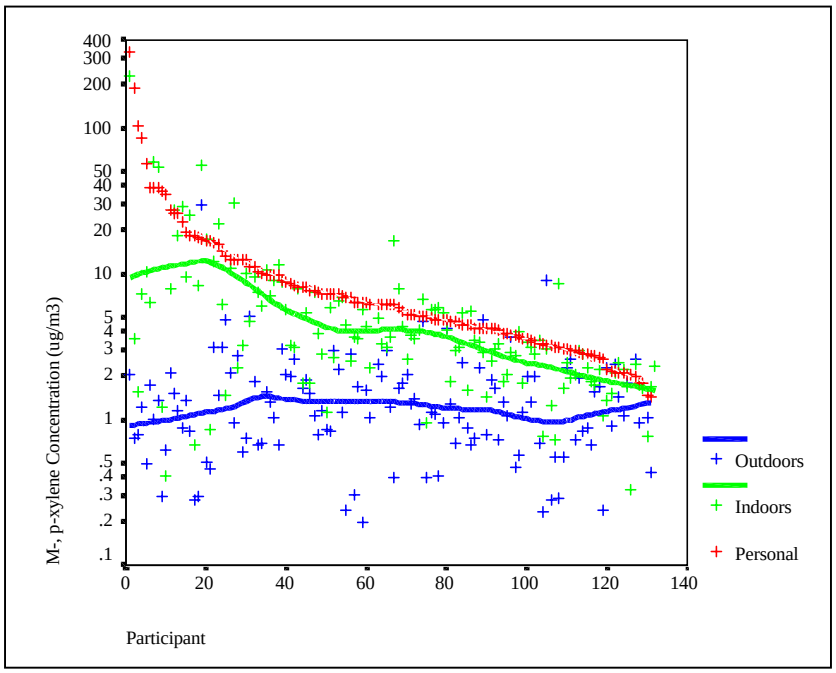


Figure 46 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations and no relationship with outdoor exposure concentrations.

**Figure 46: Relationship between Exposures to m-, p-xylene by Sampler Site**




*O-Xylene*

Figure 47 shows the cumulative distribution of o-xylene concentrations for the three types of samplers (personal, indoor, and outdoor). About 75% of the outdoor samplers had concentrations of o-xylene below detectable limits. Personal concentrations were about double indoor concentrations.

**Figure 47: Distribution of O-Xylene**

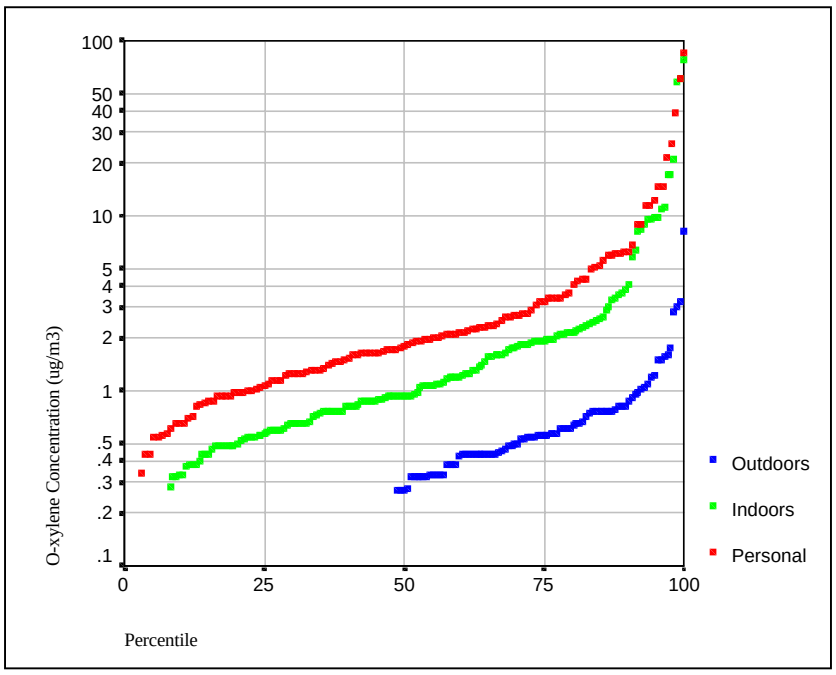



Figure 48 shows the Temporal trend in o-xylene concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons.

**Figure 48: Temporal Trend in o-xylene Concentration**

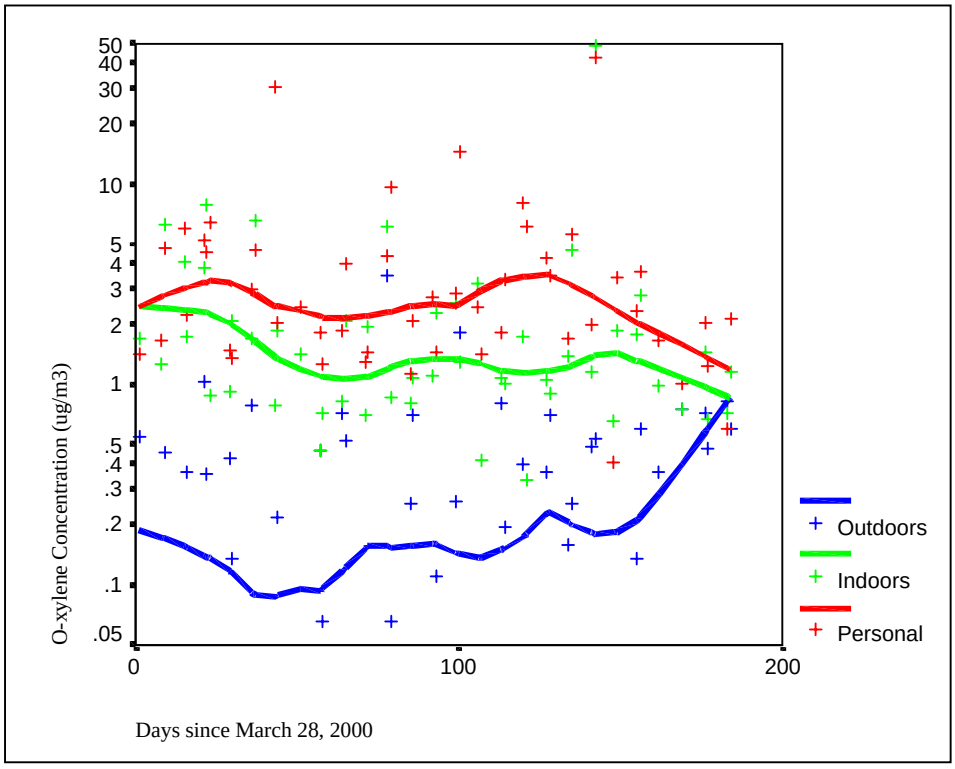
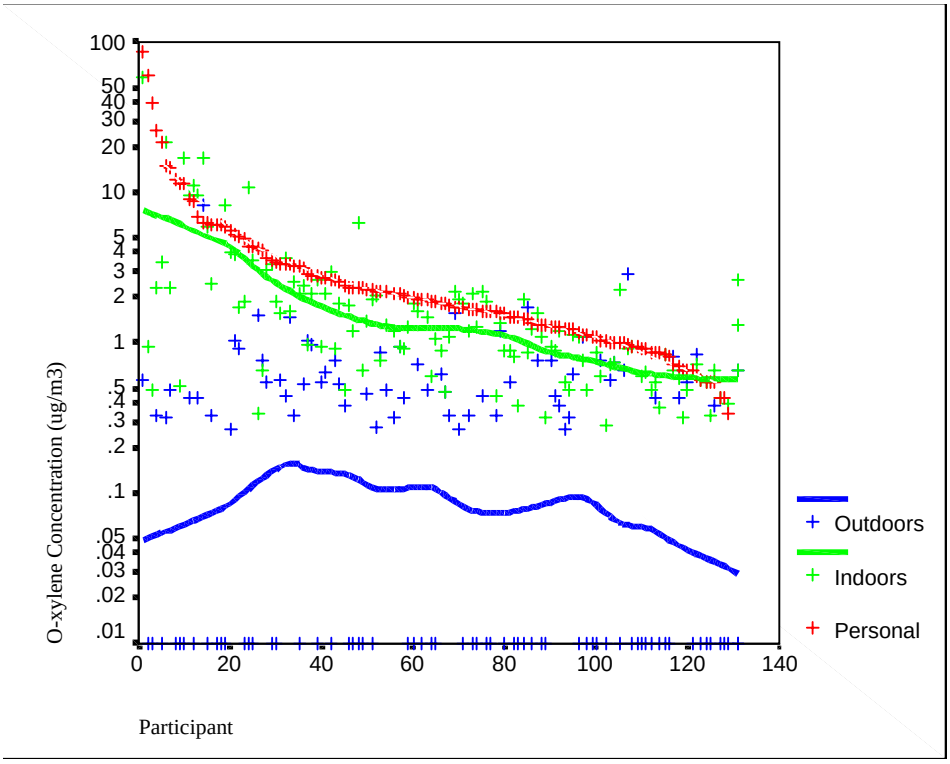


Figure 49 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations but no relationship between personal exposures and outdoor exposures.

**Figure 49: Relationship between Exposures to o-xylene by Sampler Site**




*Nonane*

Figure 50 shows the cumulative distribution of nonane concentrations for the three types of samplers (personal, indoor, and outdoor). No outdoor samplers recorded detectable nonane. Personal concentrations were higher than indoor concentrations.

**Figure 50: Distribution of Nonane**

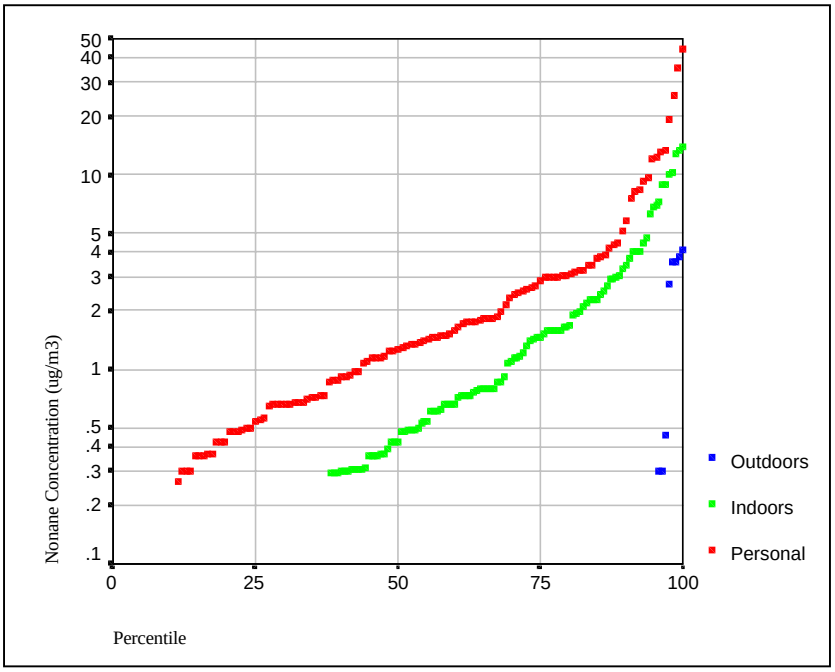



Figure 51 shows the Temporal trend in nonane concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons, though it appears that nonane levels may decrease in the summer.

**Figure 51: Temporal Trend in Nonane Concentration**

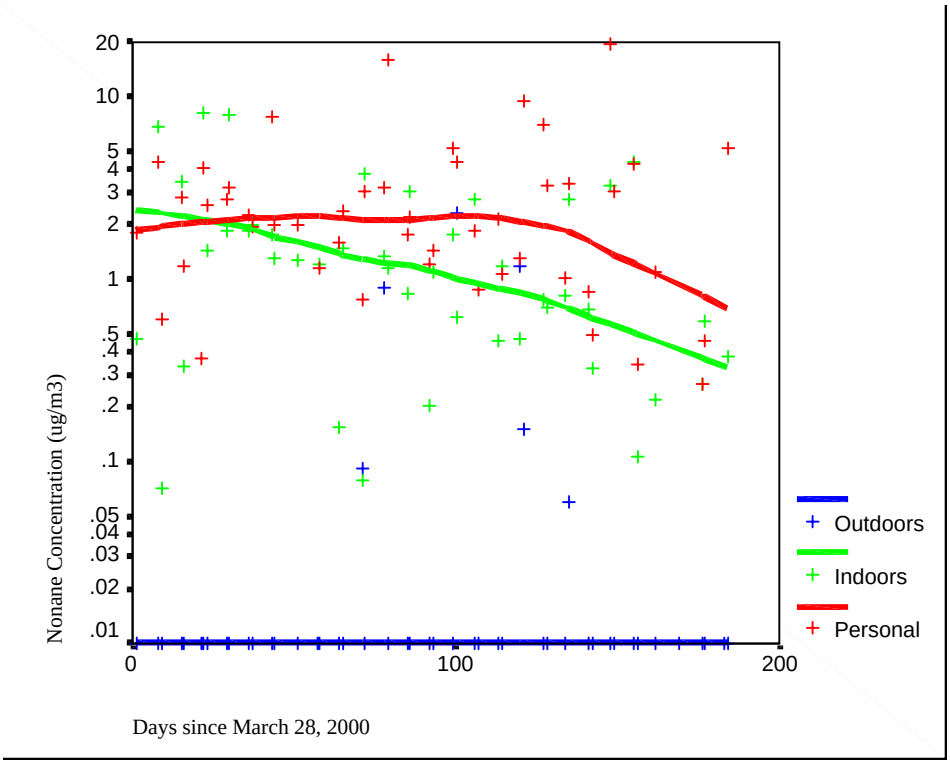
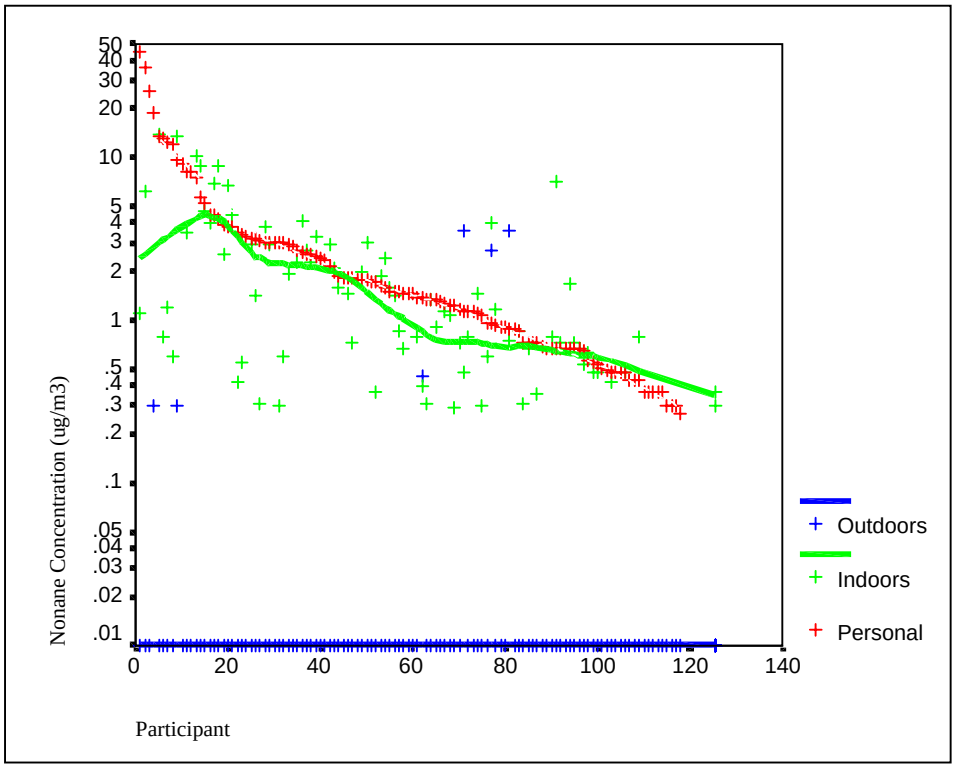


Figure 52 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations, but no relationship between personal exposures and outdoor exposures.

**Figure 52: Relationship between Exposures to Nonane by Sampler Site**




*Decane*

Figure 53 shows the cumulative distribution of decane concentrations for the three types of samplers (personal, indoor, and outdoor). Over 95% of the outdoor samplers had concentrations of decane below detectable limits. Personal concentrations were higher than indoor concentrations.

**Figure 53: Distribution of Decane**

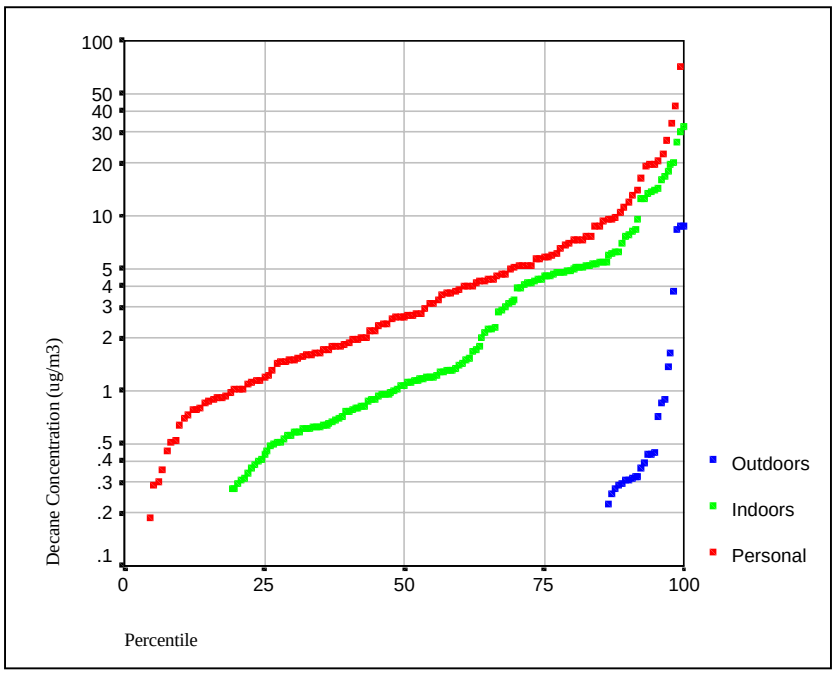



Figure 54 shows the temporal trend in decane concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons, although a summer minimum is possible.

**Figure 54: Temporal Trend in Decane Concentration**

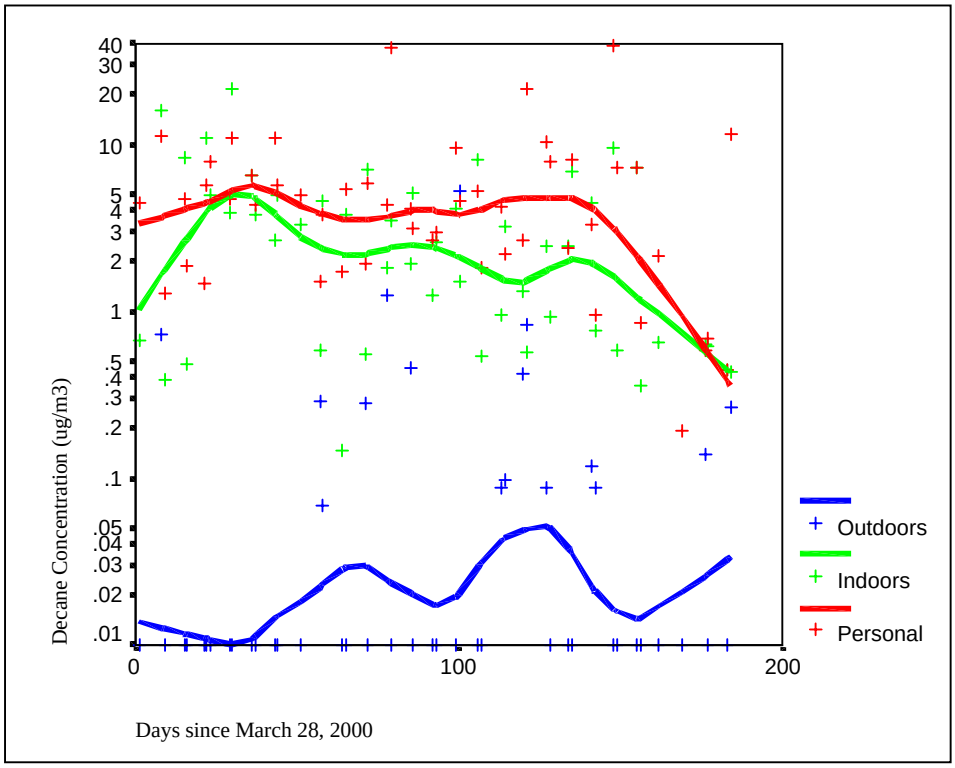
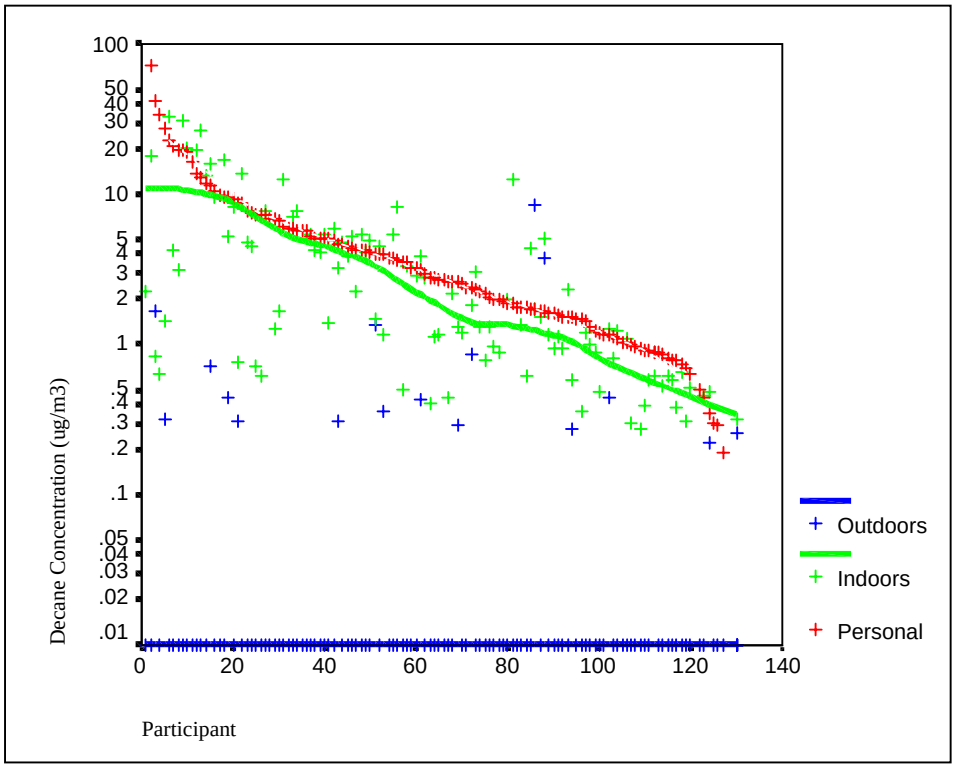


Figure 55 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a strong relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. There is no relationship between outdoor exposures and personal exposures.

**Figure 55: Relationship between Exposures to Decane by Sampler Site**




*Limonene*

Figure 56 shows the cumulative distribution of limonene concentrations for the three types of samplers (personal, indoor, and outdoor). Almost all of the personal and indoor samplers had measurable concentrations of limonene, while very few of the outdoor samplers had measurable concentrations of this contaminant. Personal exposure is slightly higher than indoor exposure.

**Figure 56: Distribution of Limonene**

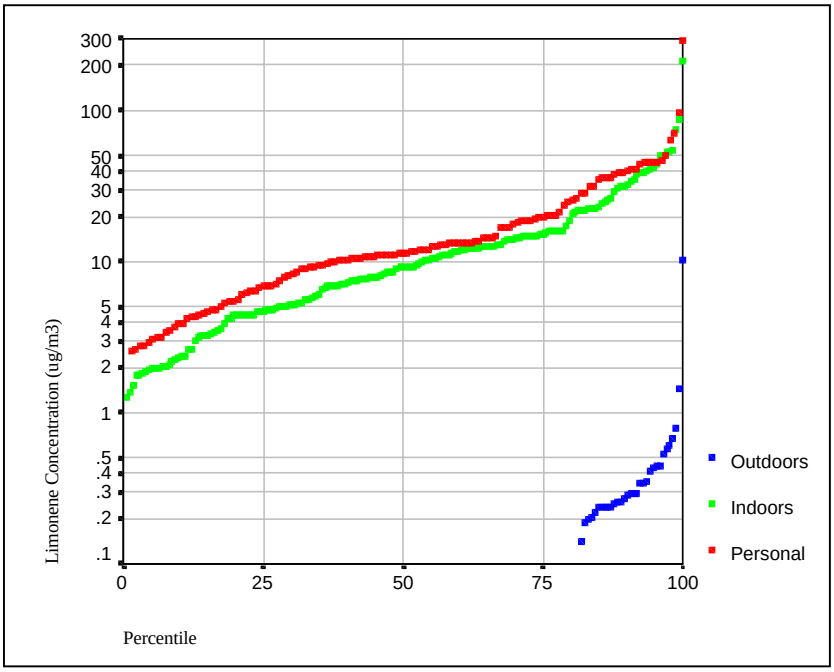



Figure 57 shows the temporal trend in limonene concentrations. There is insufficient evidence to conclude that concentrations differ across the seasons.

**Figure 57: Temporal Trend in Limonene Concentration**

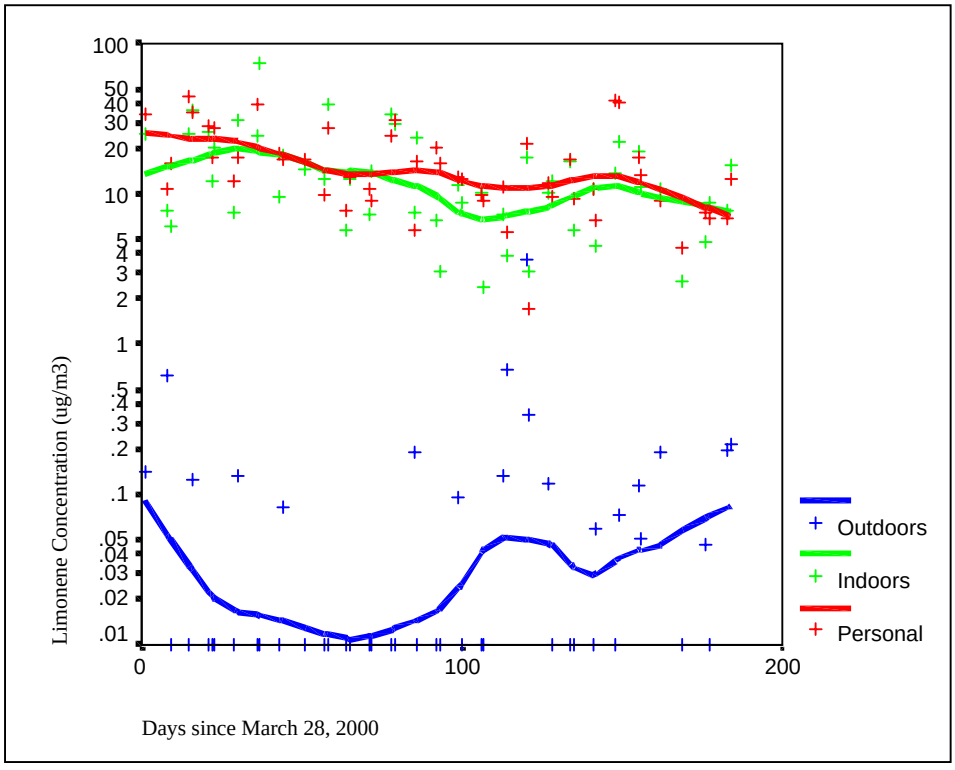
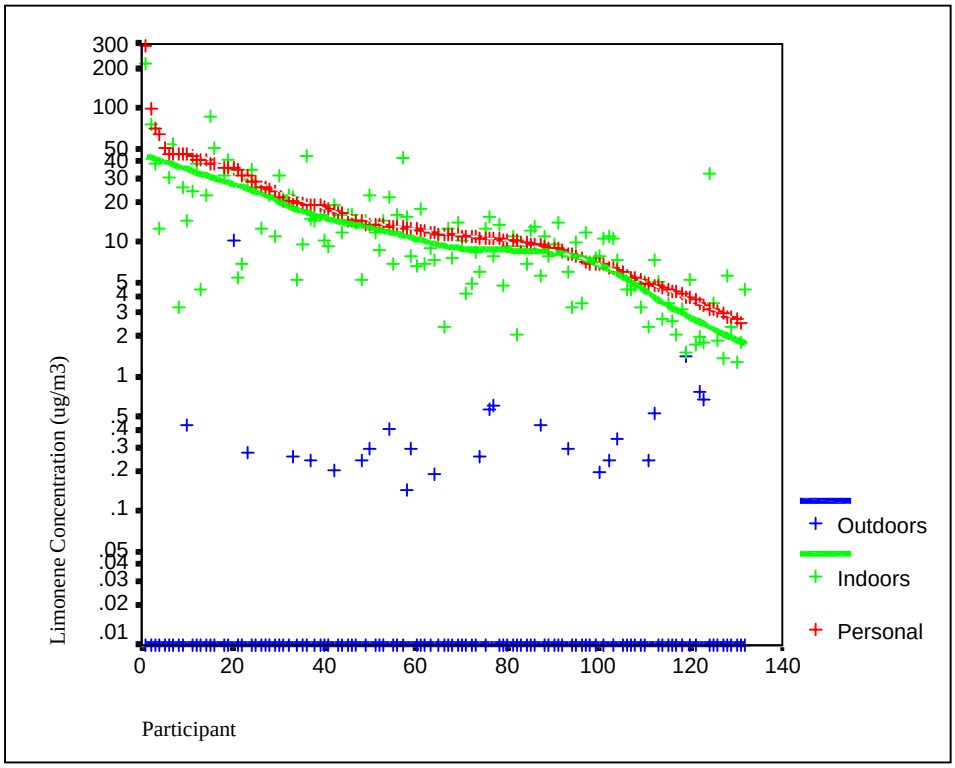


Figure 58 shows the relationships between concentrations monitored personally, indoors, and outdoors. The graph shows a very strong relationship between personal and indoor exposure concentrations such that high levels of personal exposure are consistently associated with higher levels of indoor exposure concentrations. There is no relationship between outdoor exposures and personal exposures.

**Figure 58: Relationship between Exposures to Limonene by Sampler Site**



## 7.2 Particulate Samplers

Particulate matter (PM) samples were also collected from selected participants in the Grande Prairie region. As with the PEMs, the particulate filters were deployed inside and outside the households, and attached in the area of the individual's breathing zone, and blanks were also completed for quality assurance and control purposes. Particulate matter samples were all of the $PM_{2.5}$ range (smaller air-borne particles less than 2.5 μm in size). As with the PEMs, all samples were deployed for a consecutive 7-day period.

A total of 39 Grande Prairie participants wore the particulate samplers. Table 12 shows the distribution of the 233 particulate matter filters that were used during the study.

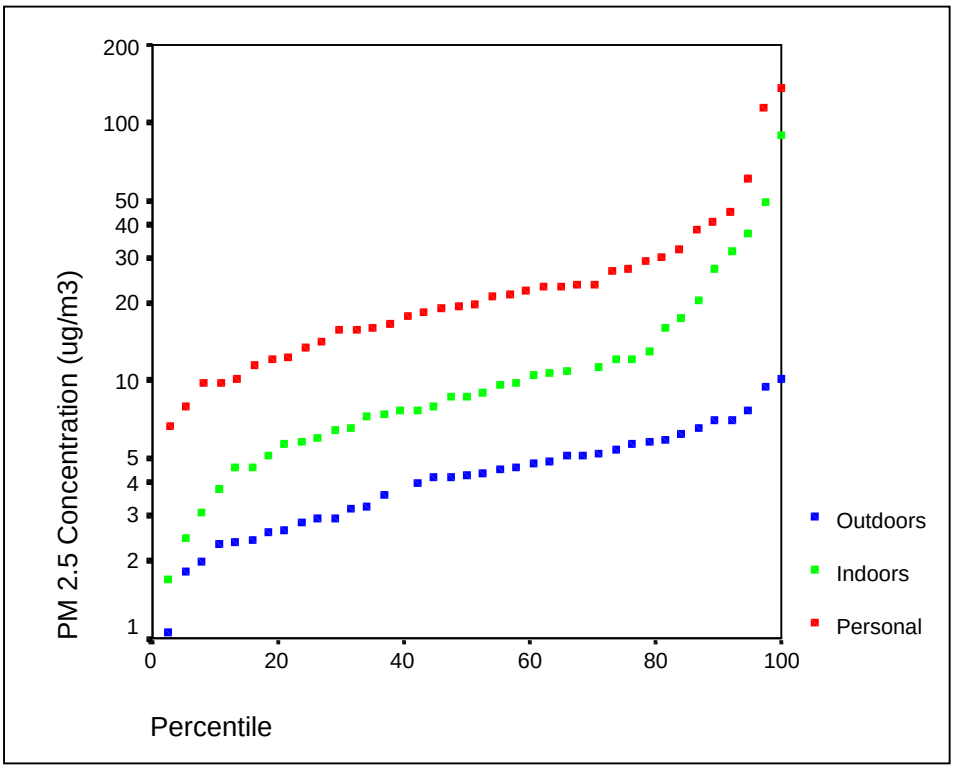
**Table 12: Distribution of Particulate Matter ($PM_{2.5}$) Filters**

| Location | Totals |
|---|---|
| Personal | 38 |
| Indoor | 66 |
| Outdoor | 66 |
| Blank | 63 |
| Total | 233 |

Figure 59 shows the cumulative distribution of $PM_{2.5}$ concentrations for the three types of samplers.

The median and 95$^{th}$ percentile $PM_{2.5}$ levels for the different locations are summarized in Table 13 and compared to guidelines and levels in other communities. The $PM_{2.5}$ guideline is currently under review and will be replaced by a Canadian wide standard of 30μg/m$^3$ over a 24-hour period by 2010. The levels of outdoor $PM_{2.5}$ levels measured in Grande Prairie were lower than other communities and well below guidelines. Median levels of indoor and personal $PM_{2.5}$ also appear low however complete analysis that includes accounting for important factors like smoking could not be done with the limited sample size.

**Figure 59: Distribution of $PM_{2.5}$**




There were too few measurements to determine temporal trends, so this graph was not produced.

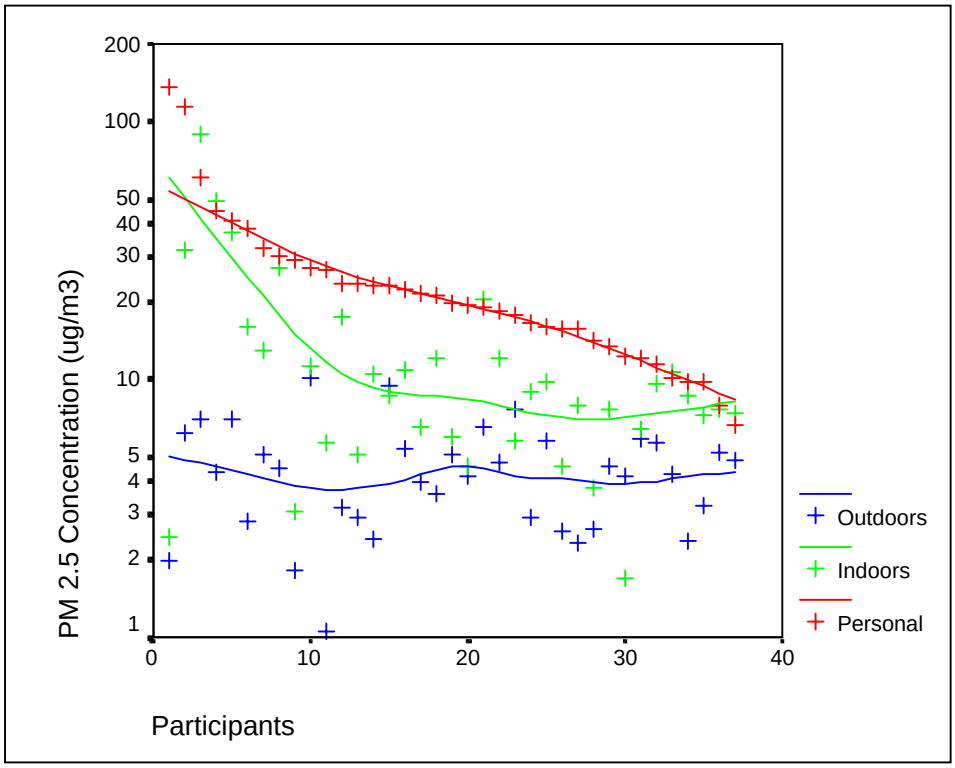
Figure 60 does show the personal exposure concentrations compared to average indoor and outdoor concentrations. There is a moderate correlation between personal and indoor concentrations, and no relationship to outdoor concentrations.

**Table 13: Comparison of $PM_{2.5}$ Levels ($\mu g/m^3$) with Guidelines and Other Studies[29, 30, 31]**

| Parameter | G.P. Median | G.P. 95th | Ft. Mc. Median | Ft. Mc. 95th | Leth. Median | Leth. 95th | Relevant Studies | Guideline/ Reference Level |
|---|---|---|---|---|---|---|---|---|
| Personal | 19.9 | 116.3 | 25 | 88 | 22.3 | 27.4 | 18.7* | N/A |
| Indoor | 8.7 | 52.9 | 8.6 | 35 | 6.7 | 12.3 | 15.4* | 40 long term*** 100 (hour)*** |
| Outdoor | 4.4 | 9.5 | 8.4 | 23.2 | 6.3 | 16.8 | 13.2* | N/A |
| Ambient Station | | | 6.2 | 13.3 | N/A | N/A | 9** | 15 (year) 65 (day) USEPA 30 (24-hour)**** |
| P/I ratio | 2.3 | 2.2 | 2.7 | 2.6 | 3.3 | 2.2 | 1.21* | N/A |
| P/O ratio | 4.6 | 12.3 | 3.20 | 4.88 | 3.55 | 1.64 | 1.42* | N/A |
| I/O ratio | 2.0 | 5.6 | 1.17 | 1.88 | 1.06 | 0.73 | 1.17* | N/A |

* Pellizzari et al., 1999.
** Cheng et al., 1998.
*** Health Canada, 1989.
**** Proposed Canadian wide standard

**Figure 60: Relationship between Exposures to $PM_{2.5}$ by Sampler Site**



# 8.0 Exposure Relationships

## 8.1 A General Model of Potential Relationships

The factors that determine the level of chemicals to which an individual is exposed are numerous, and may be very specific. The current study measured personal exposure levels integrated over one-week periods, and did not measure moment to moment concentration levels of the chemicals being monitored. This restricts the ability to provide definitive evidence of the exact causes of fluctuations in personal exposure levels. Nevertheless, a number of potential contributors to personal exposure levels were monitored and could be examined in the context of a general model of the potential causes of fluctuations in personal exposure levels. The statements below summarize some of the general expectations about relationships between exposure levels and other factors. The "→" symbol is used to postulate a causal relationship.

**Concentration Interrelations:**

Indoor concentration levels → Personal concentration levels
Outdoor concentration levels → Indoor concentration levels
Outdoor concentration levels→ Personal concentration levels

**Activity Variations:**

Fluctuations in Weekly Activity Pattern → Fluctuations in Personal concentration levels
Smoking Activity → Personal, Indoor concentration levels

**Residence Characteristics:**

Characteristics of the principal residence → Indoor, Personal concentration levels

For each of these potential relationships, variables were available. They are briefly described below, and a label is provided for use in interpreting the tables of results that follow. (Variables in brackets are reference categories against which other category members are compared).

**Exposure:**

pcon - Personal concentration levels
icon - Indoor concentration levels
ocon - Outdoor levels

**Time-Activity:**

ih Proportion of time inside the home
oh Proportion of time outside at home
iw Proportion of time inside at work
ow Proportion of time outside at work
ia Proportion of time other indoor activities
oa Proportion of time other outdoor activities
t Proportion of time in travel

**Smoking:**

| | |
|---|---|
| smkhome | Indicates if smoking occurs in the home |
| smkcar | Indicates if smoking occurs in the vehicle |
| smkamt | Number of cigarettes smoked per day (divided by 10) |
| smkexp2 | Hours per day exposed to cigarette smoke |

**Job Status:**

| | |
|---|---|
| jobft | Has a full time job |
| jobpt | Has a part time job |

**Garage**

| | |
|---|---|
| attg | Attached Garage |
| detg | Detached garage |
| (no garage) | No garage |

**Housing Characteristics:**

| | |
|---|---|
| new | Built after 1985 |
| med | Built between 1975 and 1985 |
| (old) | Built before 1975 |
| | |
| trailor | Mobile home |
| mult2 | Multiple housing (apartment or townhouse) |
| (single) | Single family detached dwelling |
| | |
| unpaved | Unpaved driveway |
| | |
| nfcdair | Indicates heating other than forced air |
| caret | Indicates presence of a cold air return |

**Urban- Rural**

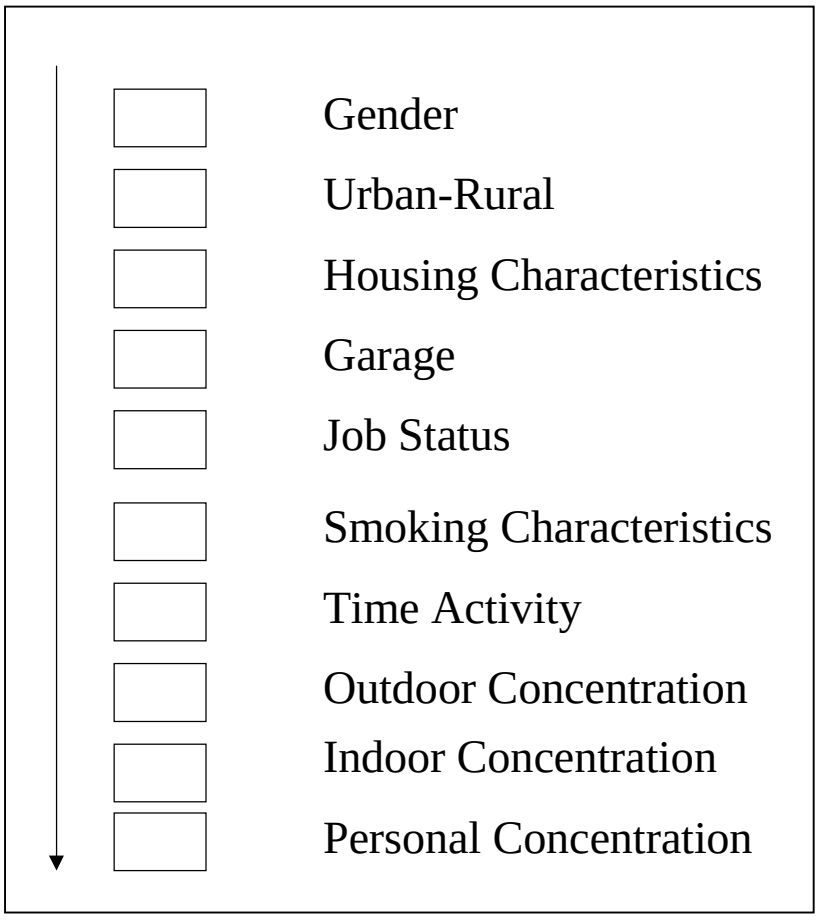
| | |
|---|---|
| urban | Grande Prairie town site vs. rural site |

**Gender**

| | |
|---|---|
| gender | Female or Male |

Further consideration of these variables and the hypothesized relationships led to the postulation of the following general recursive model to guide analysis and interpretation.

**Figure 61: A General Ordering of Factors Influencing Exposure**




A recursive ordering, such as this, is intended to capture a causal ordering among sets of variables. Specifically, as a hypothesis, it suggests that variables earlier (or higher) in the chain can have a causal effect on variables later (or lower) in the chain, but not vice versa. In addition, no reciprocal causal relations are postulated. Finally, for variables within a set, no causal ordering or priority is postulated.

There are various intuitive relationships that are captured by this ordering such as the notion that gender will influence job status, that job status will influence time and activity patterns, and that indoor concentrations will influence personal concentrations. There are a number of relationships that might be taken to be implied by the model which are not specifically intended, and which in a more detailed model could be explicitly left out (i.e. placing housing characteristics ahead of smoking characteristics in the model). There are also some relationships that may be excluded by this ordering which might nevertheless appear to obtain under some circumstances. For example, it may be postulated that indoor concentrations might have an effect on outdoor concentrations rather than the reverse. In the current model, outdoor concentrations were placed ahead of indoor concentrations because the major source of concern for exposure is the possibility that an external source leads to high indoor concentrations.

This recursive ordering was used as a heuristic device to structure the specific analyses of the concentrations of the individual chemicals. The data are blind to this ordering, and alternate hypotheses could be examined either by independent analysts or as a later follow-up to the current analyses. What the heuristic model does allow is a hypothetical partitioning of causal influence between total and direct

effects within the model. Direct effects refer to the strength of relationships directly between an independent variable or variable set and a dependent variable, while total effects include relationships between the independent variables or variable sets and the dependent variables that include other independent variables as mediators of the influence. For example, 'having a full time job' might have a total effect on 'personal exposure to octane', even though the causal force might be carried by a relationship between 'having a full time job' and 'amount of time travelling in a car' and by a relationship 'amount of time travelling in a car' and 'personal exposure to octane'. It should be noted that in the model presented in Figure 61, there are a large number of ways in which a variable group or factor may have an indirect effect on personal exposure levels.

## 8.2 Methods of Analysis

The analysis of each contaminant used regression analyses to quantify the amount of the variability in personal exposure that could be attributed to variability in each factor. The traditional measure used for this purpose is a proportion of variance, $R^2$, derived from the correlation, r, or multiple correlation, R, of the variable(s) to personal levels when the effects of including other variables in the model are taken into account. The measure $R^2$ will vary from 0.0 when there is no effect to 1.0 when personal levels can be perfectly predicted by variation in some other factor or factors. In the simplest case, where only two variables are being considered, a scatterplot of these two variables can be presented which shows the degree of relationship between them. It is usually accompanied by a correlation coefficient that quantifies the strength of that relationship and, which when squared represents the proportion of variance measure $R^2$. Unfortunately, simple scatterplots are not available as a tool when many variables are being simultaneously considered.

In general, the analysis of each contaminant proceeded as follows: a hierarchical set regression analysis[32] was performed in which variables were entered into the regression equation by set in the order specified by the recursive ordering and intermediate results were generated to give information about the relationships between variable sets. This form of analysis closely follows the logic of the recursive model in Figure 61. It can identify variables which have an indirect effect upon personal exposure levels by effecting changes in other variable sets intermediate between them and personal exposure in the recursive ordering. Such a multi-step procedure is necessary since a single analysis of all variables will obscure the intermediate relationships. In addition, since the concentration of exposures was typically positively skewed, in all cases, a generalized linear model was used in which the concentrations were assumed to follow a log normal distribution.

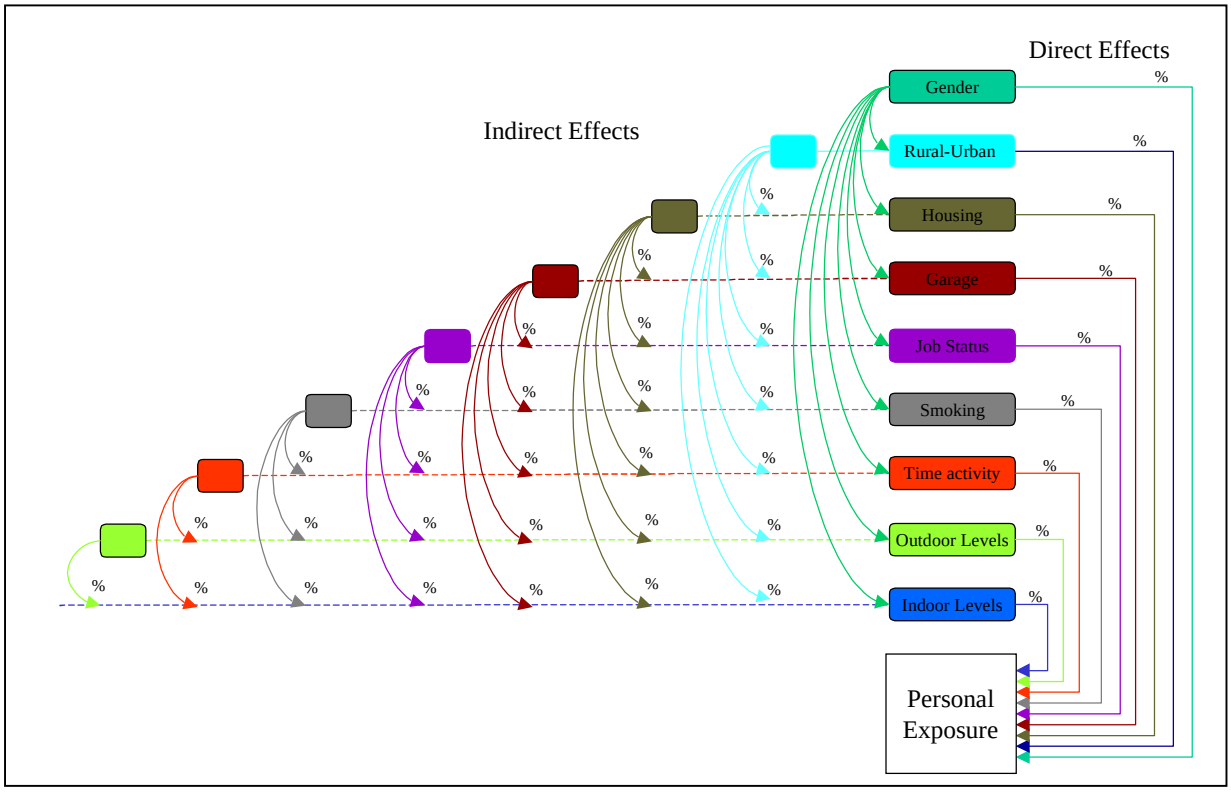
All analyses of passive samplers were conducted on 136 Grande Prairie and area residents for whom complete data were available.

## 8.3 Presentation of Results

Comparing the effect of many factors simultaneously on personal exposure can become very complex, not only because of the increased number of factors but also because of the numerous potential pathways between the factors. Communicating the results can also be difficult if the goal is to describe effects due to each factor (direct effects) as well as the numerous interrelationships between the factors (indirect effects) that may be noteworthy. In an effort to communicate these results clearly, a pictorial description of the general model used in this analysis was developed and is presented in Figure 62. The figure, which is an extension of the recursive model presented in Figure 61, shows the factor groups in colored boxes interconnected with black arrows to the box representing personal exposure. A colored arrow connecting the factor and personal exposure on the right side of the figure represents the potential direct effect of each factor group on personal exposure. The potential indirect effects of each factor on personal exposure

acting through the subsequent factors is shown by the cascading colored arrows on the left of the figure. The arrows are color coded to represent the factor groups. In subsequent sections of this report when this model is displayed for a contaminant only the largest effects and factor groups are displayed. The magnitude of the effect is written beside the arrow as a percentage and is reflected in the size of the arrow. The summations of the percentages on the figures will roughly total the variation in personal exposure described by the model and that is also noted on the figure.

**Figure 62: General Model of Personal Exposure Used to Investigate Direct and Indirect Effects**




In addition to figures such as Figure 62 that are presented for a selection of the contaminant models, two tables present the results of the hierarchical set regression conducted on each contaminant and provide the information required to construct the summary figure.

The first table presents comparative multiple correlation coefficients (Rs) derived from the hierarchical set analysis. The first column shows the total effect of the variable set in a regression analysis of personal exposure on this set of variables alone. The second column shows the total effect of the variable set with all variable sets higher in the causal ordering already entered into the regression. A decrease in the values from the first to the second column indicates that the variable sets higher in the recursive ordering had an effect on the variable set under consideration. Conversely, small differences suggest that a variable set is independent of those higher in the recursive ordering. The third column indicates the effect of a variable set (called the semi-partial R) with all other variable sets already in the regression. It indicates the direct effects of the variable set. If there is a decrease in the third column from the second column, this indicates that a variable set influences a variable set lower in the recursive ordering (and hence has an indirect effect). Small values in all columns indicate small effects. Though a detailed examination of confidence intervals was not performed, in general, multiple correlation coefficients in excess of 0.20 are likely to differ significantly from 0.0 and indicate a real effect. Clearly, the validity of this table depends upon the

validity of the chosen recursive ordering, and alternative orderings would change the values in the second column (and likely the ordering of the table which follows the recursive ordering) as well.

The square of the third column of this table (multiplied by 100) represents the percentage of the variation in personal exposure accounted for directly by a particular factor as presented on the right side of the summary figure. The total indirect effects (from which the figures on the left of the summary figure are derived) are obtained by subtracting this figure from the square of the value in the second column.

The second table reports the β weights and multiple correlation coefficient for each variable from each variable set for each stage in the recursive ordering analysis. The β weights give a method of comparing relative size of effects of different variables, though the range of variation within the sample of individuals studied, especially if small, may need to be considered in interpreting these weights. The main value of the table is that it provides insight into the relative importance of individual variables within each of the variable sets, and can also suggest direct and indirect effects for individual variables.

This table is used to partition the indirect effects of a factor between alternate pathways presented in a summary diagram. The change in the sum of the squared coefficients for the variables in a single group from column to column indicate the relative proportion of variance due to a particular set of indirect pathways (specifically that indirect pathway that is present in only one of the columns under consideration).

### 8.4 Nitrogen Dioxide ($NO_2$)

Results of the analysis of relationships between personal exposures and the factors that may affect exposure to $NO_2$ are found in Table 14. The second column of the table shows individual factors' relationships to personal exposure if considered alone. These are the $R^2$ values that resulted from simple bivariate scatterplots of the factor and personal exposure. The third column shows the amount of variation in personal exposure described by each factor in the context of the model. The fourth column shows the direct effects. Overall, the model accounted for about 72% (64% adjusted) of the variability in personal exposure levels. Table 15 shows the results of the regressions done for the modeling.

**Table 14: Summary of $NO_2$ Personal Exposure Relationships with Model Factors**

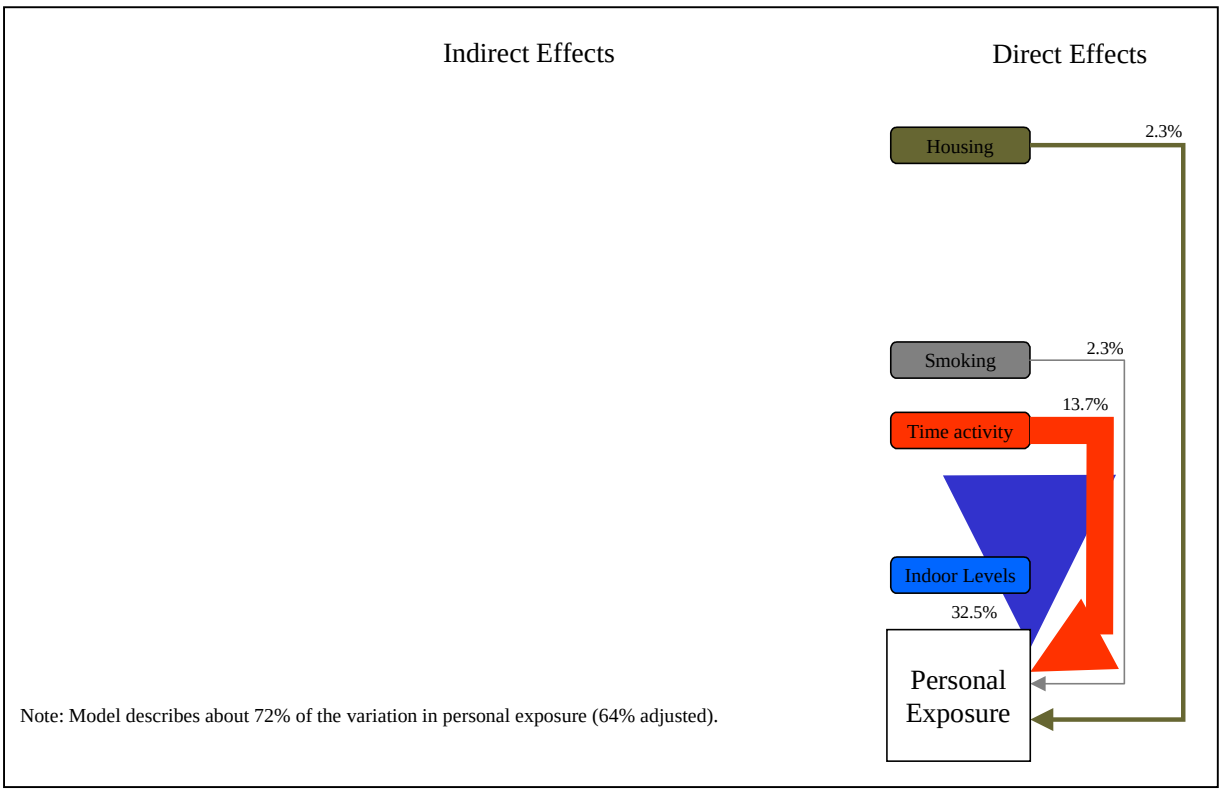
| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .09 | .09 | .01 |
| Rural-Urban | .20 | .22 | .02 |
| Housing Characteristics | .30 | .26 | .15 |
| Garage | .19 | .06 | .11 |
| Job Status | .18 | .12 | .11 |
| Smoking Characteristics | .21 | .26 | .15 |
| Time Activity | .49 | .42 | .37 |
| Outdoor Concentration | .30 | .09 | .04 |
| Indoor Concentration | .67 | .57 | .57 |

**Table 15: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.00 | .01 | -.00 | -.12 | -.12 | -.09 | -.10 | -.13 | -.09 |
| URBAN | -.04 | .04 | .12 | .13 | .11 | .14 | .18 | .22 | |
| TRAILOR | -.03 | -.15 | -.17 | -.19 | -.16 | -.18 | -.17 | | |
| MULT2 | -.01 | -.02 | -.02 | -.03 | .00 | .00 | .01 | | |
| NEW | .11 | -.05 | -.07 | -.13 | -.11 | -.10 | -.11 | | |
| MED | -.07 | -.11 | -.13 | -.13 | -.05 | -.04 | -.04 | | |
| OLD | .01 | .04 | .04 | -.01 | .01 | .01 | .00 | | |
| NFCDAIR | .06 | .19 | .19 | .14 | .11 | .15 | .15 | | |
| CARET | .01 | .08 | .07 | .06 | .02 | .02 | .02 | | |
| ATTG | -.02 | .01 | .00 | .02 | .02 | .03 | | | |
| DETG | .13 | .09 | .09 | .11 | .07 | .06 | | | |
| UNPAVED | .02 | -.03 | -.03 | -.07 | -.07 | -.06 | | | |
| JOBFT | .11 | .00 | -.06 | .12 | .08 | | | | |
| JOBPT | -.06 | -.04 | -.08 | -.01 | -.07 | | | | |
| SMKHOME | .04 | -.09 | -.09 | -.05 | | | | | |
| SMKCAR | .10 | .14 | .14 | .05 | | | | | |
| SMKAMT | -.04 | .12 | .10 | .12 | | | | | |
| SMKEXP2 | .07 | .12 | .12 | .21 | | | | | |
| IH | .14 | .00 | .01 | | | | | | |
| OH | -.18 | -.18 | -.19 | | | | | | |
| IW | .29 | .25 | .30 | | | | | | |
| OW | -.21 | -.27 | -.26 | | | | | | |
| IA | .00 | -.08 | -.06 | | | | | | |
| OA | -.01 | -.12 | -.10 | | | | | | |
| T | .14 | .10 | .10 | | | | | | |
| OCON3 | .07 | .14 | | | | | | | |
| ICON3 | .69 | | | | | | | | |
| R | .85 | .63 | .62 | .46 | .38 | .36 | .35 | .24 | .09 |

The modeling results in Tables 14 and 15 were combined and have been represented pictorially in Figure 63. Only direct effects with $R^2$ values greater than 0.02 (i.e., 2%) are displayed while indirect effects of $R^2$ greater than 5% are displayed.

**Figure 63: Results of Model of Personal Exposure to Nitrogen Dioxide**




The major effects on personal exposure levels identified in this diagram were:

- ***Indoor levels,*** directly (32.5%)
- ***Time activity,*** directly (13.7%)
- ***Smoking,*** directly (2.3%)
- ***Housing,*** directly (2.3%)

Overall, indoor variation accounted for roughly one-half of the variation in personal exposure described by the model. Time activity was also an important driver of personal exposure while smoking and housing had more minor effects.

There appears to be significant indoor sources as shown by personal exposures and indoor levels being higher than those measured outdoors. The most important factor within time activity appears to be the amount of time spent indoors at work; higher exposure is associated with more indoor work time.

### 8.5 Sulfur Dioxide ($SO_2$)

Results of the analysis of relationships between personal exposures and the factors that may affect exposure to $SO_2$ are found in Table 16. The second column of the table shows the relationship between individual factors and personal exposure if considered alone. These are the $R^2$ values that resulted from simple bivariate scatterplots of the factor and personal exposure. The third column shows the amount of variation in personal exposure described by each factor in the context of the model. The fourth column shows just the direct effects. The model accounted for about 63% (52% adjusted) of the variation in personal exposure.

**Table 16: Comparative Multiple Regression Coefficients for Variable Sets**

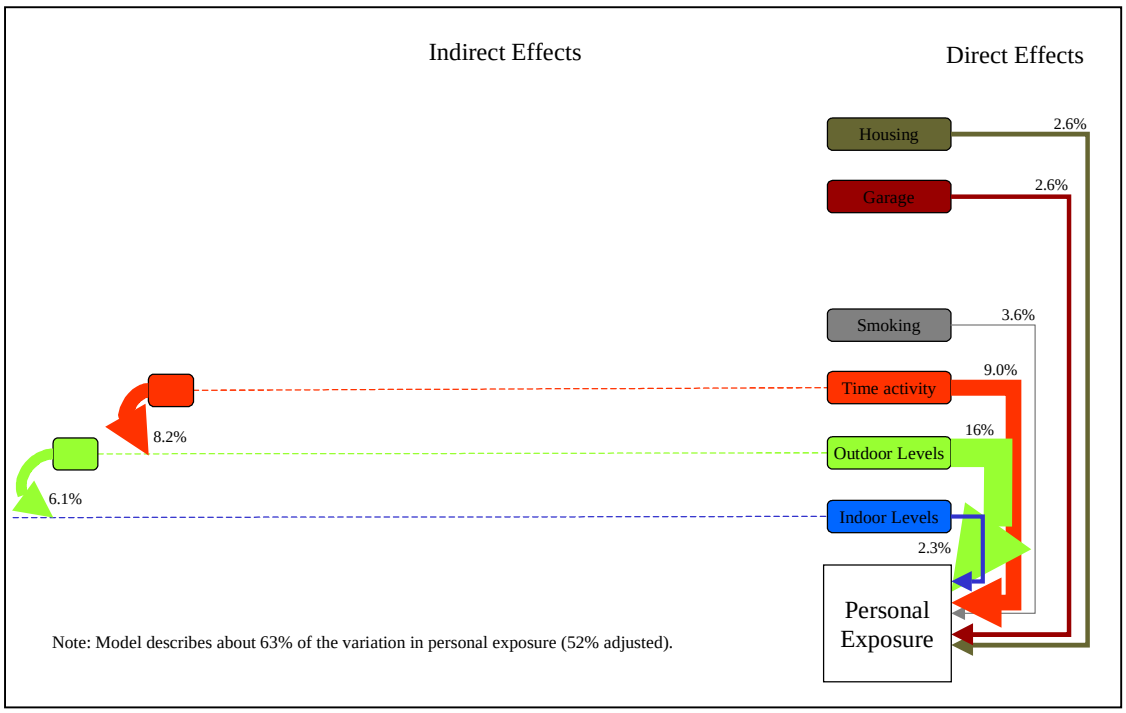
| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .03 | .03 | .06 |
| Rural-Urban | .21 | .22 | .01 |
| Housing Characteristics | .24 | .26 | .16 |
| Garage | .20 | .11 | .16 |
| Job Status | .13 | .10 | .10 |
| Smoking Characteristics | .22 | .22 | .19 |
| Time Activity | .44 | .45 | .30 |
| Outdoor Concentration | .60 | .47 | .40 |
| Indoor Concentration | .47 | .15 | .15 |

**Table 17: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.08 | -.09 | -.23 | .05 | .05 | .02 | .03 | .07 | .03 |
| URBAN | .01 | -.02 | -.01 | -.12 | -.13 | -.16 | -.19 | -.22 | |
| TRAILOR | .04 | .04 | .07 | .11 | .05 | .06 | .05 | | |
| MULT2 | -.09 | -.10 | .03 | .10 | .08 | .08 | .04 | | |
| NEW | .24 | .31 | .56 | .47 | .49 | .49 | .50 | | |
| MED | .18 | .18 | .48 | .40 | .41 | .40 | .38 | | |
| OLD | .11 | .11 | .37 | .30 | .32 | .32 | .34 | | |
| NFCDAIR | -.02 | -.02 | -.04 | -.05 | -.04 | -.07 | -.07 | | |
| CARET | -.02 | -.05 | -.02 | .04 | .00 | .00 | -.01 | | |
| ATTG | -.21 | -.20 | -.07 | -.10 | -.10 | -.10 | | | |
| DETG | -.15 | -.12 | -.06 | -.18 | -.17 | -.15 | | | |
| UNPAVED | -.04 | -.04 | -.04 | .00 | .02 | .00 | | | |
| JOBFT | -.19 | -.20 | -.25 | .01 | -.05 | | | | |
| JOBPT | -.08 | -.07 | -.07 | .12 | .07 | | | | |
| SMKHOME | .16 | .13 | .16 | .08 | | | | | |
| SMKCAR | -.14 | -.14 | -.16 | -.07 | | | | | |
| SMKAMT | .18 | .20 | .25 | .22 | | | | | |
| SMKEXP2 | -.09 | -.09 | -.11 | -.11 | | | | | |
| IH | .19 | .21 | .18 | | | | | | |
| OH | .24 | .28 | .45 | | | | | | |
| IW | .20 | .23 | .31 | | | | | | |
| OW | .29 | .29 | .34 | | | | | | |
| IA | .10 | .09 | .06 | | | | | | |
| OA | .13 | .12 | .06 | | | | | | |
| T | .21 | .23 | .28 | | | | | | |
| OCON3 | .49 | .55 | | | | | | | |
| ICON3 | .18 | | | | | | | | |
| R | .79 | .78 | .62 | .43 | .37 | .36 | .34 | .22 | .03 |

The modeling results in Tables 16 and 17 were combined and have been represented pictorially in Figure 64. Only direct effects with $R^2$ values greater than 0.02 (i.e., 2%) are displayed while indirect effects of $R^2$ greater than 5% are displayed.

**Figure 64: Results of Model of Personal Exposure to Sulfur Dioxide**




A qualitative estimate of the pathways of the indirect effects has been made. The major effects identified in the analysis were as follows:

- ***Outdoor levels,*** directly (16%)
- ***Time activity,*** directly (9.0%)
- ***Time activity,*** operating indirectly apparently through effects on outdoor levels (8.2%)
- ***Outdoor levels,*** operating indirectly through effects on indoor levels (5.1%)
- ***Smoking,*** directly (3.6%)
- ***Housing,*** directly (2.6%)
- ***Garage,*** directly (2.6%)
- ***Indoor levels,*** directly (2.3%)

Overall, variations of outdoor levels accounted for about half the variation in personal exposure described by the model acting either directly, indirectly through indoor air or influenced by time activity. The influence of time activity on outdoor level is not immediately understandable but may be related to the location of the participants home in the areas (i.e. time activity patterns and outdoor $SO_2$ may correlate for people living in the core of the community compared to the suburbs). Time activity also has a large direct effect. It tends to be associated with time spent travelling or outdoors. The other effects listed are relatively minor.

### 8.6 Ozone ($O_3$)

The results of the analysis comparing effects of factors on personal $O_3$ exposure is shown in Tables 18 and 19 and pictorially in Figure 65.

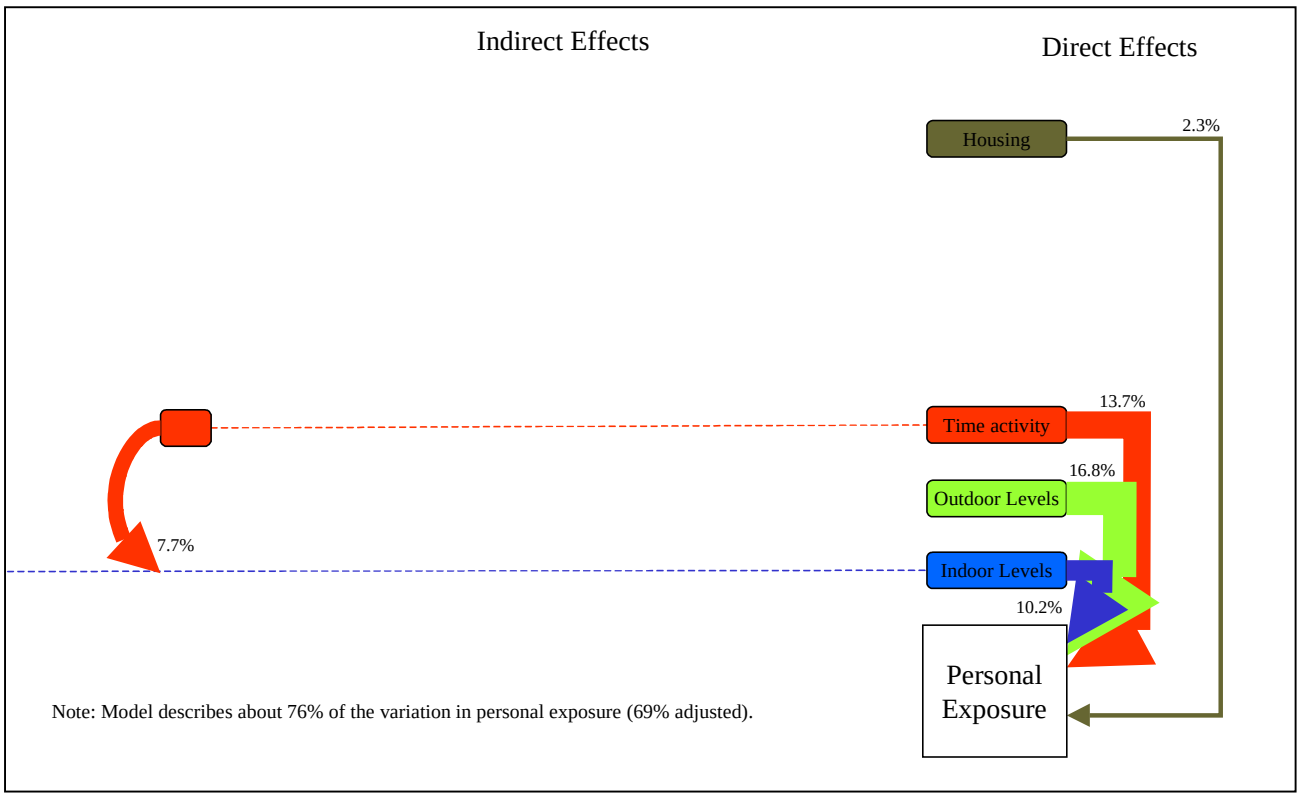
**Table 18: Summary of $O_3$ Personal Exposure Relationships with Model Factors**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .14 | .14 | .01 |
| Rural-Urban | .25 | .28 | .06 |
| Housing Characteristics | .16 | .22 | .15 |
| Garage | .18 | .19 | .04 |
| Job Status | .15 | .14 | .07 |
| Smoking Characteristics | .21 | .19 | .09 |
| Time Activity | .59 | .49 | .37 |
| Outdoor Concentration | .49 | .42 | .41 |
| Indoor Concentration | .57 | .32 | .32 |

**Table 19: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.01 | .01 | -.04 | .23 | .23 | .19 | .15 | .19 | .14 |
| URBAN | -.08 | -.07 | -.17 | -.26 | -.25 | -.29 | -.27 | -.29 | |
| TRAILOR | .00 | -.01 | .09 | .15 | .12 | .14 | .10 | | |
| MULT2 | .02 | .11 | .20 | .21 | .21 | .21 | .11 | | |
| NEW | .06 | .06 | .25 | .18 | .19 | .20 | .30 | | |
| MED | .08 | .13 | .32 | .29 | .31 | .31 | .31 | | |
| OLD | .21 | .19 | .40 | .35 | .38 | .39 | .41 | | |
| NFCDAIR | -.04 | -.10 | -.06 | -.02 | .01 | -.03 | -.03 | | |
| CARET | .02 | -.03 | .04 | .02 | .00 | -.01 | -.02 | | |
| ATTG | .04 | .10 | .12 | .14 | .15 | .15 | | | |
| DETG | .04 | .10 | .00 | -.14 | -.14 | -.12 | | | |
| UNPAVED | -.05 | -.06 | -.07 | .00 | .02 | .00 | | | |
| JOBFT | -.03 | .01 | .03 | .04 | -.04 | | | | |
| JOBPT | .04 | .14 | .12 | .18 | .13 | | | | |
| SMKHOME | -.06 | -.01 | .00 | -.10 | | | | | |
| SMKCAR | -.01 | -.02 | -.07 | .00 | | | | | |
| SMKAMT | .08 | .12 | .21 | .21 | | | | | |
| SMKEXP2 | .04 | .04 | .04 | -.03 | | | | | |
| IH | .14 | .19 | .26 | | | | | | |
| OH | .20 | .33 | .44 | | | | | | |
| IW | .06 | .00 | .11 | | | | | | |
| OW | .18 | .21 | .18 | | | | | | |
| IA | .07 | .11 | .15 | | | | | | |
| OA | .38 | .49 | .41 | | | | | | |
| T | .12 | .11 | .18 | | | | | | |
| OCON3 | .48 | .51 | | | | | | | |
| ICON3 | .40 | | | | | | | | |
| R | .87 | .81 | .70 | .49 | .45 | .43 | .39 | .32 | .14 |

**Figure 65: Results of Model of Personal Exposure to Ozone**




The model predicted about 70% of the variation in personal $O_3$ exposure across individuals and days. Important factors influencing variations in $O_3$ exposures were as follows:

- ***Outdoor levels,*** directly (16.8%)
- ***Time activity,*** directly (13.7%)
- ***Indoor levels,*** directly (10.2%)
- ***Time activity,*** operating indirectly through effects on indoor air (7.7%)
- ***Housing,*** directly (2.3%)

The variation in personal exposure described by the model was due to outdoor levels and time activity acting directly and indirectly through indoor levels. Indoor concentrations were also an important factor and housing characteristics were found to be of relatively minor importance.

### 8.7 Volatile Organic Chemicals

The results of the investigation into the VOCs will be presented as a group. A more focused discussion is presented for benzene due to the unique exposure pattern and the health concerns associated with this compound.

Tables 20 to 45 show the modeled results for all the VOCs investigated. Figures 66 to 78 show pictorial representations of the exposure model results for these compounds.

All the VOC compounds investigated in this study except benzene demonstrate a pattern of exposure that showed the variation in indoor air levels dominated personal exposure and account for at least half of the variation explained by the model. Benzene exposure showed the factor of time activity patterns to be slightly more important then indoor air levels with each factor accounting for roughly $1/5^{th}$ of the variation in personal exposure. Outdoor air levels acting through indoor air was the next most important factor to benzene exposure. Among the time activity factors, time spent outdoor at home and at work were the important influences.

Exposure to butanone was another VOC compound that showed evidence of outdoor air influence on exposure, again, acting through indoor air. Hexane and ethylbenzene also showed exposure influenced by factors acting through indoor air. The factors time activity, garage, housing, and smoking generally showed significant direct effects for nearly all the compounds. Time activity was the most important of this group of factors. It should be emphasized that all of these factors are minor in comparison to indoor concentration levels.

**Figure 66: Results of Model of Personal Exposure to Benzene**

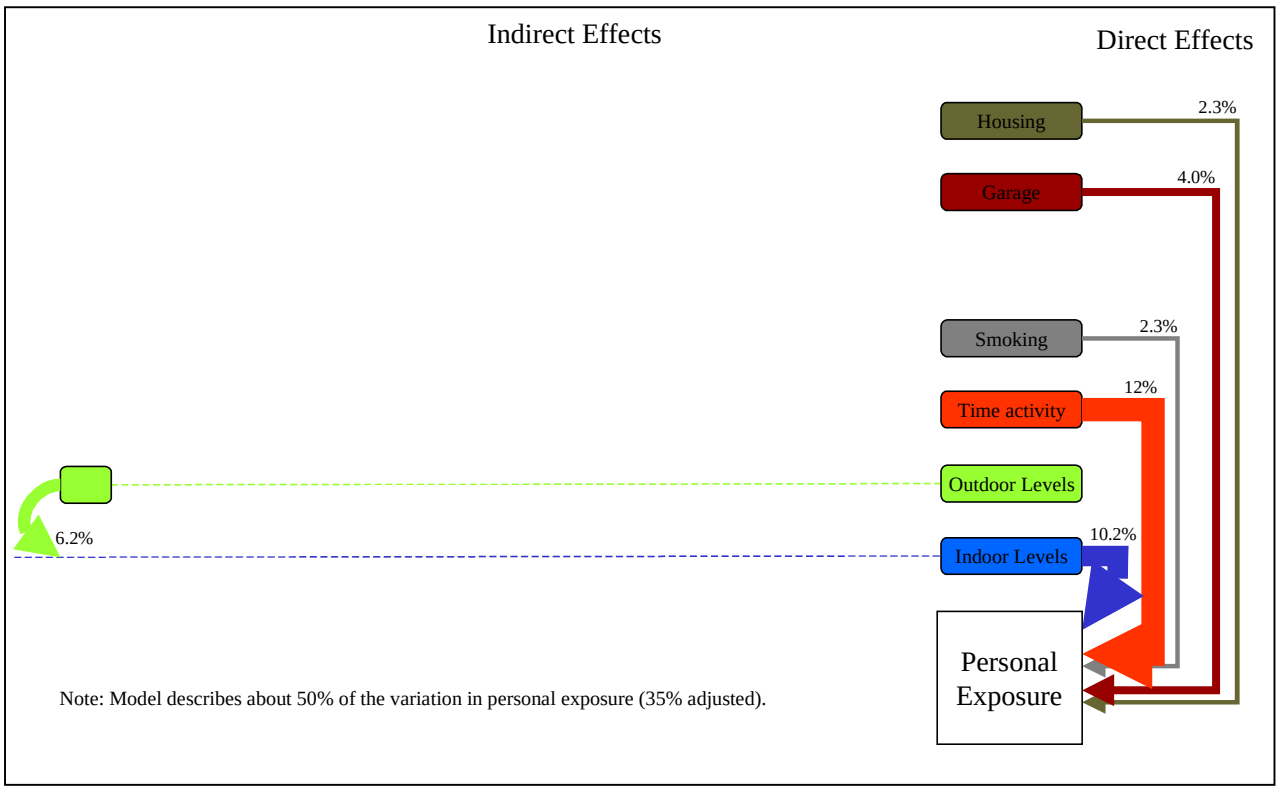



**Table 20: Comparative Multiple Regression Coefficients for Variable Sets for Benzene**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .02 | .02 | .08 |
| Rural-Urban | .16 | .16 | .03 |
| Housing Characteristics | .28 | .27 | .15 |
| Garage | .36 | .30 | .20 |
| Job Status | .08 | .08 | .05 |
| Smoking Characteristics | .27 | .23 | .15 |
| Time Activity | .29 | .26 | .34 |
| Outdoor Concentration | .20 | .28 | .13 |
| Indoor Concentration | .50 | .31 | .32 |

**Table 21: Comparative Multiple Regression Coefficients for Variable Sets for Benzene**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.09 | -.06 | -.07 | .05 | .06 | .08 | .06 | .04 | .02 |
| URBAN | -.04 | -.15 | -.08 | -.12 | -.15 | -.13 | -.15 | -.16 | |
| TRAILOR | -.01 | -.09 | -.08 | -.10 | -.08 | -.08 | -.15 | | |
| MULT2 | -.12 | -.11 | .01 | .02 | .03 | .03 | -.14 | | |
| NEW | .05 | .02 | .05 | -.08 | -.09 | -.08 | .03 | | |
| MED | .04 | .07 | .14 | .01 | .05 | .07 | .05 | | |
| OLD | .04 | .04 | .16 | .03 | .02 | .03 | .07 | | |
| NFCDAIR | -.07 | -.07 | -.05 | -.05 | -.08 | -.06 | -.04 | | |
| CARET | .03 | .08 | .15 | .16 | .13 | .13 | .10 | | |
| ATTG | -.27 | -.03 | -.02 | -.04 | -.04 | -.03 | | | |
| DETG | -.19 | -.22 | -.31 | -.34 | -.38 | -.39 | | | |
| UNPAVED | .01 | -.01 | -.05 | -.02 | -.01 | .00 | | | |
| JOBFT | .08 | .09 | .09 | .09 | .08 | | | | |
| JOBPT | -.01 | .02 | -.01 | .02 | -.01 | | | | |
| SMKHOME | -.02 | .08 | .11 | .10 | | | | | |
| SMKCAR | -.02 | .02 | -.05 | -.01 | | | | | |
| SMKAMT | .06 | .08 | .08 | .03 | | | | | |
| SMKEXP2 | .16 | .23 | .23 | .19 | | | | | |
| IH | -.18 | -.17 | -.17 | | | | | | |
| OH | .19 | .16 | .10 | | | | | | |
| IW | -.17 | -.17 | -.16 | | | | | | |
| OW | .18 | .15 | .11 | | | | | | |
| IA | -.04 | .00 | -.01 | | | | | | |
| OA | .01 | -.06 | -.11 | | | | | | |
| T | .07 | .09 | .08 | | | | | | |
| OCON3 | .19 | .35 | | | | | | | |
| ICON3 | .50 | | | | | | | | |
| R | .70 | .63 | .56 | .50 | .44 | .44 | .32 | .16 | .02 |

### *Toluene*

**Figure 67: Results of Model of Personal Exposure to Toluene**

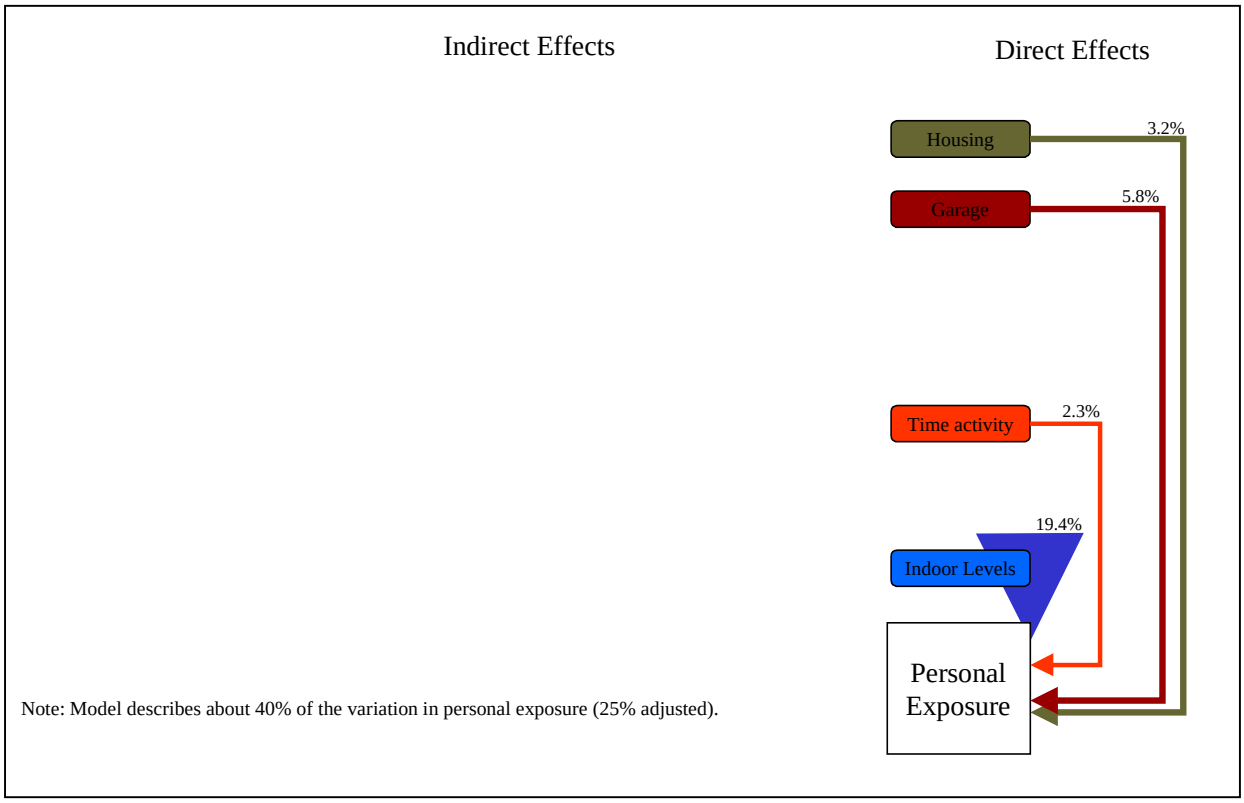



**Table 22: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .04 | 0.04 | .10 |
| Rural-Urban | .01 | 0.01 | .01 |
| Housing Characteristics | .35 | 0.36 | .18 |
| Garage | .28 | 0.19 | .24 |
| Job Status | .11 | 0.08 | .02 |
| Smoking Characteristics | .12 | 0.09 | .04 |
| Time Activity | .11 | 0.10 | .15 |
| Outdoor Concentration | .17 | 0.16 | .03 |
| Indoor Concentration | .51 | 0.44 | .44 |

**Table 23: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .13 | .14 | .10 | .07 | .07 | .08 | .09 | .04 | .04 |
| URBAN | .01 | -.02 | .01 | .02 | .01 | .03 | .01 | -.01 | |
| TRAILOR | -.15 | -.13 | -.14 | -.16 | -.18 | -.18 | -.21 | | |
| MULT2 | -.14 | -.19 | -.18 | -.20 | -.21 | -.20 | -.27 | | |
| NEW | -.05 | .01 | .05 | .02 | .02 | .04 | .05 | | |
| MED | .02 | .08 | .11 | .07 | .07 | .09 | .07 | | |
| OLD | -.10 | -.13 | -.04 | -.08 | -.08 | -.07 | -.06 | | |
| NFCDAIR | -.04 | .00 | .02 | .02 | .01 | .02 | .04 | | |
| CARET | -.02 | .08 | .10 | .11 | .09 | .08 | .07 | | |
| ATTG | -.32 | -.16 | -.19 | -.19 | -.19 | -.18 | | | |
| DETG | -.22 | -.27 | -.28 | -.25 | -.25 | -.26 | | | |
| UNPAVED | -.12 | -.05 | -.06 | -.06 | -.05 | -.04 | | | |
| JOBFT | .01 | .12 | .14 | .10 | .09 | | | | |
| JOBPT | -.01 | .09 | .05 | .02 | .01 | | | | |
| SMKHOME | -.02 | .06 | .06 | .08 | | | | | |
| SMKCAR | -.01 | -.02 | .00 | -.01 | | | | | |
| SMKAMT | .05 | .07 | .07 | .06 | | | | | |
| SMKEXP2 | .00 | -.03 | -.04 | -.05 | | | | | |
| IH | -.07 | -.15 | -.12 | | | | | | |
| OH | .04 | -.13 | -.08 | | | | | | |
| IW | -.05 | -.15 | -.12 | | | | | | |
| OW | .04 | -.03 | -.02 | | | | | | |
| IA | .10 | -.01 | .00 | | | | | | |
| OA | .03 | -.09 | -.08 | | | | | | |
| T | -.14 | -.12 | -.06 | | | | | | |
| OCON3 | .03 | .18 | | | | | | | |
| ICON3 | .57 | | | | | | | | |
| R | .64 | .46 | .43 | .42 | .41 | .41 | .36 | .04 | .04 |

### *Ethylbenzene*

**Figure 68: Results of Model of Personal Exposure to Ethylbenzene**

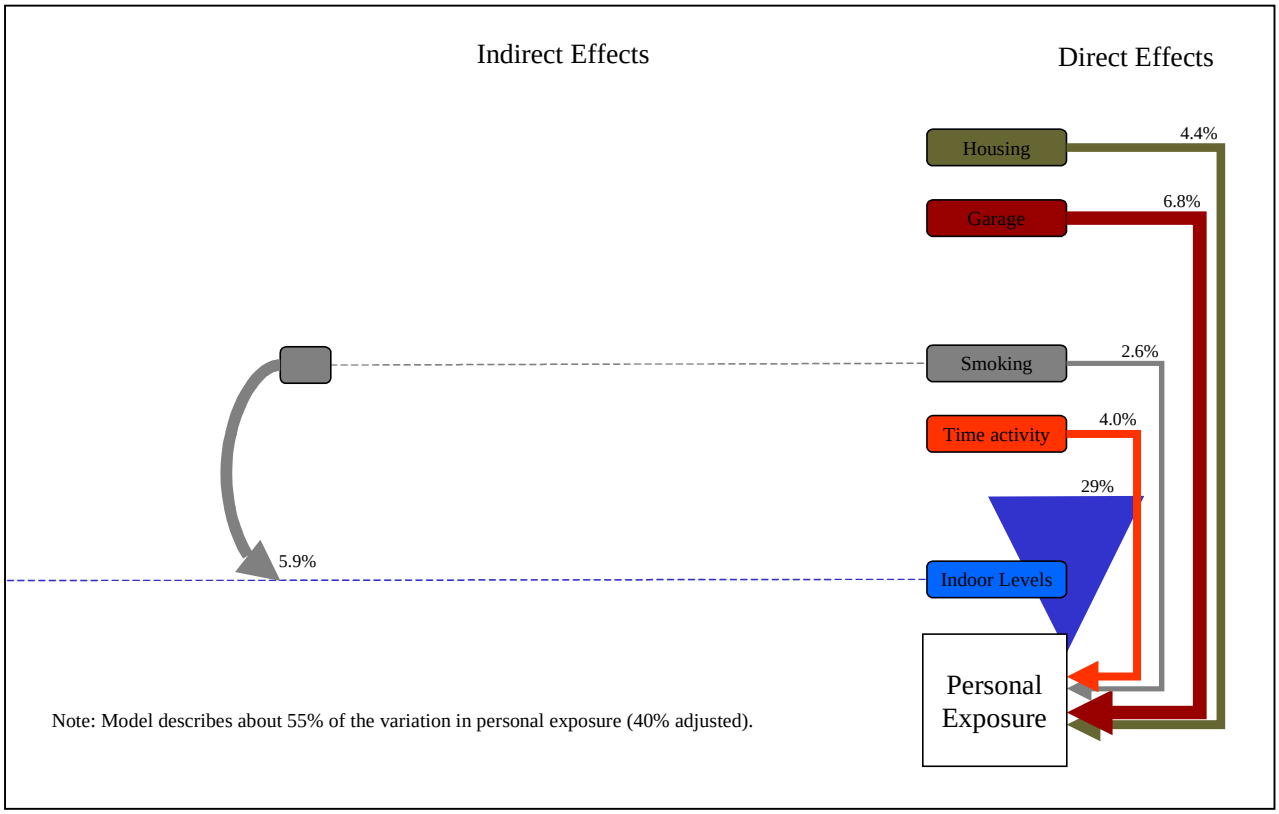



**Table 24: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .10 | .10 | .05 |
| Rural-Urban | .04 | .02 | .05 |
| Housing Characteristics | .25 | .27 | .21 |
| Garage | .20 | .15 | .26 |
| Job Status | .16 | .13 | .09 |
| Smoking Characteristics | .27 | .29 | .16 |
| Time Activity | .15 | .22 | .20 |
| Outdoor Concentration | .08 | .04 | .12 |
| Indoor Concentration | .59 | .54 | .54 |

**Table 25: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .05 | .07 | .06 | .06 | .07 | .11 | .11 | .09 | .10 |
| URBAN | .06 | .00 | .01 | -.02 | -.06 | -.02 | .03 | .03 | |
| TRAILOR | -.15 | -.13 | -.13 | -.14 | -.13 | -.15 | -.16 | | |
| MULT2 | -.09 | -.03 | -.02 | -.05 | -.05 | -.05 | -.10 | | |
| NEW | .03 | .20 | .20 | .17 | .14 | .14 | .14 | | |
| MED | .14 | .16 | .17 | .13 | .17 | .18 | .16 | | |
| OLD | .06 | .18 | .20 | .15 | .13 | .12 | .12 | | |
| NFCDAIR | .00 | -.01 | -.01 | -.02 | -.05 | -.02 | .00 | | |
| CARET | .10 | .19 | .20 | .17 | .11 | .12 | .11 | | |
| ATTG | -.37 | -.12 | -.12 | -.10 | -.11 | -.10 | | | |
| DETG | -.17 | -.09 | -.09 | -.09 | -.13 | -.15 | | | |
| UNPAVED | -.10 | -.19 | -.20 | -.17 | -.14 | -.13 | | | |
| JOBFT | .14 | .30 | .30 | .10 | .06 | | | | |
| JOBPT | .02 | .05 | .05 | -.05 | -.10 | | | | |
| SMKHOME | .10 | .17 | .17 | .18 | | | | | |
| SMKCAR | -.14 | -.09 | -.09 | -.09 | | | | | |
| SMKAMT | .10 | .11 | .11 | .12 | | | | | |
| SMKEXP2 | .14 | .25 | .25 | .19 | | | | | |
| IH | -.01 | -.11 | -.12 | | | | | | |
| OH | .14 | .02 | .02 | | | | | | |
| IW | -.08 | -.29 | -.29 | | | | | | |
| OW | .05 | -.14 | -.14 | | | | | | |
| IA | .06 | -.02 | -.02 | | | | | | |
| OA | .12 | .05 | .04 | | | | | | |
| T | -.02 | -.04 | -.03 | | | | | | |
| OCON3 | -.15 | .04 | | | | | | | |
| ICON3 | .68 | | | | | | | | |
| R | .74 | .51 | .50 | .45 | .35 | .32 | .28 | .10 | .10 |

### *O-Xylene*

**Figure 69: Results of Model of Personal Exposure to O-Xylene**

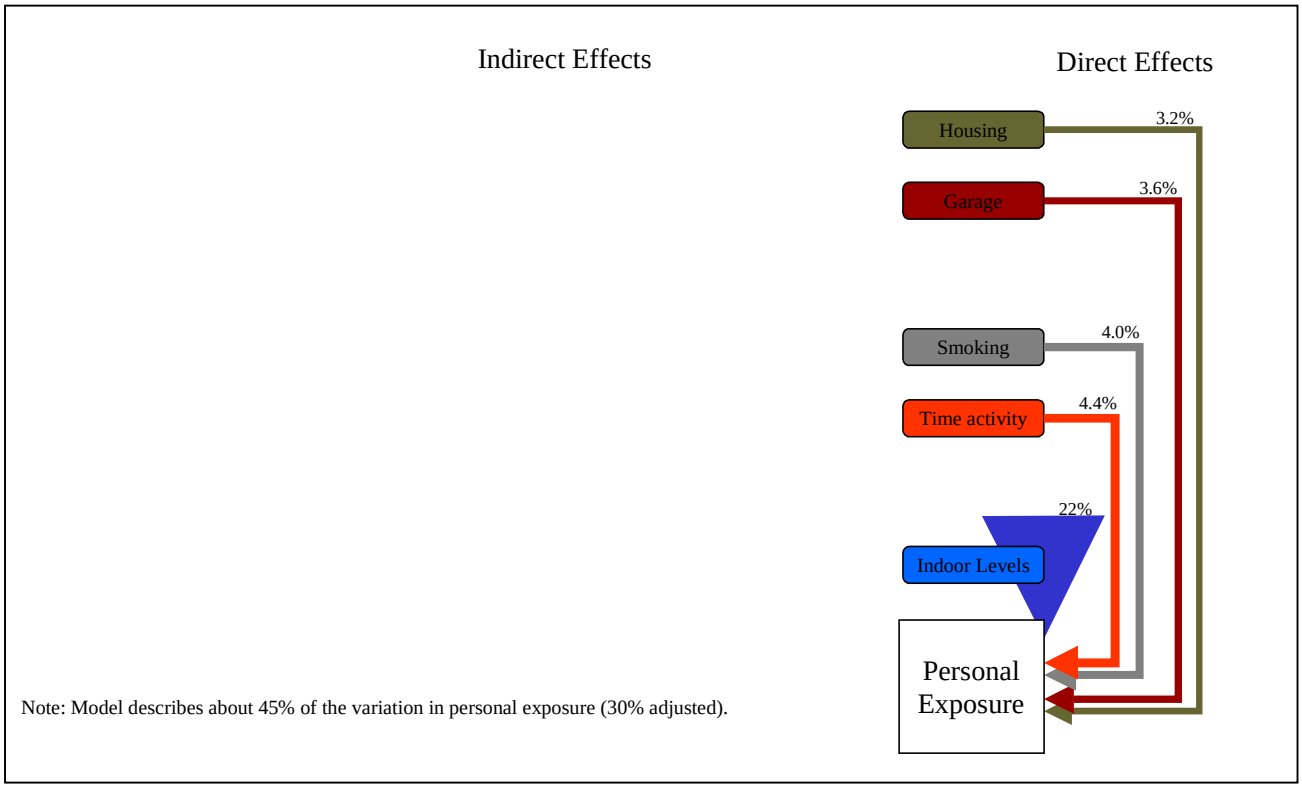



**Table 26: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .04 | 0.04 | .00 |
| Rural-Urban | .01 | 0.00 | .05 |
| Housing Characteristics | .25 | 0.26 | .18 |
| Garage | .17 | 0.12 | .19 |
| Job Status | .14 | 0.13 | .10 |
| Smoking Characteristics | .27 | 0.30 | .20 |
| Time Activity | .17 | 0.22 | .21 |
| Outdoor Concentration | .05 | 0.02 | .05 |
| Indoor Concentration | .55 | 0.47 | .47 |

**Table 27: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.02 | .00 | -.01 | .03 | .04 | .06 | .05 | .04 | .04 |
| URBAN | .08 | .00 | .01 | -.03 | -.07 | -.04 | .01 | .00 | |
| TRAILOR | -.10 | -.11 | -.11 | -.12 | -.11 | -.12 | -.15 | | |
| MULT2 | -.09 | .01 | .02 | .00 | .00 | .00 | -.06 | | |
| NEW | .00 | .12 | .12 | .09 | .08 | .09 | .12 | | |
| MED | .06 | .11 | .11 | .08 | .13 | .15 | .15 | | |
| OLD | .02 | .14 | .15 | .11 | .09 | .10 | .11 | | |
| NFCDAIR | .03 | .00 | .01 | -.01 | -.05 | -.02 | .00 | | |
| CARET | .12 | .24 | .25 | .21 | .16 | .16 | .15 | | |
| ATTG | -.27 | -.02 | -.02 | -.01 | -.02 | .00 | | | |
| DETG | -.06 | -.03 | -.03 | -.06 | -.10 | -.12 | | | |
| UNPAVED | -.04 | -.15 | -.15 | -.12 | -.09 | -.08 | | | |
| JOBFT | .18 | .33 | .32 | .16 | .13 | | | | |
| JOBPT | .08 | .10 | .10 | .03 | -.02 | | | | |
| SMKHOME | .05 | .18 | .18 | .18 | | | | | |
| SMKCAR | -.09 | -.07 | -.07 | -.07 | | | | | |
| SMKAMT | .13 | .12 | .12 | .13 | | | | | |
| SMKEXP2 | .18 | .22 | .22 | .18 | | | | | |
| IH | .04 | -.02 | -.03 | | | | | | |
| OH | .20 | .11 | .12 | | | | | | |
| IW | -.02 | -.18 | -.18 | | | | | | |
| OW | .03 | -.12 | -.12 | | | | | | |
| IA | .02 | -.02 | -.02 | | | | | | |
| OA | .14 | .08 | .08 | | | | | | |
| T | .05 | .03 | .03 | | | | | | |
| OCON3 | -.08 | .03 | | | | | | | |
| ICON3 | .61 | | | | | | | | |
| R | .68 | .49 | .49 | .43 | .31 | .29 | .26 | .04 | .04 |

### *M-, P-Xylene*

**Figure 70: Results of Model of Personal Exposure to M-, P-Xylene**

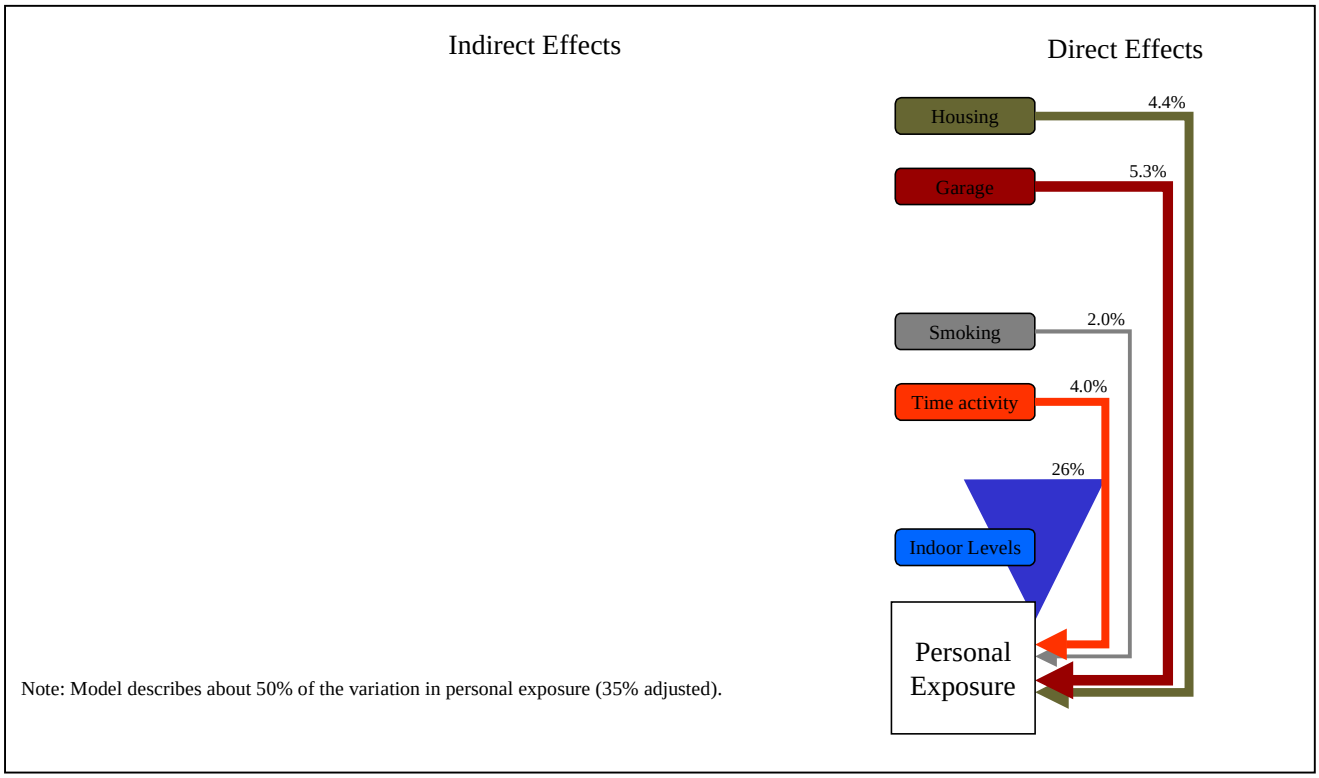



**Table 28: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .12 | 0.12 | .07 |
| Rural-Urban | .05 | 0.03 | .06 |
| Housing Characteristics | .29 | 0.31 | .21 |
| Garage | .22 | 0.15 | .23 |
| Job Status | .16 | 0.12 | .07 |
| Smoking Characteristics | .23 | 0.25 | .14 |
| Time Activity | .15 | 0.21 | .20 |
| Outdoor Concentration | .09 | 0.01 | .08 |
| Indoor Concentration | .58 | 0.51 | .51 |

**Table 29: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .08 | .09 | .09 | .09 | .11 | .14 | .13 | .12 | .12 |
| URBAN | .09 | .01 | .01 | -.01 | -.05 | -.02 | .04 | .03 | |
| TRAILOR | -.16 | -.15 | -.15 | -.16 | -.16 | -.17 | -.20 | | |
| MULT2 | -.12 | -.02 | -.02 | -.04 | -.04 | -.04 | -.11 | | |
| NEW | .00 | .17 | .17 | .13 | .11 | .12 | .14 | | |
| MED | .10 | .15 | .15 | .11 | .14 | .15 | .14 | | |
| OLD | .05 | .20 | .20 | .14 | .13 | .12 | .13 | | |
| NFCDAIR | .02 | .01 | .01 | .00 | -.03 | .00 | .02 | | |
| CARET | .09 | .24 | .24 | .21 | .16 | .16 | .15 | | |
| ATTG | -.33 | -.06 | -.06 | -.05 | -.05 | -.05 | | | |
| DETG | -.14 | -.10 | -.10 | -.10 | -.13 | -.15 | | | |
| UNPAVED | -.10 | -.18 | -.18 | -.16 | -.13 | -.12 | | | |
| JOBFT | .10 | .26 | .25 | .08 | .05 | | | | |
| JOBPT | .03 | .04 | .03 | -.05 | -.09 | | | | |
| SMKHOME | .07 | .17 | .17 | .18 | | | | | |
| SMKCAR | -.10 | -.06 | -.06 | -.06 | | | | | |
| SMKAMT | .09 | .11 | .11 | .12 | | | | | |
| SMKEXP2 | .11 | .17 | .17 | .12 | | | | | |
| IH | -.02 | -.11 | -.11 | | | | | | |
| OH | .13 | .02 | .03 | | | | | | |
| IW | -.08 | -.25 | -.25 | | | | | | |
| OW | .06 | -.11 | -.11 | | | | | | |
| IA | .05 | .00 | .00 | | | | | | |
| OA | .13 | .05 | .05 | | | | | | |
| T | .00 | -.03 | -.02 | | | | | | |
| OCON3 | -.14 | .01 | | | | | | | |
| ICON3 | .66 | | | | | | | | |
| R | .72 | .50 | .50 | .46 | .38 | .37 | .33 | .13 | .12 |

### *Limonene*

**Figure 71: Results of Model of Personal Exposure to Limonene**

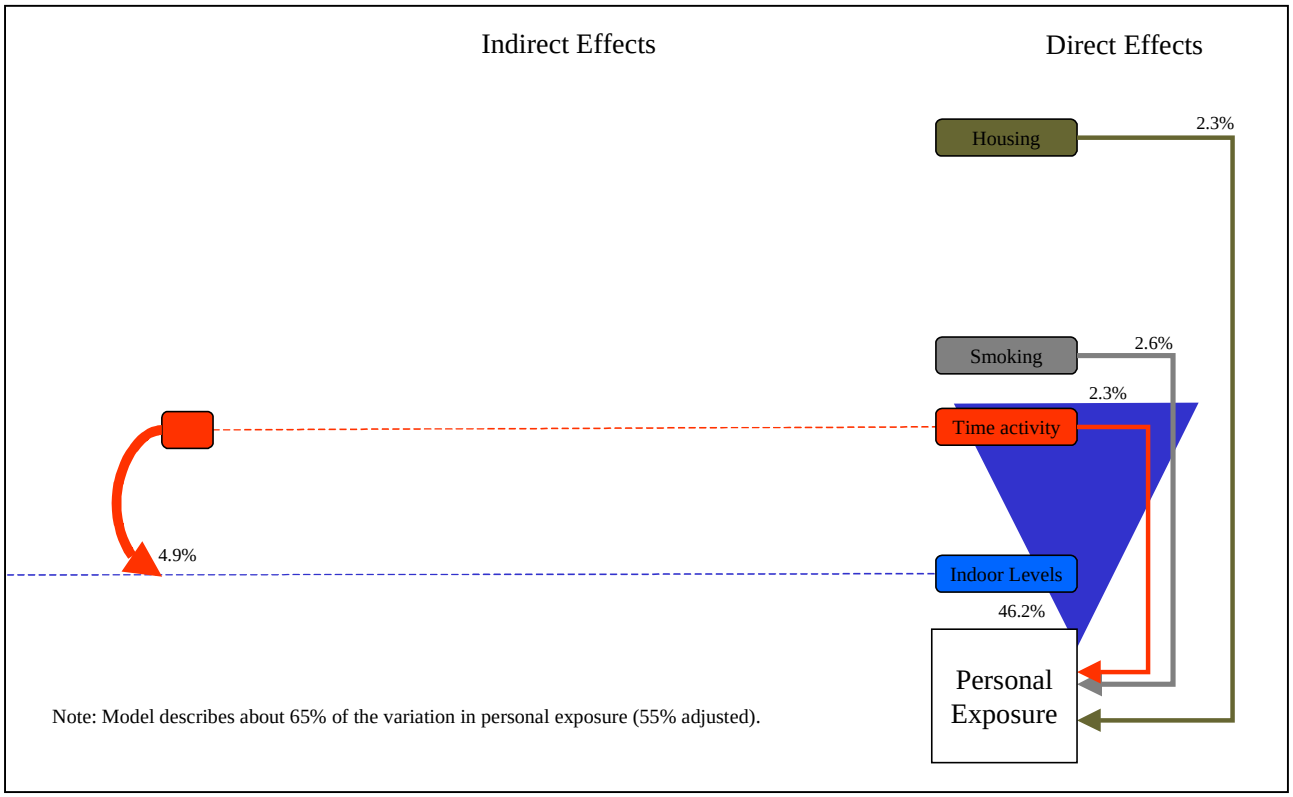



**Table 30: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .05 | 0.05 | .03 |
| Rural-Urban | .03 | 0.02 | .06 |
| Housing Characteristics | .26 | 0.27 | .15 |
| Garage | .19 | 0.16 | .06 |
| Job Status | .12 | 0.13 | .01 |
| Smoking Characteristics | .22 | 0.18 | .16 |
| Time Activity | .31 | 0.27 | .15 |
| Outdoor Concentration | .01 | 0.03 | .00 |
| Indoor Concentration | .75 | 0.67 | .68 |

**Table 31: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.03 | .01 | .01 | -.02 | -.02 | -.01 | -.02 | -.05 | -.05 |
| URBAN | -.08 | -.16 | -.16 | -.15 | -.13 | -.14 | -.04 | -.02 | |
| TRAILOR | -.13 | -.11 | -.11 | -.16 | -.20 | -.20 | -.22 | | |
| MULT2 | .01 | -.04 | -.03 | -.07 | -.09 | -.09 | -.13 | | |
| NEW | -.08 | .08 | .09 | -.09 | -.08 | -.11 | -.11 | | |
| MED | -.03 | .09 | .09 | -.05 | -.08 | -.12 | -.12 | | |
| OLD | -.08 | -.04 | -.03 | -.17 | -.15 | -.17 | -.18 | | |
| NFCDAIR | -.11 | -.01 | -.01 | .00 | .03 | .02 | .04 | | |
| CARET | .00 | .08 | .08 | .08 | .08 | .09 | .09 | | |
| ATTG | -.07 | .00 | -.01 | -.02 | -.01 | -.03 | | | |
| DETG | -.05 | -.11 | -.12 | -.09 | -.06 | -.06 | | | |
| UNPAVED | -.08 | -.16 | -.16 | -.19 | -.19 | -.19 | | | |
| JOBFT | -.01 | -.09 | -.09 | -.13 | -.17 | | | | |
| JOBPT | -.02 | -.11 | -.11 | -.10 | -.11 | | | | |
| SMKHOME | .07 | -.04 | -.04 | -.04 | | | | | |
| SMKCAR | -.01 | -.09 | -.09 | -.06 | | | | | |
| SMKAMT | .13 | .17 | .17 | .12 | | | | | |
| SMKEXP2 | -.14 | -.13 | -.13 | -.14 | | | | | |
| IH | -.03 | -.03 | -.03 | | | | | | |
| OH | -.03 | -.15 | -.15 | | | | | | |
| IW | -.05 | -.12 | -.12 | | | | | | |
| OW | .06 | .04 | .04 | | | | | | |
| IA | .11 | .08 | .09 | | | | | | |
| OA | -.03 | -.25 | -.25 | | | | | | |
| T | .04 | .06 | .06 | | | | | | |
| OCON3 | .00 | .03 | | | | | | | |
| ICON3 | .77 | | | | | | | | |
| R | .82 | .47 | .47 | .39 | .34 | .32 | .27 | .06 | .05 |

### *Hexane*

**Figure 72: Results of Model of Personal Exposure to Hexane**

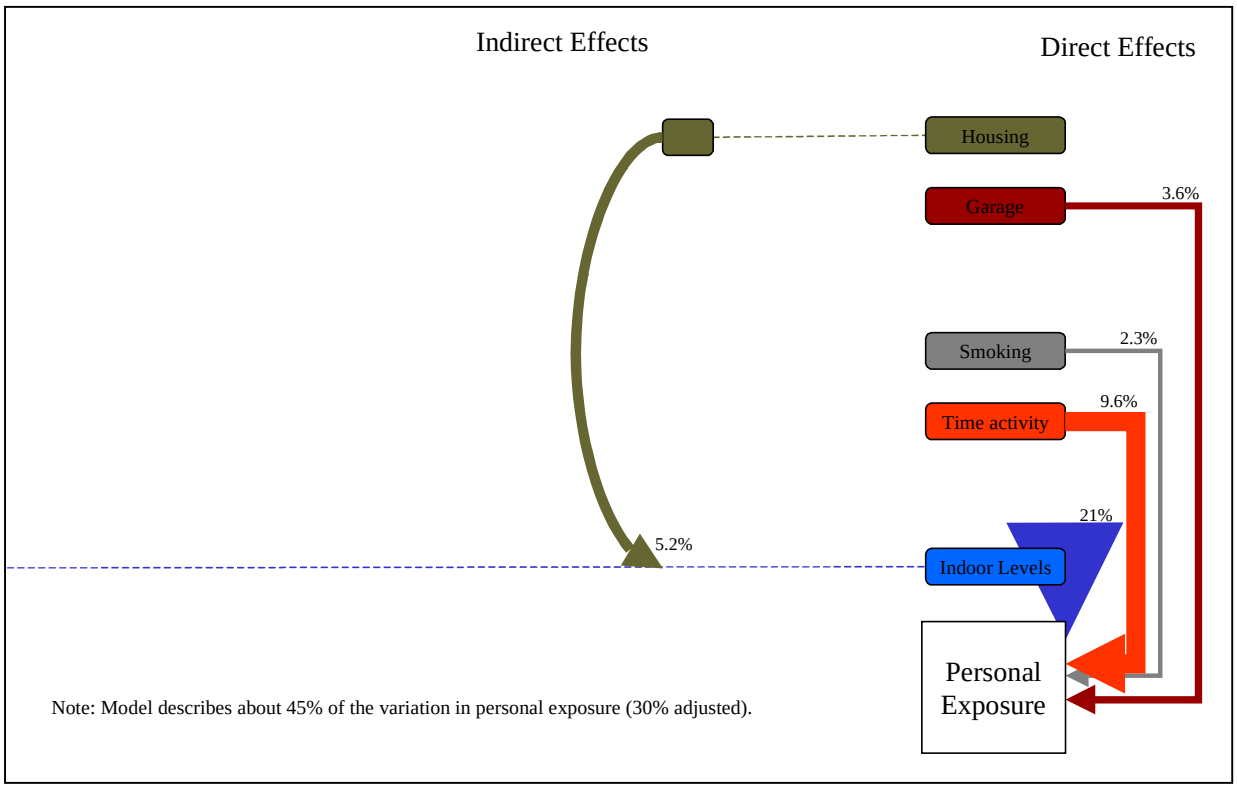



**Table 32: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .08 | 0.08 | .05 |
| Rural-Urban | .12 | 0.11 | .01 |
| Housing Characteristics | .30 | 0.28 | .12 |
| Garage | .21 | 0.10 | .19 |
| Job Status | .08 | 0.07 | .06 |
| Smoking Characteristics | .14 | 0.13 | .15 |
| Time Activity | .27 | 0.29 | .31 |
| Outdoor Concentration | .18 | 0.19 | .09 |
| Indoor Concentration | .51 | 0.46 | .46 |

**Table 33: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.07 | -.12 | -.11 | -.02 | -.01 | -.02 | -.02 | -.07 | -.08 |
| URBAN | .01 | -.03 | -.03 | -.05 | -.07 | -.06 | -.10 | -.11 | |
| TRAILOR | -.05 | -.10 | -.15 | -.16 | -.14 | -.14 | -.16 | | |
| MULT2 | -.14 | -.19 | -.19 | -.17 | -.17 | -.17 | -.21 | | |
| NEW | -.06 | .03 | .03 | -.08 | -.09 | -.08 | -.05 | | |
| MED | .02 | .11 | .10 | -.03 | -.02 | .00 | -.01 | | |
| OLD | -.01 | .06 | .06 | -.04 | -.07 | -.05 | -.04 | | |
| NFCDAIR | .05 | .16 | .15 | .17 | .14 | .15 | .15 | | |
| CARET | .02 | .13 | .10 | .13 | .13 | .12 | .11 | | |
| ATTG | -.26 | -.02 | -.02 | -.04 | -.05 | -.04 | | | |
| DETG | -.19 | -.12 | -.11 | -.11 | -.14 | -.13 | | | |
| UNPAVED | .01 | .04 | .01 | .03 | .04 | .04 | | | |
| JOBFT | -.09 | .00 | .04 | .06 | .09 | | | | |
| JOBPT | -.09 | .04 | .03 | .04 | .05 | | | | |
| SMKHOME | -.04 | .06 | .07 | .08 | | | | | |
| SMKCAR | .00 | .01 | .01 | .04 | | | | | |
| SMKAMT | -.07 | -.05 | -.04 | -.08 | | | | | |
| SMKEXP2 | .18 | .13 | .12 | .07 | | | | | |
| IH | -.18 | -.24 | -.27 | | | | | | |
| OH | .14 | .04 | .05 | | | | | | |
| IW | -.09 | -.19 | -.21 | | | | | | |
| OW | .20 | .13 | .15 | | | | | | |
| IA | .16 | .09 | .04 | | | | | | |
| OA | -.10 | -.14 | -.12 | | | | | | |
| T | -.04 | -.04 | -.04 | | | | | | |
| OCON3 | .10 | .22 | | | | | | | |
| ICON3 | .58 | | | | | | | | |
| R | .68 | .50 | .47 | .36 | .34 | .33 | .32 | .14 | .08 |

### *2-butanone*

**Figure 73: Results of Model of Personal Exposure to 2-Butanone**

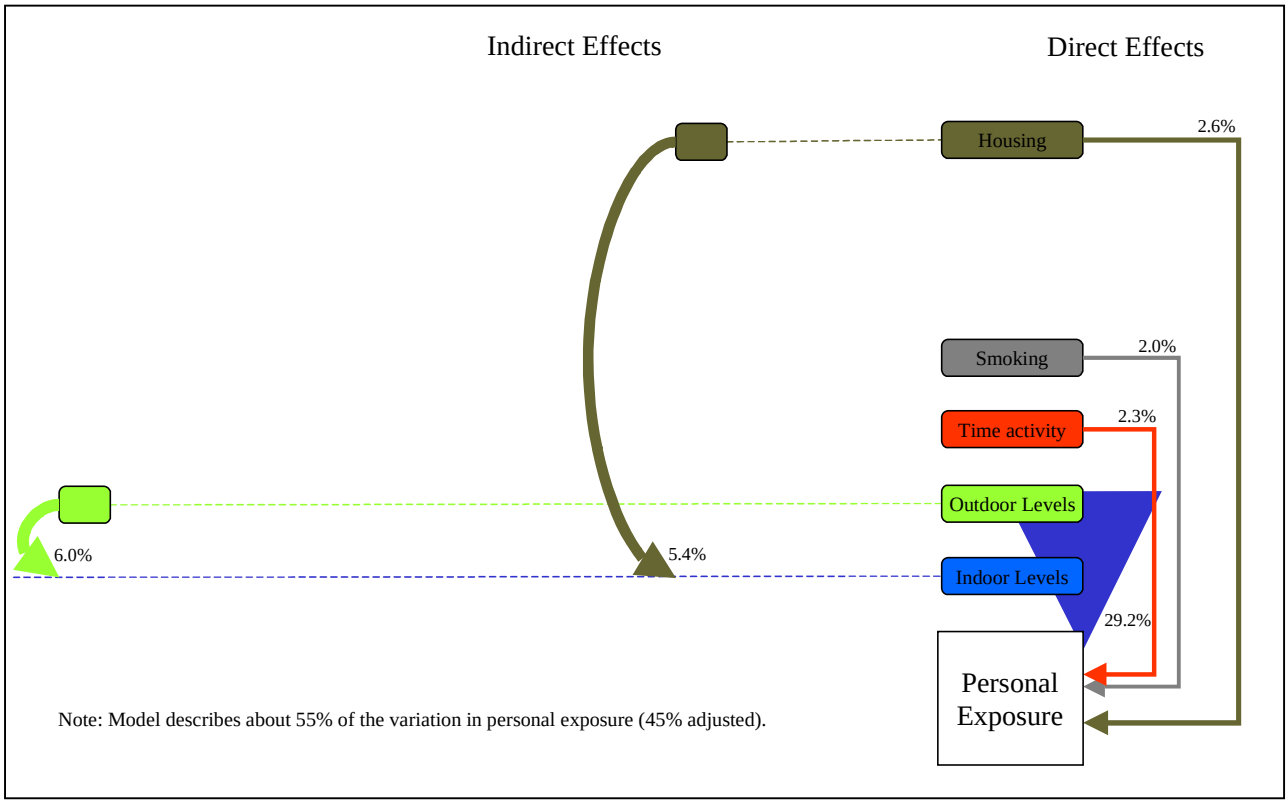



**Table 34: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .14 | .14 | .10 |
| Rural-Urban | .02 | .00 | .05 |
| Housing Characteristics | .32 | .30 | .16 |
| Garage | .12 | .11 | .08 |
| Job Status | .06 | .05 | .03 |
| Smoking Characteristics | .22 | .20 | .14 |
| Time Activity | .18 | .21 | .15 |
| Outdoor Concentration | .26 | .25 | .05 |
| Indoor Concentration | .68 | .54 | .54 |

**Table 35: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .12 | .12 | .11 | .09 | .09 | .10 | .09 | .14 | .14 |
| URBAN | .07 | .00 | .02 | -.02 | -.03 | -.03 | .04 | .00 | |
| TRAILOR | .06 | .06 | .10 | .10 | .04 | .04 | .05 | | |
| MULT2 | -.13 | -.14 | -.14 | -.14 | -.16 | -.16 | -.14 | | |
| NEW | .07 | .26 | .20 | .12 | .14 | .13 | .11 | | |
| MED | -.06 | -.04 | -.08 | -.13 | -.14 | -.15 | -.15 | | |
| OLD | .05 | .17 | .14 | .05 | .05 | .05 | .03 | | |
| NFCDAIR | .04 | -.03 | -.03 | -.07 | -.10 | -.10 | -.09 | | |
| CARET | .02 | .03 | .05 | .02 | -.01 | -.01 | .00 | | |
| ATTG | -.02 | .05 | .05 | .07 | .05 | .04 | | | |
| DETG | -.04 | .09 | .09 | .09 | .10 | .10 | | | |
| UNPAVED | -.06 | -.10 | -.11 | -.12 | -.11 | -.11 | | | |
| JOBFT | -.06 | -.10 | -.09 | -.05 | -.05 | | | | |
| JOBPT | -.04 | -.06 | -.06 | -.05 | -.06 | | | | |
| SMKHOME | .01 | .10 | .11 | .12 | | | | | |
| SMKCAR | .07 | .07 | .09 | .04 | | | | | |
| SMKAMT | .10 | .05 | .05 | .08 | | | | | |
| SMKEXP2 | -.13 | -.21 | -.22 | -.21 | | | | | |
| IH | .10 | -.06 | -.05 | | | | | | |
| OH | -.07 | -.04 | -.02 | | | | | | |
| IW | .11 | .05 | .05 | | | | | | |
| OW | -.06 | -.21 | -.20 | | | | | | |
| IA | -.05 | -.02 | -.03 | | | | | | |
| OA | .08 | .01 | .05 | | | | | | |
| T | .10 | .05 | .11 | | | | | | |
| OCON3 | .05 | .26 | | | | | | | |
| ICON3 | .64 | | | | | | | | |
| R | .75 | .52 | .46 | .41 | .35 | .35 | .33 | .14 | .14 |

### *3-Methylhexane*

**Figure 74: Results of Model of Personal Exposure to Methylhexane**

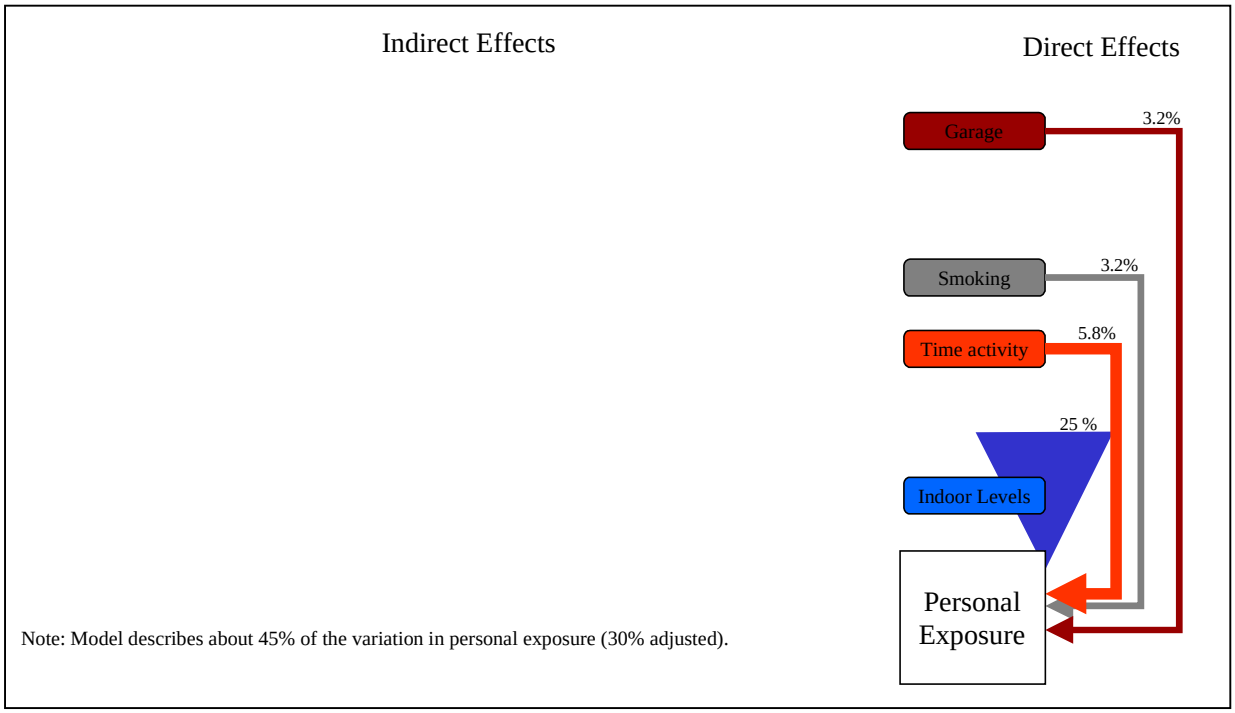



**Table 36: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .00 | 0.00 | .04 |
| Rural-Urban | .08 | 0.08 | .02 |
| Housing Characteristics | .19 | 0.21 | .09 |
| Garage | .22 | 0.19 | .18 |
| Job Status | .11 | 0.10 | .09 |
| Smoking Characteristics | .19 | 0.18 | .18 |
| Time Activity | .25 | 0.23 | .24 |
| Outdoor Concentration | .15 | 0.13 | .02 |
| Indoor Concentration | .53 | 0.50 | .50 |

**Table 37: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | -.06 | .01 | -.01 | .01 | .02 | .03 | .02 | .01 | .00 |
| URBAN | .03 | -.01 | .02 | .01 | -.02 | .00 | -.06 | -.08 | |
| TRAILOR | .00 | .01 | .02 | .00 | .01 | .00 | -.04 | | |
| MULT2 | -.03 | .03 | .07 | .05 | .05 | .05 | -.05 | | |
| NEW | .04 | .19 | .21 | .09 | .06 | .08 | .16 | | |
| MED | .10 | .21 | .27 | .15 | .17 | .20 | .19 | | |
| OLD | .09 | .22 | .30 | .20 | .18 | .20 | .23 | | |
| NFCDAIR | -.02 | .03 | .06 | .07 | .04 | .07 | .07 | | |
| CARET | .04 | .14 | .17 | .19 | .16 | .16 | .14 | | |
| ATTG | -.23 | -.01 | -.01 | -.02 | -.02 | .00 | | | |
| DETG | -.21 | -.21 | -.21 | -.21 | -.23 | -.24 | | | |
| UNPAVED | .03 | .05 | .04 | .06 | .08 | .09 | | | |
| JOBFT | .08 | .20 | .16 | .13 | .12 | | | | |
| JOBPT | -.04 | .10 | .06 | .05 | .02 | | | | |
| SMKHOME | -.02 | .10 | .12 | .14 | | | | | |
| SMKCAR | -.04 | -.05 | -.06 | -.06 | | | | | |
| SMKAMT | .07 | .07 | .08 | .05 | | | | | |
| SMKEXP2 | .19 | .16 | .14 | .11 | | | | | |
| IH | -.22 | -.26 | -.25 | | | | | | |
| OH | .11 | -.03 | -.01 | | | | | | |
| IW | -.12 | -.24 | -.20 | | | | | | |
| OW | .08 | -.01 | .01 | | | | | | |
| IA | .02 | .03 | .04 | | | | | | |
| OA | -.11 | -.20 | -.21 | | | | | | |
| T | .01 | -.10 | -.08 | | | | | | |
| OCON3 | .02 | .16 | | | | | | | |
| ICON3 | .60 | | | | | | | | |
| R | .67 | .45 | .43 | .36 | .31 | .29 | .23 | .08 | .00 |

### *Heptane*

**Figure 75: Results of Model of Personal Exposure to Heptane**

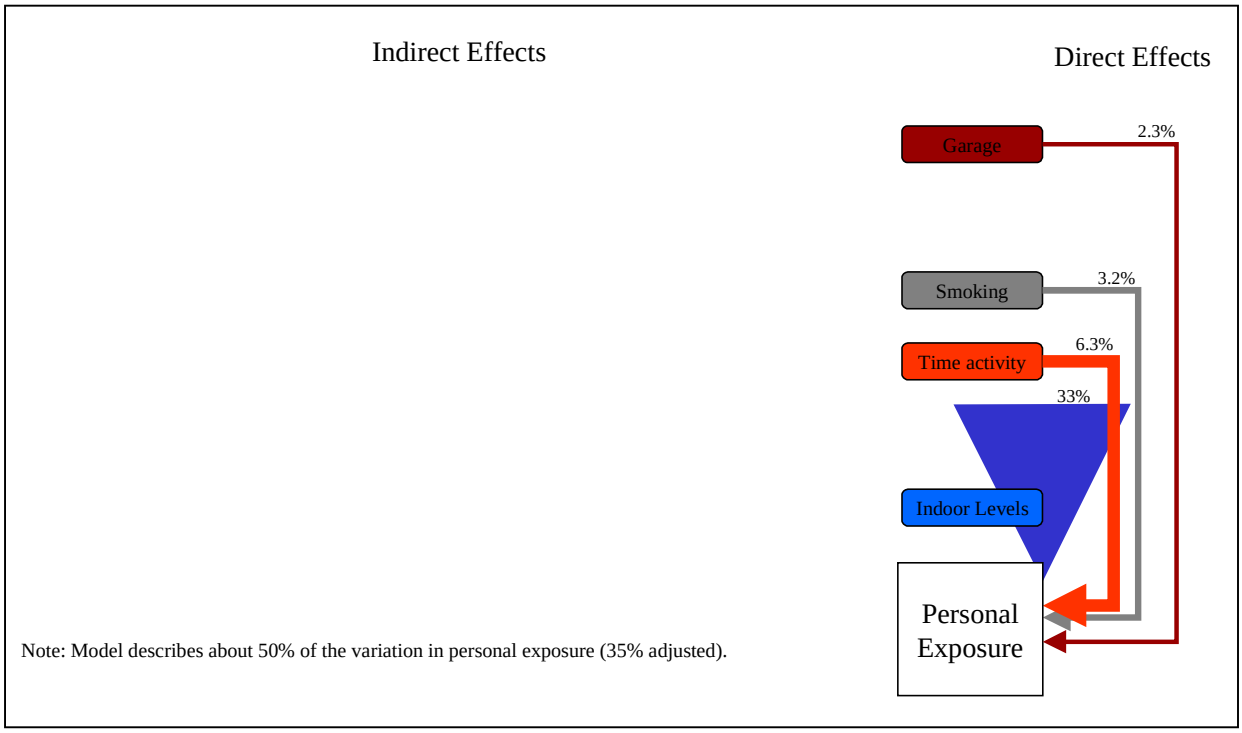



**Table 38: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .01 | .01 | .02 |
| Rural-Urban | .06 | .06 | .05 |
| Housing Characteristics | .22 | .25 | .11 |
| Garage | .15 | .1 | .15 |
| Job Status | .08 | .05 | .04 |
| Smoking Characteristics | .17 | .2 | .18 |
| Time Activity | .23 | .24 | .25 |
| Outdoor Concentration | .18 | .17 | .05 |
| Indoor Concentration | .61 | .56 | .57 |

**Table 39: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .01 | .02 | -.01 | .03 | .04 | .03 | .04 | .00 | .01 |
| URBAN | .07 | .14 | .14 | .12 | .10 | .10 | .09 | .06 | |
| TRAILOR | -.06 | -.05 | -.05 | -.06 | -.05 | -.05 | -.06 | | |
| MULT2 | -.08 | -.15 | -.12 | -.11 | -.10 | -.09 | -.13 | | |
| NEW | .04 | .18 | .24 | .11 | .14 | .15 | .16 | | |
| MED | .11 | .21 | .28 | .16 | .22 | .23 | .22 | | |
| OLD | .04 | .12 | .25 | .15 | .17 | .18 | .18 | | |
| NFCDAIR | .04 | .15 | .16 | .20 | .17 | .17 | .17 | | |
| CARET | .01 | .14 | .14 | .17 | .13 | .13 | .12 | | |
| ATTG | -.20 | -.07 | -.08 | -.08 | -.09 | -.08 | | | |
| DETG | -.16 | -.09 | -.10 | -.10 | -.13 | -.13 | | | |
| UNPAVED | -.01 | .01 | -.03 | -.03 | -.02 | -.02 | | | |
| JOBFT | .06 | .09 | .07 | .08 | .06 | | | | |
| JOBPT | .02 | .08 | .08 | .09 | .05 | | | | |
| SMKHOME | -.03 | .01 | .00 | .02 | | | | | |
| SMKCAR | -.05 | .06 | .05 | .06 | | | | | |
| SMKAMT | .07 | .10 | .10 | .10 | | | | | |
| SMKEXP2 | .20 | .15 | .15 | .11 | | | | | |
| IH | -.15 | -.20 | -.19 | | | | | | |
| OH | .10 | .01 | .03 | | | | | | |
| IW | -.05 | -.18 | -.14 | | | | | | |
| OW | .16 | .06 | .09 | | | | | | |
| IA | .16 | .15 | .14 | | | | | | |
| OA | -.05 | -.13 | -.15 | | | | | | |
| T | -.08 | -.12 | -.08 | | | | | | |
| OCON3 | .07 | .20 | | | | | | | |
| ICON3 | .63 | | | | | | | | |
| R | .72 | .45 | .42 | .34 | .28 | .28 | .26 | .06 | .01 |

### *Octane*

**Figure 76: Results of Model of Personal Exposure to Octane**

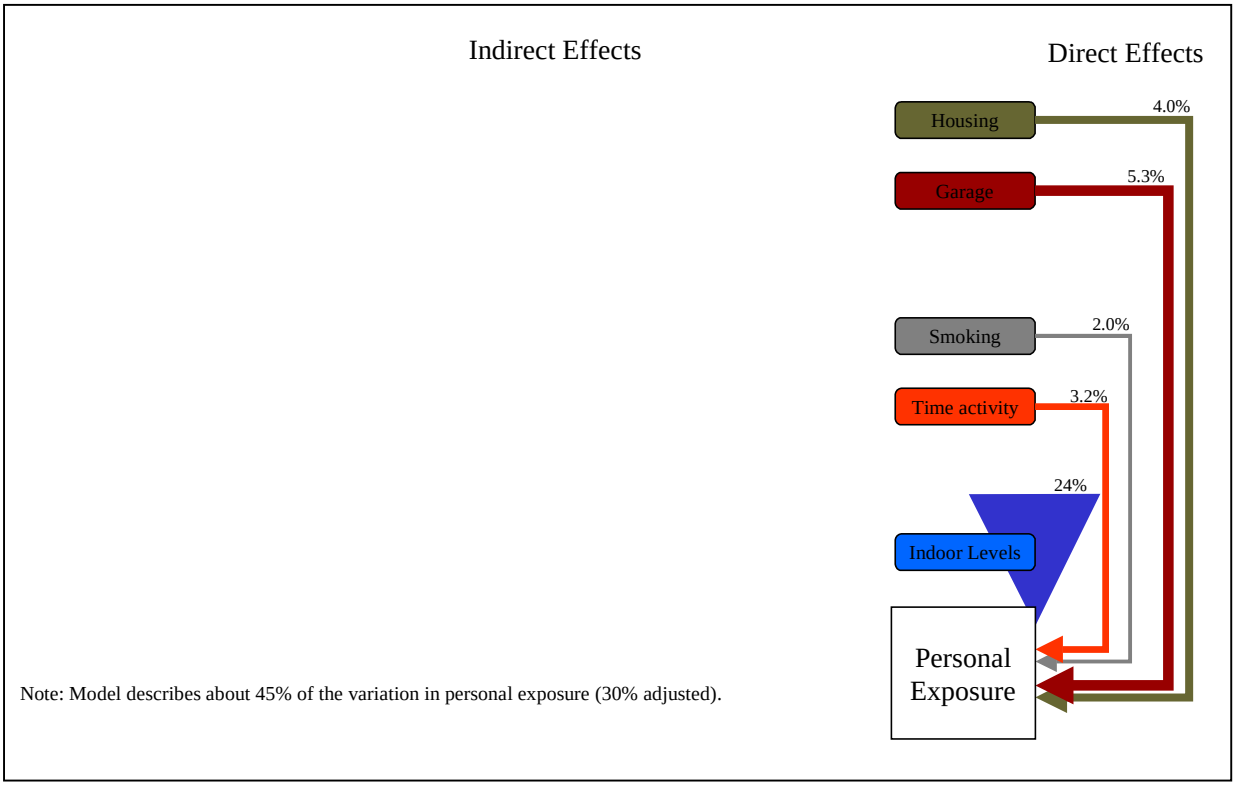



**Table 40: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .07 | 0.07 | .08 |
| Rural-Urban | .02 | 0.02 | .02 |
| Housing Characteristics | .25 | 0.27 | .20 |
| Garage | .24 | 0.16 | .23 |
| Job Status | .10 | 0.12 | .08 |
| Smoking Characteristics | .21 | 0.21 | .14 |
| Time Activity | .21 | 0.20 | .18 |
| Outdoor Concentration | .09 | 0.08 | .08 |
| Indoor Concentration | .53 | 0.49 | .49 |

**Table 41: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .10 | .11 | .11 | .10 | .11 | .09 | .10 | .06 | .07 |
| URBAN | .03 | .02 | .01 | .05 | .02 | .02 | .03 | .02 | |
| TRAILOR | -.10 | -.11 | -.11 | -.11 | -.12 | -.12 | -.14 | | |
| MULT2 | -.19 | -.17 | -.17 | -.16 | -.18 | -.18 | -.24 | | |
| NEW | .06 | -.12 | -.12 | -.10 | -.13 | -.10 | -.10 | | |
| MED | .02 | -.10 | -.10 | -.11 | -.11 | -.08 | -.10 | | |
| OLD | .03 | -.14 | -.14 | -.11 | -.15 | -.12 | -.12 | | |
| NFCDAIR | -.08 | .00 | .01 | .04 | .00 | .00 | .02 | | |
| CARET | .00 | .05 | .06 | .10 | .06 | .05 | .04 | | |
| ATTG | -.33 | -.16 | -.14 | -.15 | -.16 | -.13 | | | |
| DETG | -.18 | -.25 | -.24 | -.20 | -.21 | -.21 | | | |
| UNPAVED | -.10 | -.10 | -.10 | -.11 | -.08 | -.08 | | | |
| JOBFT | .13 | .13 | .09 | .15 | .15 | | | | |
| JOBPT | .13 | .14 | .13 | .14 | .12 | | | | |
| SMKHOME | .05 | .23 | .24 | .25 | | | | | |
| SMKCAR | -.12 | -.10 | -.10 | -.09 | | | | | |
| SMKAMT | .10 | .07 | .08 | .06 | | | | | |
| SMKEXP2 | .12 | .03 | .02 | .02 | | | | | |
| IH | -.06 | -.15 | -.16 | | | | | | |
| OH | -.02 | -.10 | -.10 | | | | | | |
| IW | -.04 | -.08 | -.05 | | | | | | |
| OW | .18 | .12 | .14 | | | | | | |
| IA | .10 | .00 | .00 | | | | | | |
| OA | -.04 | -.12 | -.12 | | | | | | |
| T | -.08 | -.14 | -.14 | | | | | | |
| OCON3 | -.09 | -.09 | | | | | | | |
| ICON3 | .58 | | | | | | | | |
| R | .67 | .45 | .45 | .40 | .34 | .32 | .27 | .07 | .07 |

### *Nonane*

**Figure 77: Results of Model of Personal Exposure to Nonane**

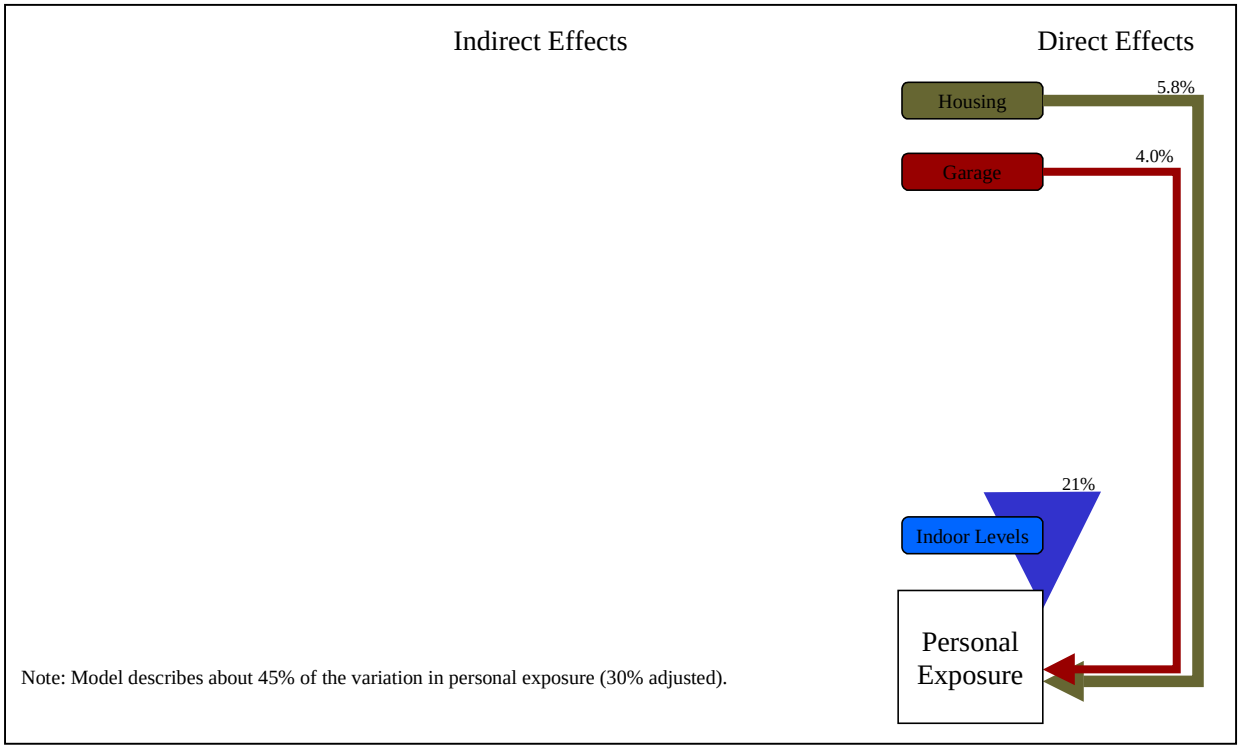



**Table 42: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .09 | 0.09 | .13 |
| Rural-Urban | .07 | 0.08 | .00 |
| Housing Characteristics | .34 | 0.34 | .24 |
| Garage | .23 | 0.14 | .20 |
| Job Status | .01 | 0.01 | .05 |
| Smoking Characteristics | .26 | 0.21 | .11 |
| Time Activity | .23 | 0.24 | .12 |
| Outdoor Concentration | .00 | 0.02 | .03 |
| Indoor Concentration | .52 | 0.45 | .46 |

**Table 43: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .17 | .10 | .10 | .10 | .10 | .11 | .11 | .10 | .09 |
| URBAN | .01 | -.07 | -.07 | -.06 | -.08 | -.08 | -.05 | -.09 | |
| TRAILOR | -.04 | -.03 | -.03 | -.04 | -.08 | -.08 | -.09 | | |
| MULT2 | -.22 | -.19 | -.19 | -.18 | -.22 | -.22 | -.27 | | |
| NEW | .12 | .13 | .13 | .11 | .07 | .07 | .07 | | |
| MED | .09 | .13 | .13 | .10 | .06 | .06 | .04 | | |
| OLD | -.01 | .02 | .01 | -.01 | -.06 | -.06 | -.06 | | |
| NFCDAIR | -.16 | -.12 | -.12 | -.15 | -.18 | -.18 | -.17 | | |
| CARET | -.10 | -.08 | -.08 | -.05 | -.08 | -.08 | -.09 | | |
| ATTG | -.26 | -.10 | -.10 | -.11 | -.12 | -.12 | | | |
| DETG | -.15 | -.21 | -.21 | -.17 | -.16 | -.16 | | | |
| UNPAVED | -.15 | -.14 | -.14 | -.13 | -.10 | -.10 | | | |
| JOBFT | .02 | -.12 | -.12 | -.01 | .01 | | | | |
| JOBPT | .06 | -.04 | -.04 | -.01 | -.01 | | | | |
| SMKHOME | .09 | .26 | .25 | .28 | | | | | |
| SMKCAR | -.11 | -.08 | -.07 | -.11 | | | | | |
| SMKAMT | .11 | .06 | .06 | .03 | | | | | |
| SMKEXP2 | .00 | -.13 | -.13 | -.10 | | | | | |
| IH | -.12 | -.34 | -.34 | | | | | | |
| OH | .02 | -.13 | -.13 | | | | | | |
| IW | -.01 | -.05 | -.05 | | | | | | |
| OW | .07 | .00 | .00 | | | | | | |
| IA | .02 | -.17 | -.17 | | | | | | |
| OA | -.02 | -.14 | -.13 | | | | | | |
| T | -.08 | -.07 | -.07 | | | | | | |
| OCON3 | -.03 | -.02 | | | | | | | |
| ICON3 | .56 | | | | | | | | |
| R | .68 | .50 | .50 | .44 | .39 | .39 | .36 | .12 | .09 |

### *Decane*

**Figure 78: Results of Model of Personal Exposure to Decane**

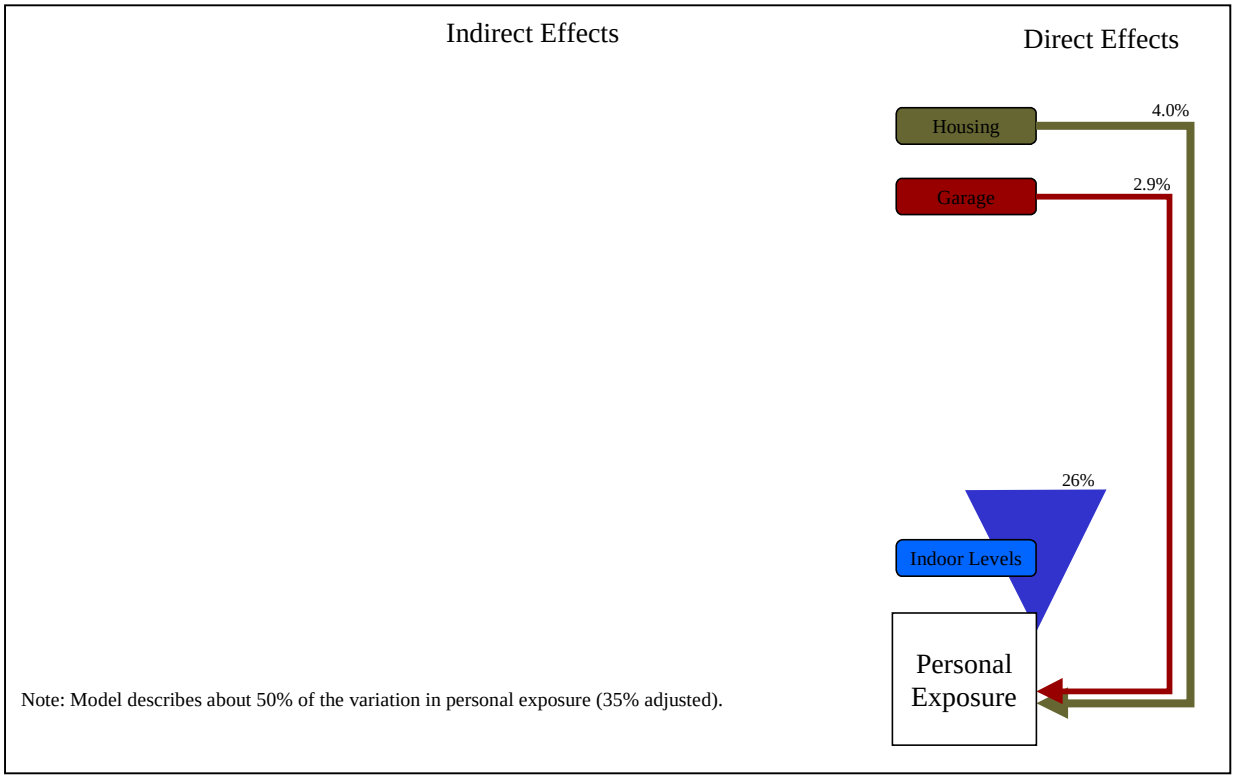



**Table 44: Comparative Multiple Regression Coefficients for Variable Sets**

| Source | Total Effects | Model-Derived Total Effects | Direct Effects: Semi-Partial R |
|---|---|---|---|
| Gender | .12 | .12 | .13 |
| Rural-Urban | .04 | .06 | .06 |
| Housing Characteristics | .30 | .30 | .20 |
| Garage | .19 | .11 | .17 |
| Job Status | .08 | .06 | .02 |
| Smoking Characteristics | .26 | .23 | .11 |
| Time Activity | .23 | .23 | .09 |
| Outdoor Concentration | .02 | .04 | .01 |
| Indoor Concentration | .60 | .51 | .51 |

**Table 45: Beta Weights for Hierarchical Set Regression of Personal Exposure Concentrations**

| Source | Step 9 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|---|---|
| GENDER | .17 | .09 | .10 | .12 | .12 | .14 | .15 | .13 | .12 |
| URBAN | .09 | -.04 | -.05 | -.05 | -.07 | -.05 | -.02 | -.06 | |
| TRAILOR | -.01 | -.04 | -.04 | -.06 | -.10 | -.11 | -.11 | | |
| MULT2 | -.21 | -.20 | -.21 | -.20 | -.24 | -.24 | -.26 | | |
| NEW | .13 | .23 | .21 | .17 | .13 | .12 | .10 | | |
| MED | .12 | .20 | .19 | .15 | .10 | .08 | .07 | | |
| OLD | .04 | .13 | .11 | .06 | .01 | .00 | -.01 | | |
| NFCDAIR | -.05 | .00 | .00 | -.05 | -.08 | -.07 | -.06 | | |
| CARET | -.07 | -.04 | -.05 | -.04 | -.05 | -.05 | -.05 | | |
| ATTG | -.21 | -.08 | -.08 | -.09 | -.10 | -.11 | | | |
| DETG | -.12 | -.11 | -.12 | -.10 | -.08 | -.09 | | | |
| UNPAVED | -.10 | -.14 | -.14 | -.13 | -.10 | -.10 | | | |
| JOBFT | -.04 | -.18 | -.18 | -.07 | -.05 | | | | |
| JOBPT | -.01 | -.14 | -.15 | -.10 | -.09 | | | | |
| SMKHOME | .09 | .31 | .29 | .30 | | | | | |
| SMKCAR | -.10 | -.10 | -.08 | -.10 | | | | | |
| SMKAMT | .11 | .03 | .04 | -.02 | | | | | |
| SMKEXP2 | -.04 | -.19 | -.19 | -.15 | | | | | |
| IH | -.07 | -.24 | -.24 | | | | | | |
| OH | .00 | -.09 | -.10 | | | | | | |
| IW | .04 | .01 | .00 | | | | | | |
| OW | .06 | -.03 | -.03 | | | | | | |
| IA | -.01 | -.22 | -.22 | | | | | | |
| OA | .00 | -.13 | -.13 | | | | | | |
| T | .02 | .07 | .06 | | | | | | |
| OCON3 | -.01 | -.05 | | | | | | | |
| ICON3 | .63 | | | | | | | | |
| R | .70 | .48 | .48 | .42 | .35 | .35 | .33 | .13 | .12 |

## 8.8 Summary of Exposure Relationships for Passive Samplers

The previous sections have presented a large amount of information about a number of chemicals each analyzed separately. Within each analysis, careful examination of the tables can allow a sophisticated picture of causal influences to be postulated. However, little has yet been said about the manner in which the causal influences are similar across chemicals. In the following section, a higher order analysis is presented which can allow preliminary statements about the full domain of chemicals collected by passive samplers.

The starting point of this analysis is the semi-partial correlation coefficients for each of the sets of influences included in the modeling process for each chemical. (These were presented in the column for the last step of the set of tables entitled, "Comparative Multiple Regression Coefficients for Variable Sets" for each analysis of personal exposure). Basically, these numbers were brought together into a single table (with a separate column for each chemical and separate row for each set of influences) for the current analysis.

Next, a principal component decomposition of this table was performed, and the largest two dimensions of this analysis were used for a single biplot representation. With proper interpretation, this diagram summarizes the information present in the original table (to a substantial degree, though more dimensions would be required to allow complete reconstruction). The advantage of this analysis is that it can represent the relative importance of the causal influences across chemicals, and the relative similarity of chemicals with respect to their causal influence structure within a single graphic representation.

The interpretation of this diagram is as follows: each causal influence and each chemical has a co-ordinate in the two dimensional space. In absolute terms, the average size of the semi-partial multiple correlation coefficient across all chemicals considered together can be determined by the relative location of the points representing the causal influences on the first dimension. That is, the orderings of the coefficients on the first dimension gives the average ordering of the coefficient across all chemicals. In the current case, it can be seen that the influence of indoor concentrations is the single largest influence on the personal concentrations across this set of chemicals (because it has the highest positive value on the first dimension; it is located to the extreme right).

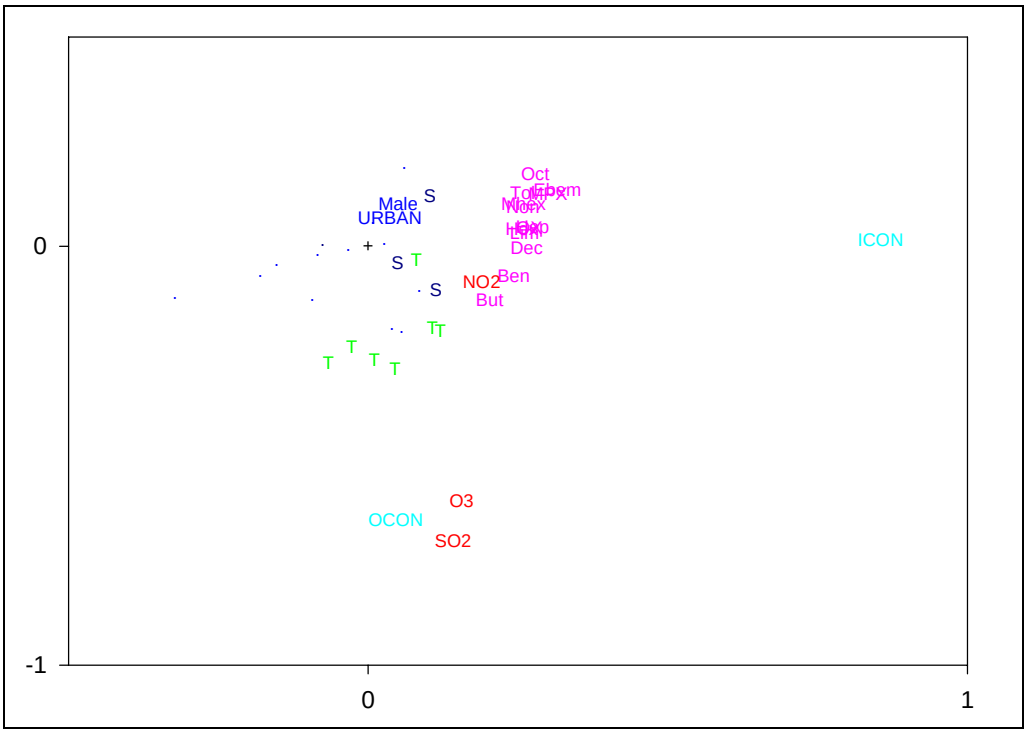
The inclusion of the second dimension on each of the diagrams allows chemicals to be separated based upon differences in the pattern and magnitude of the set of influences. To determine the nature of these differences, follow this basic procedure for each chemical point: mentally draw a line from its co-ordinate through the '+' located on the graph at the 0,0 point (the origin). Consider this line as a new dimension. Values of the causal influences are ordered on this dimension in terms of their order of magnitude in predicting the concentration of that chemical. (Mentally, the operation to determine the values of the influences on this new dimension requires that you draw a perpendicular line from the point to the new point to the axis dimension (technically, "orthogonally project"). This operation is entirely analogous to determining the value of a point on a labeled dimension, as was necessary to determine the magnitudes (described above). Notice that the actual pattern and ordering of the influences will differ for chemicals located in different quadrants of the space. Thus, for $SO_2$ outdoor concentration is as important as indoor concentration in the prediction of personal concentration levels.

The analogous procedure can be performed for each set of influences to derive an ordering of chemicals for which this set of influences is relatively more or less important.

Finally, a global mode of interpretation is possible by combining all of this information as follows: chemicals in the same radial sector have similar patterns of influence, those farther from the origin (the '+' point) are more predictable than those nearer the origin. For personal concentration levels, this pattern is shown by $SO_2$ and $O_3$. Chemicals that are close together on the plot have similar patterns of influence

and similar levels of predictability. For personal concentrations, this condition is clearly met for the VOC chemicals.

**Figure 79: Biplot Representation of Semi-Partial R for Determinant Sets of Chemicals (Personal Concentrations)**




The overall impression that is left by this global mode of interpretation is as follows:

- For all chemicals, indoor levels are an important causal feature
- Outdoor concentrations are relatively more important for $SO_2$ and $O_3$ than for $NO_2$ and the VOCs.
- Time-activity patterns and smoking behavior are relatively more important for $SO_2$ ,$O_3$, and $NO_2$ than for (most of) the VOCs.

## 8.9 Particulate Analysis: $PM_{2.5}$

Since only a very small number of $PM_{2.5}$ samples were available, an analysis such as was performed on the exposures collected from passive samplers was not viable. Instead, a stepwise regression was employed on each of the personal concentration level and the indoor concentration level, using each of the variables detailed in section 8.1 as candidate predictors. In the analysis of personal concentration level, the only variable that emerged as predictive in the stepwise analysis was indoor concentration level. In the analysis of indoor concentration level, the only variable that emerged as predictive was the number of cigarettes smoked. These findings are consistent with findings from earlier studies, and suggest that smoking is a major determinant of the amount of particulate to which an individual is exposed.

## 9.0 Biomarkers of Exposure

Blood and urine samples were obtained during the assessment period for each participant.

The analysis of the blood samples included measures of nicotine, while the analysis of the urine samples included measures of BTEX compounds. The following table summarizes information from the blood and urine data analysis.

As shown by the table, only nicotine, muconic acid, and hippuric acid were above detection limits in some samples, but analysis did not reveal any relationship between personal exposure to benzene, toluene, or nicotine and their respective biomarker levels.

**Table 46: Analysis of Metabolites**

| Biomarkers Measured in Blood | Number of Samples Analyzed | Number of Samples Above Detection Limit and Range |
|---|---|---|
| Nicotine | 127 | 6 (5.5 – 31.2 ng/mL) |
| **Biomarkers Measured in Urine** | | |
| Muconic acid (metabolite of benzene) | 126 | 69 (0.05 – 0.90 µg/mL) |
| Hippuric acid (metabolite of toluene) | 126 | 126 (13.8 – 988.1 µg/mL) |
| Mandelic acid (metabolite of ethylbenzene) | 126 | 0 |
| 2-Methylhippuric acid (metabolite of o-xylene) | 126 | 0 |
| 3-, 4- Methylhippuric acid (metabolite of m-xylene) | 126 | 0 |

# 10.0 Biomarkers of Effect

The biomarkers of effect included in the Grande Prairie Community Exposure and Health Effects Assessment Program consisted of a neurocognitive assessment and a respiratory health assessment.

## 10.1 Neurocognitive Functioning

Neuropsychological assessment was included as a non-invasive means of evaluating associations between exposure and effects in neurocognitive function. Participants completed the Neurobehavioral Evaluation System 2 (NES2), Neuropsychological Impairment Scale (NIS), the Verbal Digit Span section of the Wechsler Memory Scale – Revised (WMS-R), and the Weekly Stress Inventory (WSI). Comparisons were made between the current study and that of control groups of previous studies which have used these assessments.

### *Neurobehavioral Evaluation System (NES2)*

The NES2 is a computerized program that assesses a number of basic neurological and cognitive parameters, including finger tapping, continuous performance, hand-eye co-ordination, associate learning, simple reaction time, symbol-digit, pattern comparison, pattern memory, serial digit learning, switching attention, colour-word, and delayed associate recognition. The following graphs compare the performance of the Grande Prairie sample with other studies using unexposed populations. Overall, there were no significant differences observed between the Grande Prairie sample and the results from these other studies.

**Figure 80: Finger Tapping Test**

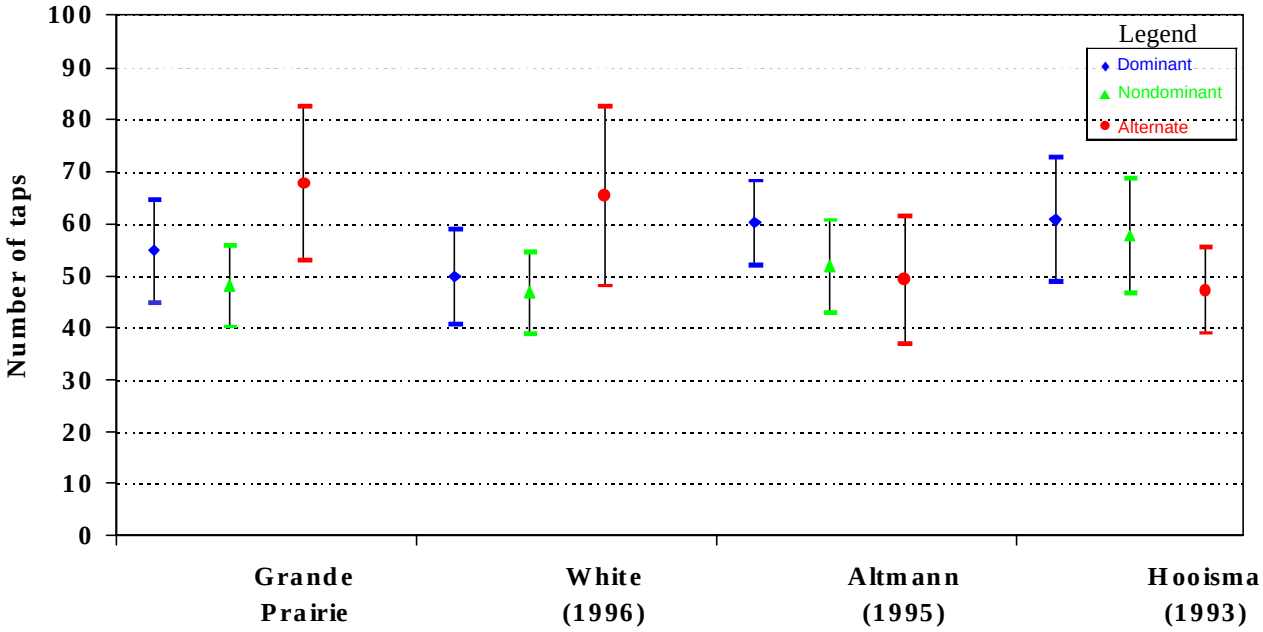


**Figure 81: Associate Learning Test**

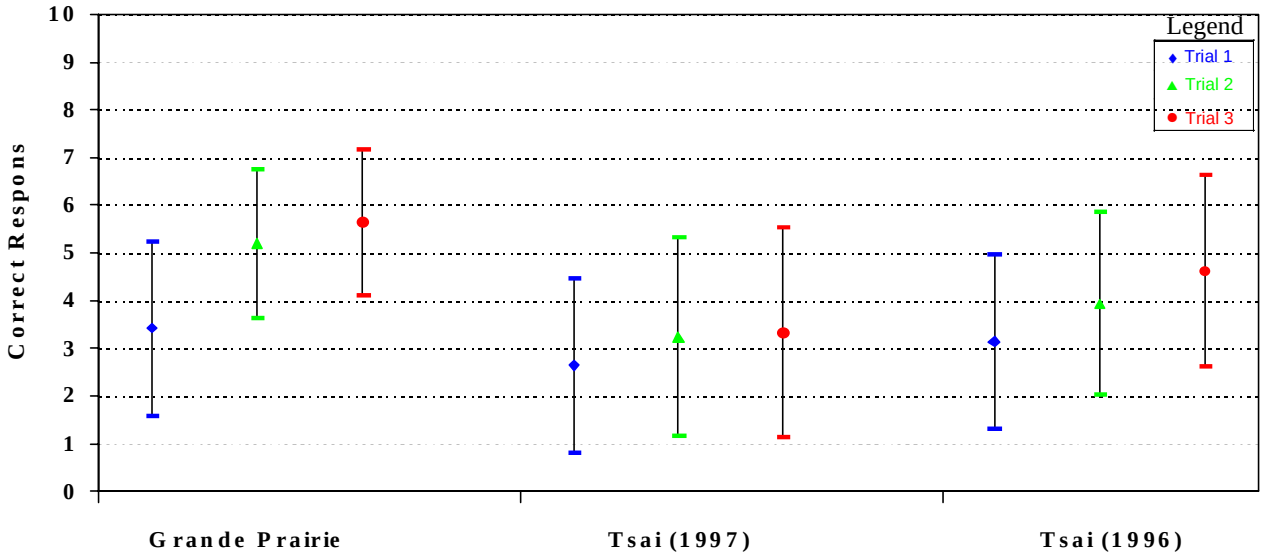



**Figure 82: Switching Attention Test**

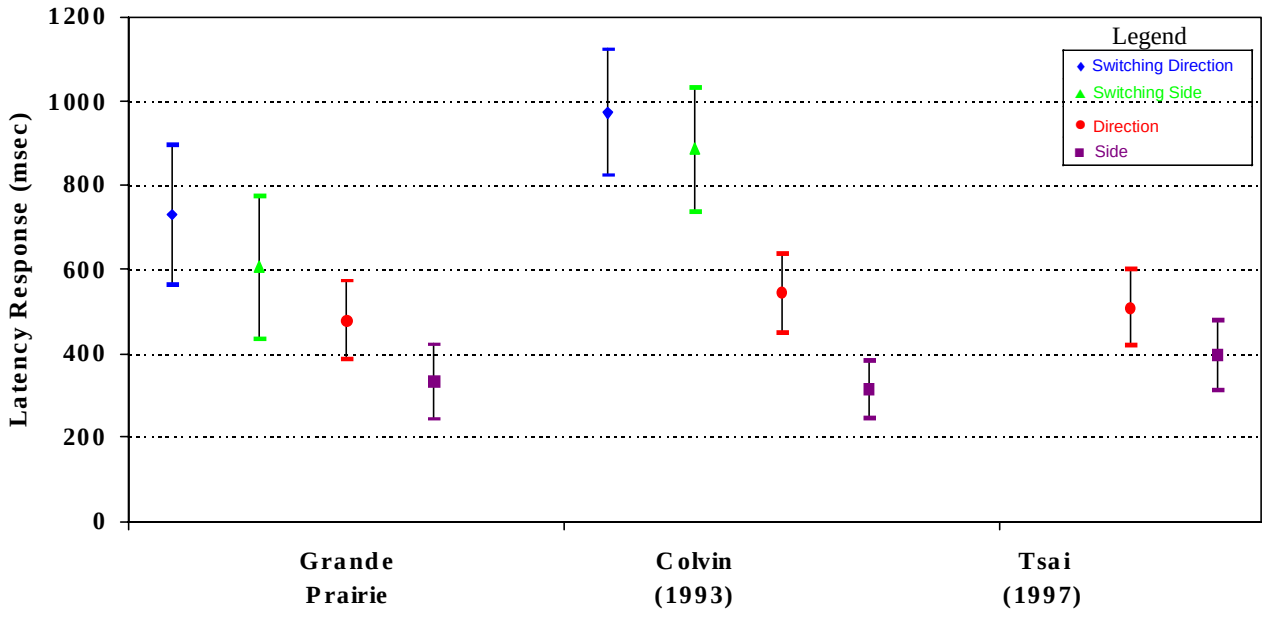


**Figure 83: Mood Scales**

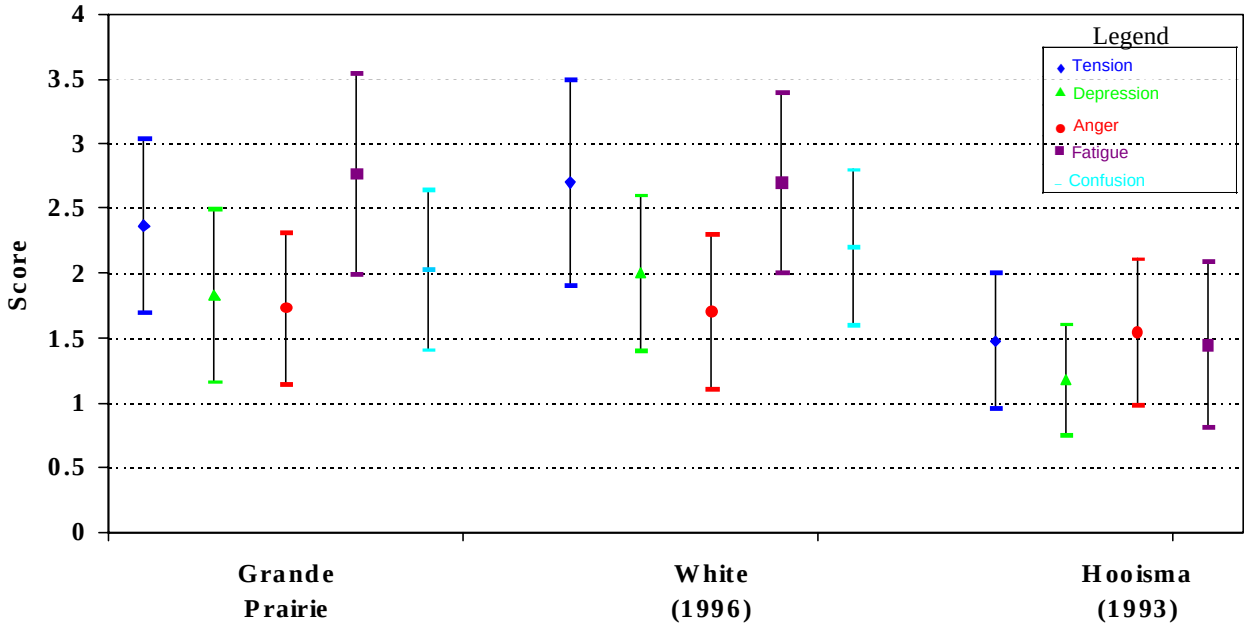



**Figure 84: Continuous Performance Test**

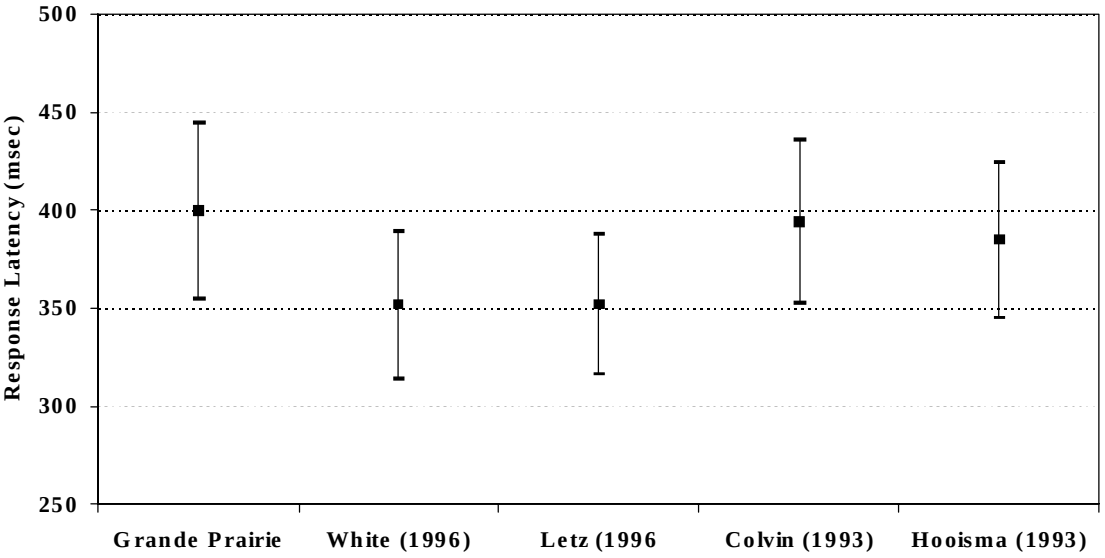


**Figure 85: Hand-Eye Co-ordination Test**

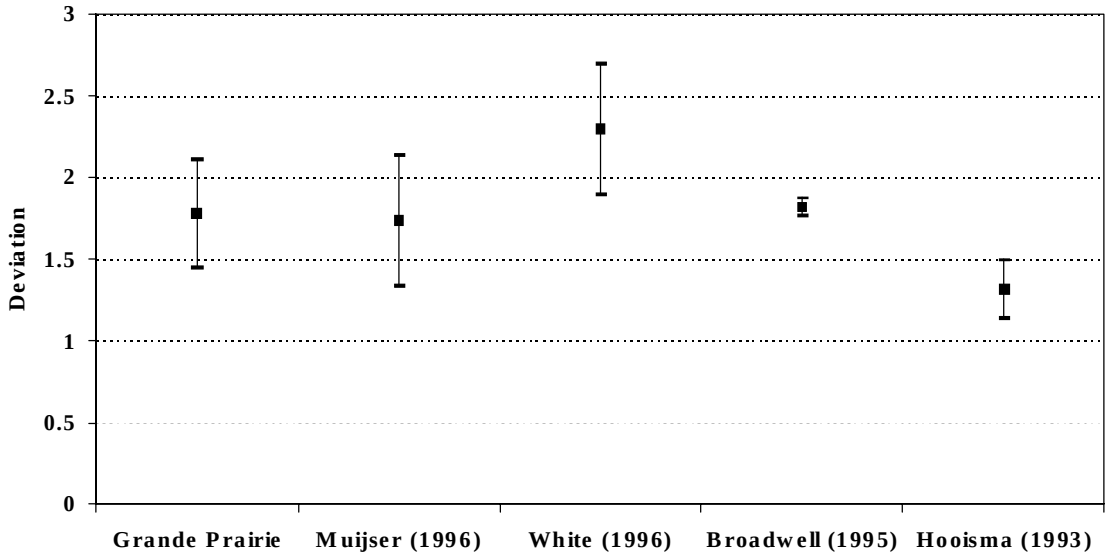



**Figure 86: Simple Reaction Time**

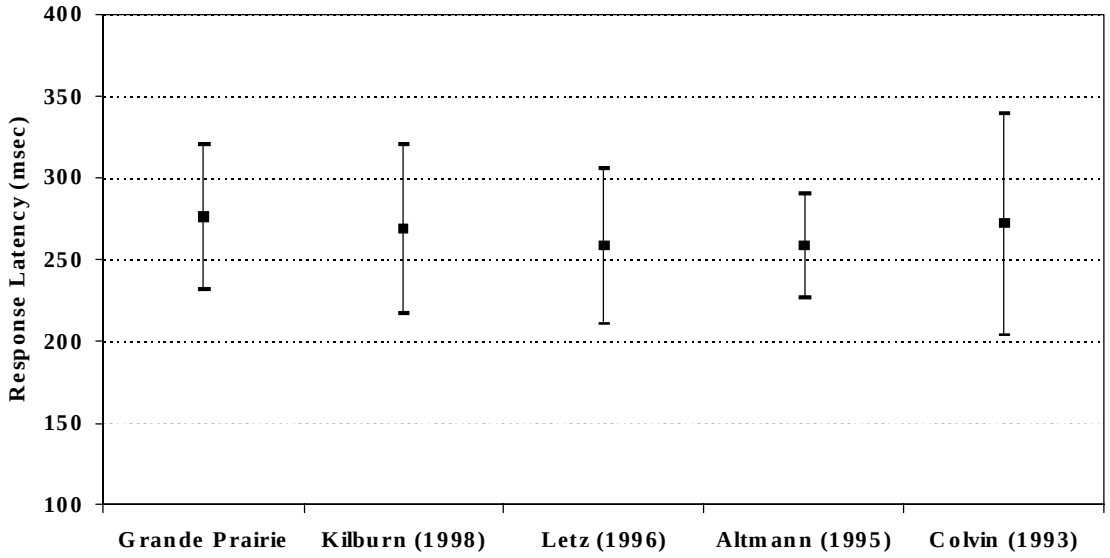


**Figure 87: Symbol Digit Test**

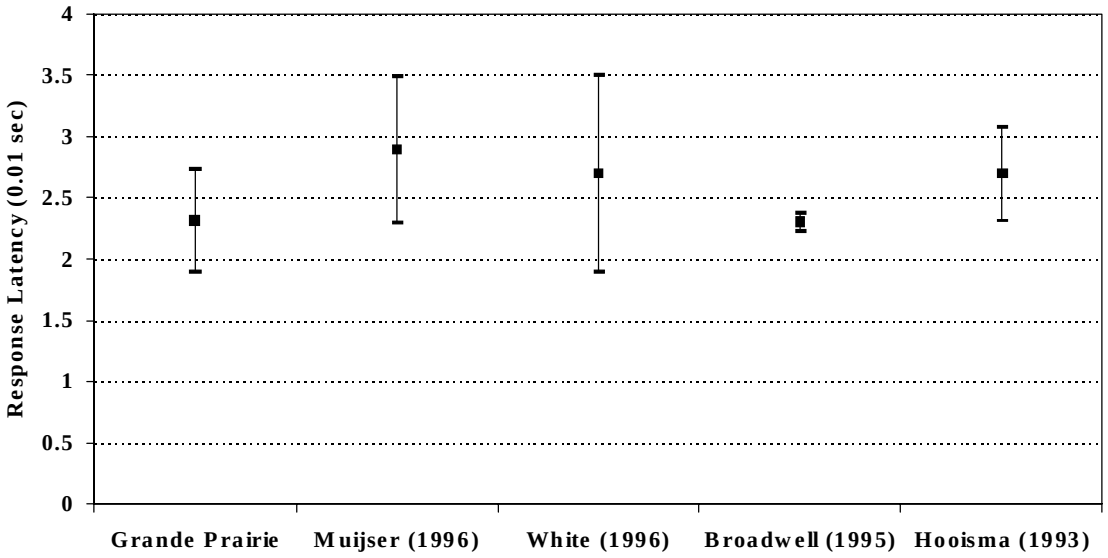



**Figure 88: Pattern Comparison Test**

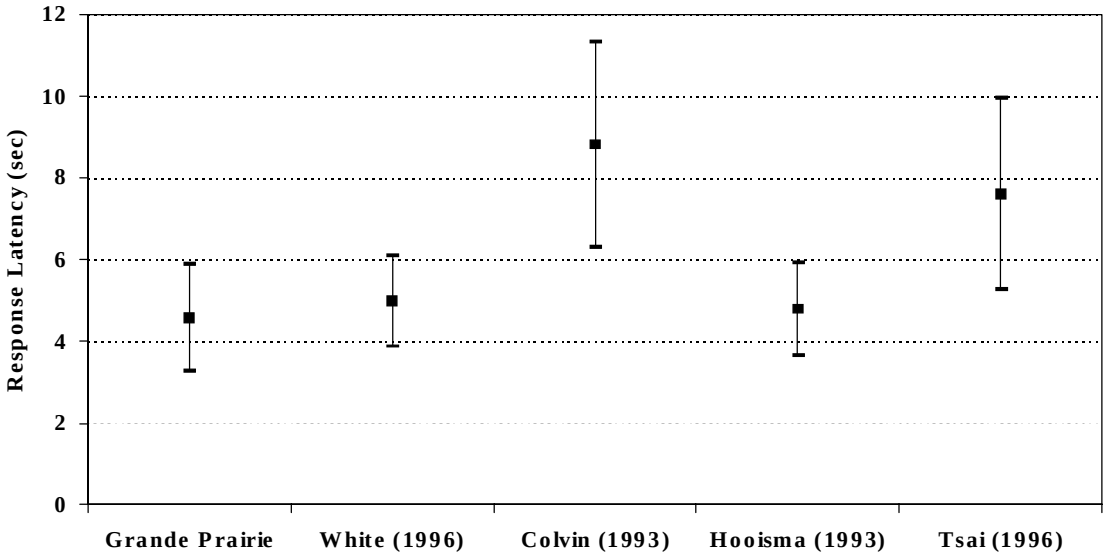


**Figure 89: Pattern Memory**

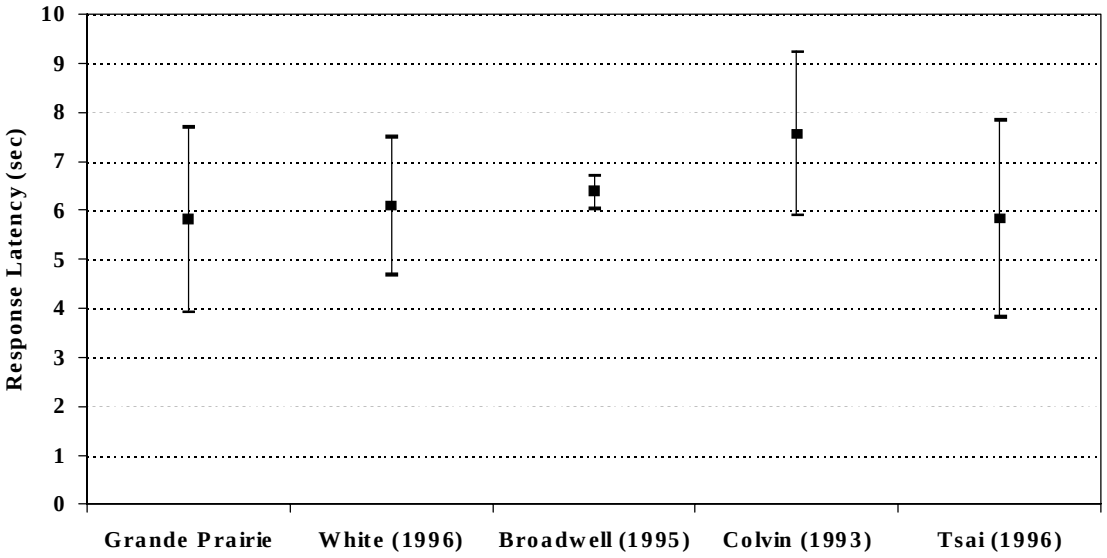



**Figure 90: Serial Digit Learning Test**

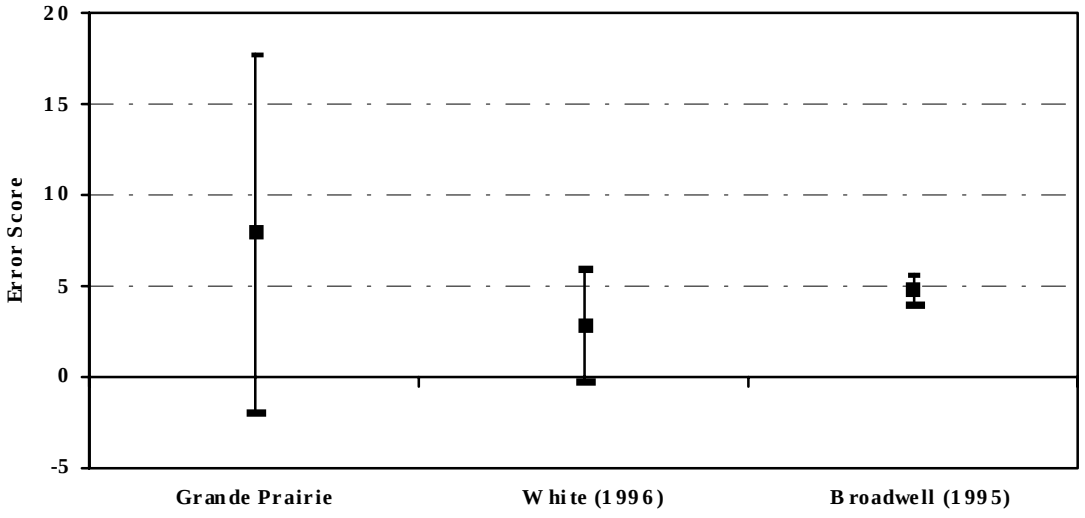


**Figure 91: Colour-Word Test**

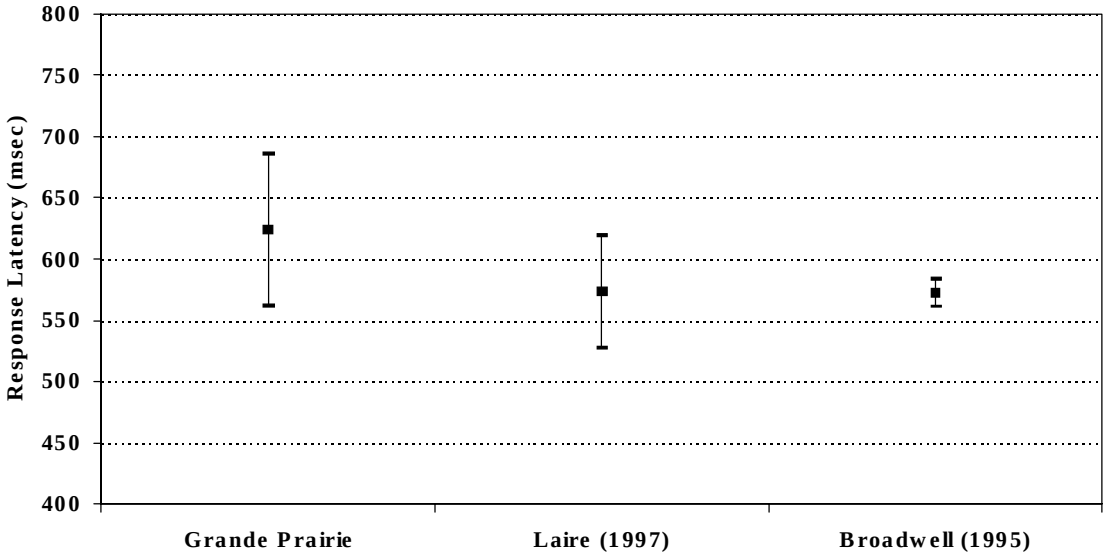



**Figure 92: Vocabulary Test**

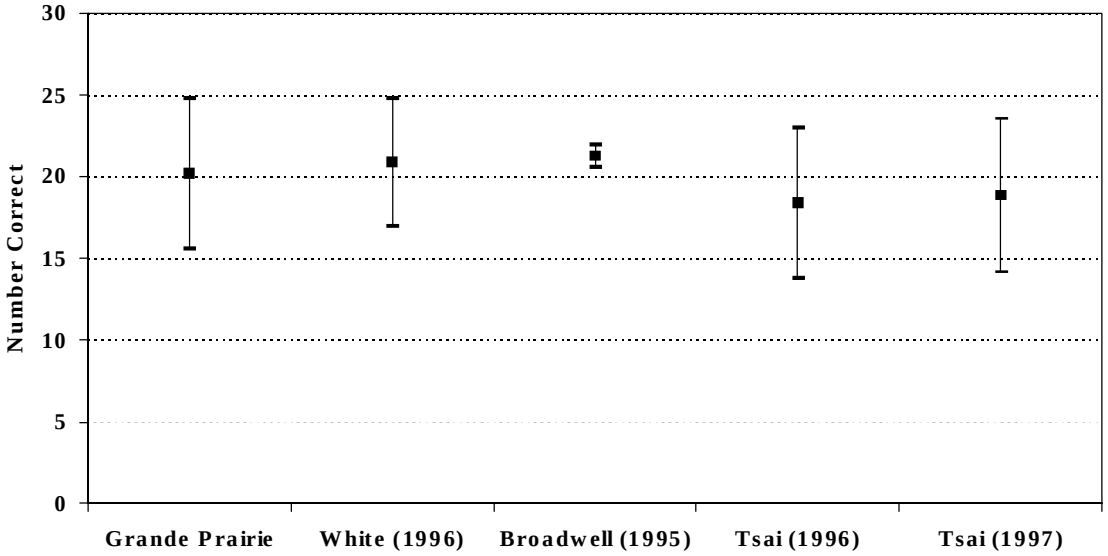
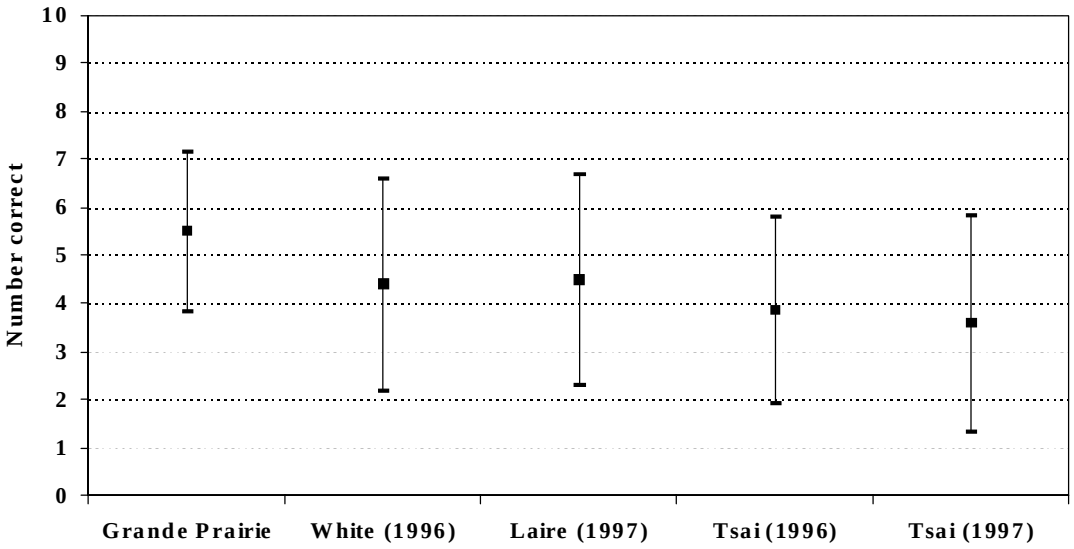


**Figure 93: Delayed Associate Recognition Test**




### *Symptoms Questionnaire*

A symptom questionnaire was administered to measure the frequency of experiencing a variety of symptoms often associated with exposure to neurotoxic agents. Participants were required to estimate how often they experienced each of a number of symptoms in the past month, rating each symptom on a scale ranging from 1 (did not occur) to 4 (occurred a lot). The percentage of participants experiencing each symptom is presented in Table 47. Very few participants reported experiencing symptoms listed. A small percentage of participants indicated frequently experiencing: feeling tired (16.2%), having difficulty falling asleep (10.3%), lack of sexual drive (14.7%), having to make notes to remember things (11.0%), and dry skin (4.4%).

**Table 47: Frequency of Experiencing Symptoms**

| Symptom | Percentage of Sample (N=140) | | | |
|---|---|---|---|---|
| | Not at all | A little | Fair | A lot |
| Feeling tired | 3.7 | 46.3 | 33.8 | 16.2 |
| Difficulty concentrating | 33.8 | 53.7 | 11.0 | 1.5 |
| Difficulty remembering things | 24.3 | 62.5 | 10.3 | 2.9 |
| Seizures | 99.3 | 0.0 | 0.0 | 0.7 |
| Headaches | 49.3 | 35.3 | 10.3 | 5.1 |
| Difficulty falling asleep | 45.6 | 36.8 | 7.4 | 10.3 |
| Lack of sexual drive | 30.9 | 38.2 | 16.2 | 14.7 |
| Tingling in my fingers or toes | 75.7 | 15.4 | 5.1 | 3.7 |
| Loss of appetite | 76.5 | 22.1 | 0.7 | 0.7 |
| Diarrhea | 58.8 | 33.1 | 6.6 | 1.5 |
| Dry mouth | 55.1 | 33.8 | 10.3 | 0.7 |
| Feeling depressed for no reason | 62.5 | 30.1 | 5.9 | 1.5 |
| Confusion | 64.0 | 30.9 | 4.4 | 0.7 |
| Having to make notes to remember | 22.1 | 42.6 | 24.3 | 11.0 |
| Hallucinations | 99.3 | 0.7 | 0.0 | 0.0 |
| Heart palpitations | 86.0 | 11.8 | 1.5 | 0.7 |
| Lack of coordination | 73.5 | 24.3 | 1.5 | 0.7 |
| Sleeping more than usual | 75.7 | 20.6 | 2.9 | 0.7 |
| Perspiring for no reason | 86.0 | 11.8 | 1.5 | 0.7 |
| Skin dryness | 56.6 | 28.7 | 10.3 | 4.4 |
| Unexplained weight loss | 99.3 | 0.7 | 0.0 | 0.0 |
| Indigestion | 54.4 | 37.5 | 5.1 | 2.9 |
| Excessive salivation | 96.3 | 2.2 | 1.5 | 0.0 |
| Feeling irritable | 36.0 | 49.3 | 14.0 | 0.7 |
| Feeling light-headed or "high" | 83.8 | 14.0 | 1.5 | 0.7 |
| Lack of muscle strength | 62.5 | 30.1 | 4.4 | 2.9 |
| Tightness in my chest | 86.0 | 10.3 | 2.9 | 0.7 |
| Feeling excitable | 55.1 | 33.1 | 10.3 | 1.5 |
| Nausea | 83.8 | 13.2 | 1.5 | 1.5 |
| Inflamed gums | 87.5 | 11.0 | 1.5 | 0.0 |
| Feeling anxious | 42.6 | 46.3 | 8.8 | 2.2 |
| Tremor in my fingers | 94.9 | 3.7 | 0.7 | 0.7 |
| Loose teeth | 95.6 | 2.9 | 1.5 | 0.0 |
| Trembling eyelids, lips or tongue | 89.7 | 6.6 | 2.2 | 1.5 |
| Difficulty buttoning clothes | 97.8 | 0.7 | 0.7 | 0.7 |

The items of the symptom questionnaire can be further combined to form seven scales, which are shown in Table 48. The values reflect the average responses, based on the 4-point scale, of all the symptoms corresponding to their respective categories. These composite scales measure lassitude (weariness), neurasthenia (experience of physical symptoms such as tiredness or exhaustion with no physical

justification), memory, confusion, co-ordination, neurological symptoms, and physical symptoms. The scales can be interpreted as the average reported experience of symptoms associated with the scale. For example, the memory scale suggests that the sample population reported only "a little" experience with the symptoms associated with a memory deficit. The table compares scale values for the Grande Prairie sample with a representative control sample from Lethbridge. There were no significant differences between the two sample populations.

**Table 48 Symptom Composite Scales (NES2)**

| Scale | Grande Prairie Mean (SD) | Lethbridge Mean (SD) |
|---|---|---|
| Lassitude | 2.06 (0.62) | 1.98 (0.57) |
| Neurasthenia | 1.54 (0.40) | 1.55 (0.35) |
| Memory | 2.06 (0.66) | 2.14 (0.70) |
| Confusion | 1.50 (0.46) | 1.53 (0.47) |
| Co-ordination | 1.17 (0.38) | 1.21 (0.28) |
| Neurologic | 1.22 (0.28) | 1.19 (0.18) |
| Physical | 1.33 (0.25) | 1.35 (0.23) |
| Symptom mean intensity | 1.47 (0.38) | 1.45 (0.22) |

### *Neuropsychological Impairment Scale (NIS)*

The Neuropsychological Impairment Scale (NIS) was developed as a self-reported questionnaire consisting of 50 items which measure potential neuropsychological symptoms concerned with language usage, memory, sensory capabilities, head injuries, motor capabilities, frustration tolerance, and mental alertness. The NIS can be used to identify general neurocognitive deficits and as a tool for evaluating neurocognitive impairments in the general population.

The NIS was developed to produce eight separate scores. A measure of test-taking attitude (LIE) is obtained from the answers to five independent items not included in any of the other scales. A raw score sum of the 45 items yields a Global Measure of Impairment (GMI) which indicates the patient's self-perceived adaptive deficiencies. The Total Items Checked (TIC) provides an additional index of symptom presence. The Symptom Intensity Measure (SIM) is a gauge of symptom severity calculated by dividing GMI by TIC. The General Impairment Scale (PAT) identifies previous diagnosis of symptoms such as seizures, head injury, paralysis or other physical problems that may lead to possible neurocognitive deficits. The Learning-Verbal Scale (L-V) is a scale of verbal ability. The Frustration Scale (FRU) identifies behavioral signs of anger, frustration, and resentment.

In the following graphs, the results from the Grande Prairie population are compared to the control groups of other studies. There are no significant differences between the Grande Prairie sample and any of the reference populations.

**Figure 94: NIS General Measure of Impairment (GMI)**

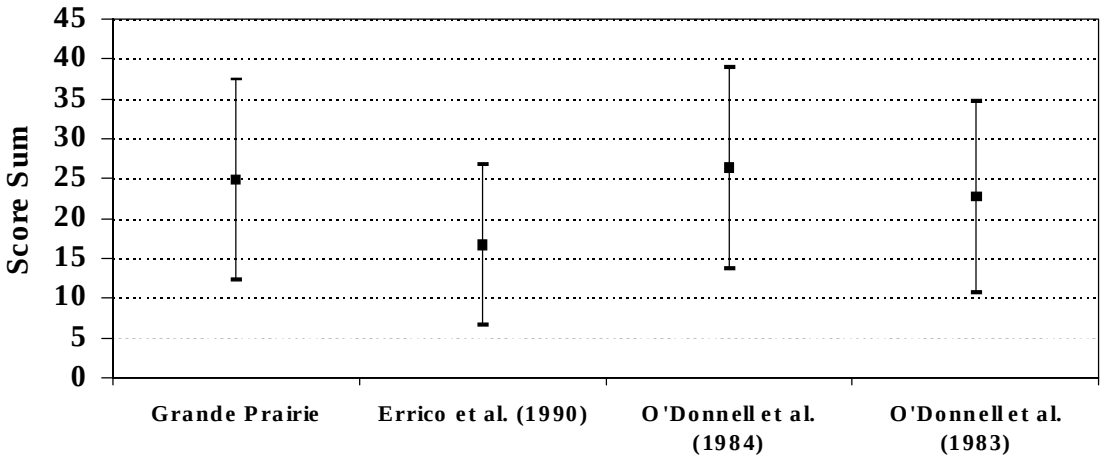



**Figure 95: NIS Total Items Checked (TIC)**

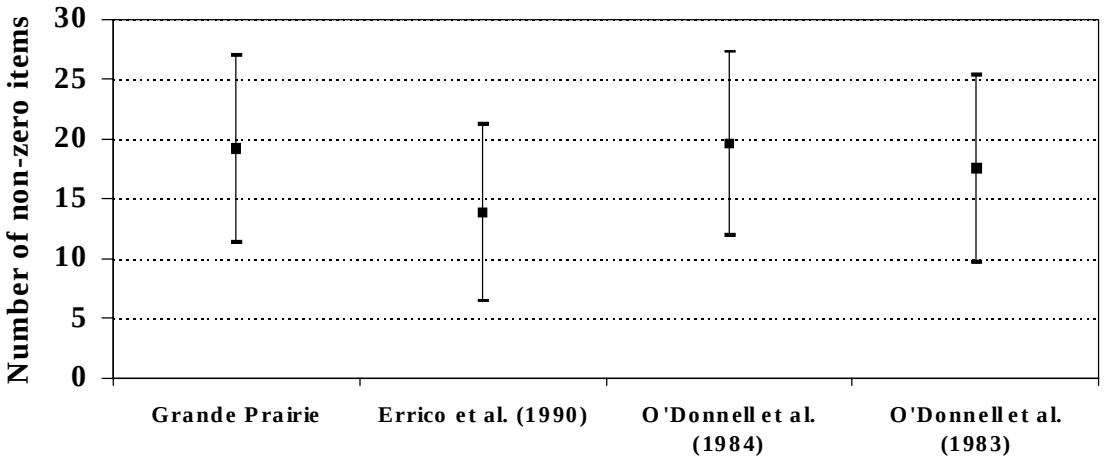


**Figure 96: NIS Symptom Intensity Measure (SIM)**

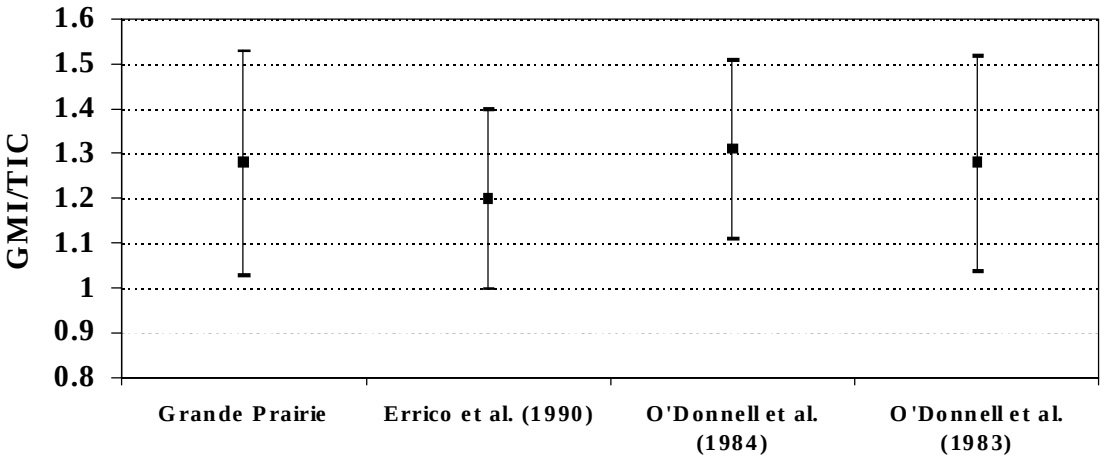



**Figure 97: NIS General Scale (GEN)**

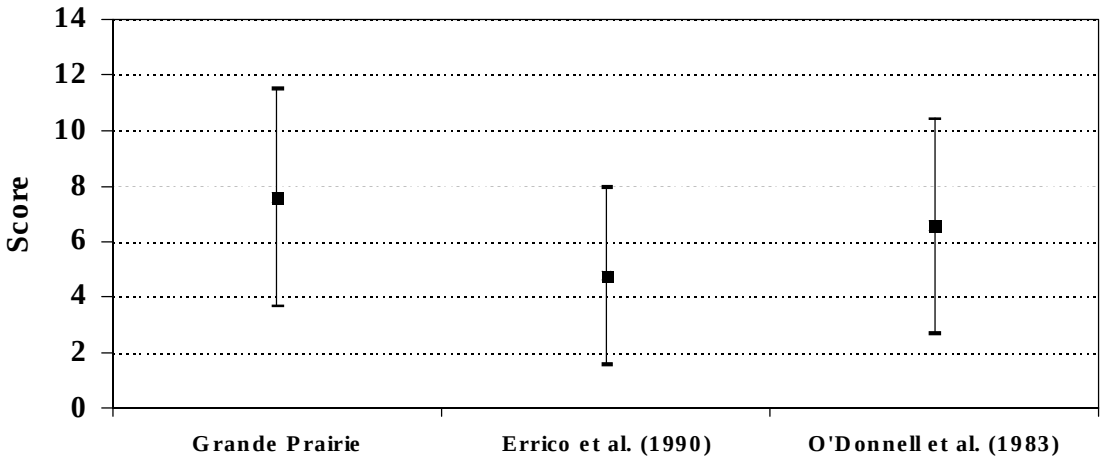


**Figure 98: NIS Pathognomic Scale (PAT)**

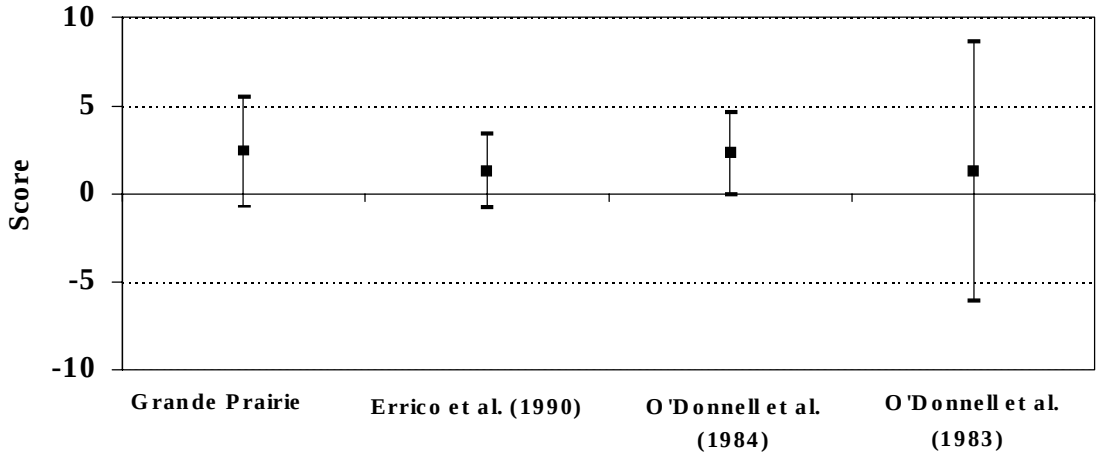



**Figure 99: NIS Frustration Scale (FRU)**

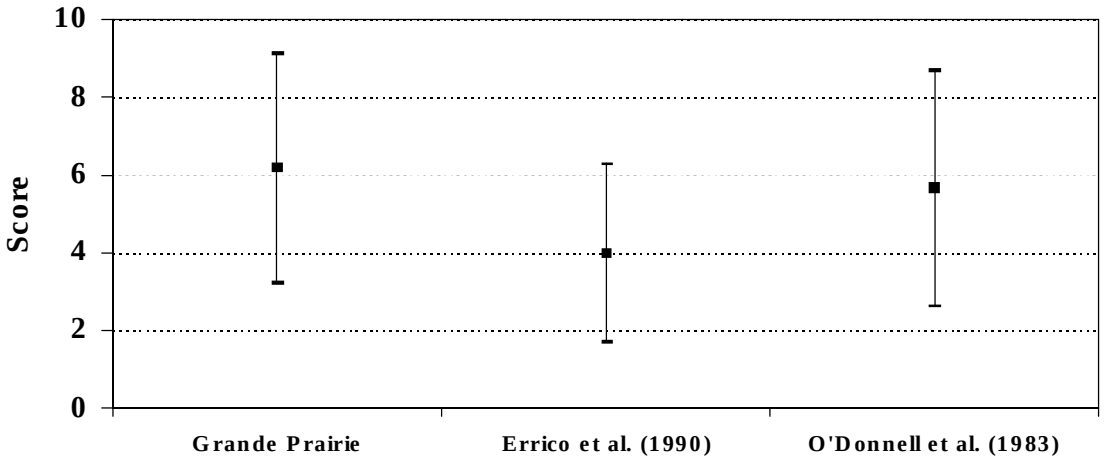


**Figure 100: NIS Learning-Verbal Scale (L-V)**

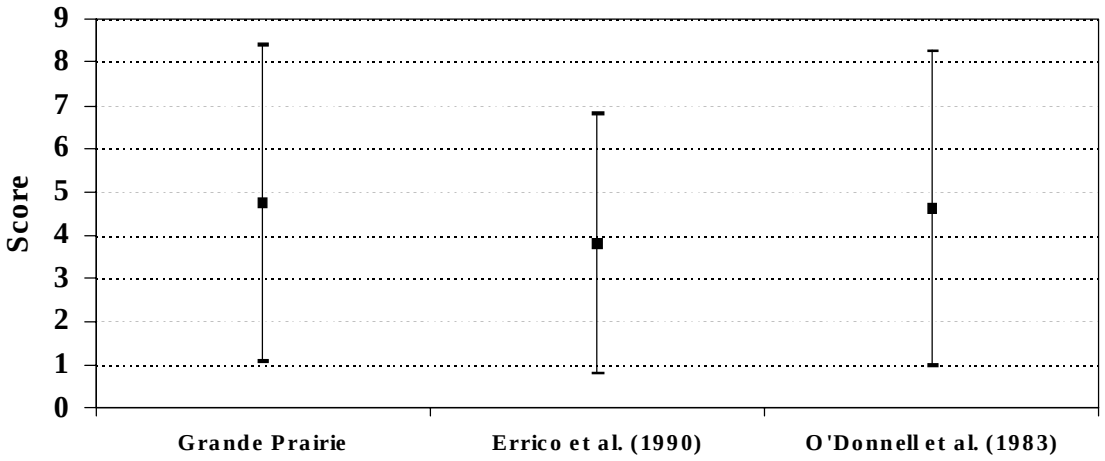
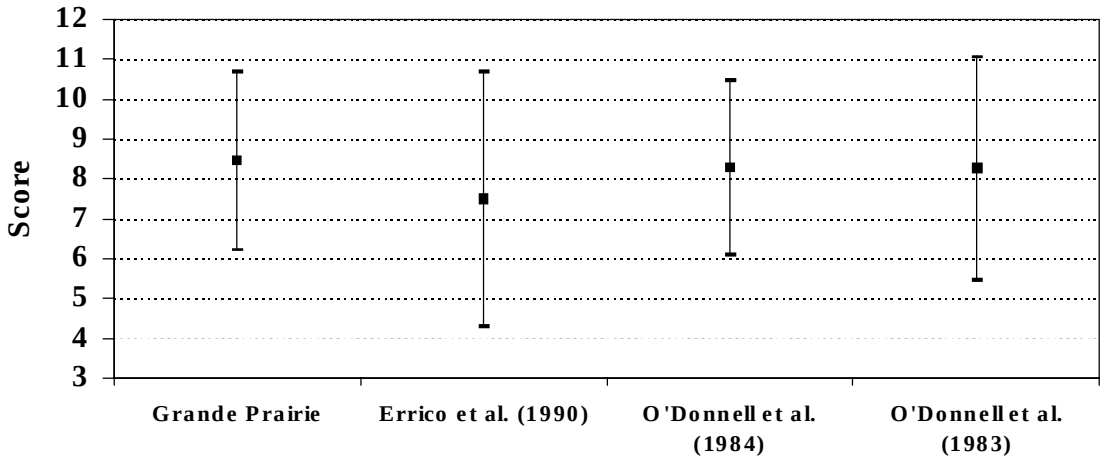



**Figure 101: NIS Lie Scale (LIE)**




### *Verbal Digit Span (VDS)*

The Verbal Digit Span is a section of the Wechsler Memory Scale – Revised (WMS-R) and was administered to each participant to include an assessment of auditory processing. There are two parts to the WMS-R version of the Digit Span, the Digits Forward and the Digits Backward, which are administered separately. During administration of the tests, each participant is asked to repeat increasingly long strings of numbers either forwards or backwards, which provides a measurement of the participants' short-term memory.

Table 49 shows the Verbal Digit Span results for both Digits Forward and Digits Backward for the Grande Prairie sample. As is shown, the results of the Grande Prairie sample did not differ significantly in comparison to the results of other researchers. In the study conducted by Amitai (1988), the control group

participants were young (average age = 22.2 years), healthy university students.[33] Fastenau (1996) used a comparable group of healthy adults with a mean age of 43.5 years.[34]

**Table 49: Verbal Digit Span**

| | Grande Prairie (n=140) | Amitai et al., 1998 (n=47) | Fastenau et al., 1996 (n=47) |
|---|---|---|---|
| Digits Forward | 8.34 (1.87) | 8.98 (1.80) | 7.5 (2.10) |
| Digits Backward | 6.64 (2.21) | 7.83 (2.00) | n/a |

### *Weekly Stress Inventory (WSI)*

The Weekly Stress Inventory (WSI) was developed as a self-report questionnaire consisting of a variety of examples of daily stressors. Each respondent was asked to identify events that occurred during the previous week, and rate the perceived stressfulness of the event on a scale from 1 (occurred, but was not stressful) to 7 (extremely stressful). The items of the WSI were selected to represent relatively minor events having a high potential of occurring in any given week. The items were also chosen to describe observable events with a discrete beginning and end. The questions focus on such events as arguments with loved ones or co-workers, problems at work, financial situations and social events which occur everyday. Two measures are calculated from the inventory: the WSI-Event scale, which measures the number of stressful events that occurred each week; and the WSI-Impact scale, which estimates the impact of the events for each participant.

The following table presents the mean scale score for Grande Prairie respondents and compares it to the mean score of the reference study from Jones & Brantley.[35] Differences between the two populations are not significant.

**Table 50: Average Number and Impact of Weekly Stressful Events**

| | Number of Events<br>Mean (S.D.) | Stress Impact<br>Mean (S.D.) |
|---|---|---|
| Grande Prairie | 25.53 (12.55) | 60.16 (41.13) |
| Jones & Brantley (1989) | 32.16 (19.46) | 105.38 (84.74) |

## 11.0 Measures of Health

Several standardized questionnaires were included to obtain measures of the participant's perceived health, as well as measures of mental and psychosocial health. The data collected using the three questionnaires are discussed below.

### 11.1 Occupational Health Questionnaire

A standard occupational health questionnaire was used to measure symptoms typically associated with the work environment. The Ontario Ministry of the Environment originally adopted it for the Windsor Winter '92 Personal Exposure Pilot (PEP) Study. The questionnaire uses a standard list of symptoms, which are characteristically associated with indoor air quality, and requires the respondent to specify the environmental location where the physical symptom is felt. Respondents were allowed to specify multiple locations.

The following table identifies the percentage of respondents from the Grande Prairie sample who reported experiencing the specified symptoms in the past year, which are then divided into the specified locations. The symptoms reported most frequently were eye and nose irritation, mental and physical fatigue, headaches, scratchy throat and cough, colds and flu, dry, itching or tearing eyes, aching joints and back pain. Participants reported experiencing dry skin, colds and flu, physical fatigue, aching joints and back pain as occurring most frequently at home while the most common symptoms at work were mental fatigue, eye irritation and strained eyes. Nose irritation occurred most frequently while commuting.

**Table 51: Frequency of Experiencing Symptoms**

| Symptom | None | Home | Work | Commuting | Combination |
|---|---|---|---|---|---|
| Eye irritation | 43.9 | 14.6 | 10.6 | 2.4 | 28.3 |
| Nose irritation | 39.8 | 20.3 | 0.0 | 10.6 | 29.1 |
| Throat irritation | 53.7 | 17.9 | 8.9 | 1.6 | 17.8 |
| Dry mucous membranes | 59.3 | 19.5 | 4.9 | 0.0 | 16.2 |
| Dry skin | 42.3 | 30.1 | 2.4 | 0.8 | 24.4 |
| Erythema | 90.2 | 4.1 | 0.0 | 0.0 | 5.7 |
| Mental fatigue | 35.0 | 17.9 | 17.1 | 1.6 | 28.5 |
| Physical fatigue | 33.3 | 24.4 | 5.7 | 0.0 | 36.6 |
| Headaches | 32.5 | 19.5 | 9.8 | 0.0 | 38.2 |
| Unspecified airway infections | 91.1 | 1.6 | 1.6 | 0.0 | 5.7 |
| Scratchy throats or coughs | 44.7 | 18.7 | 3.3 | 0.8 | 32.5 |
| Colds and flu | 35.8 | 26.8 | 2.4 | 0.0 | 35.0 |
| Nausea | 74.8 | 13.8 | 1.6 | 0.8 | 9.0 |
| Dizziness | 68.3 | 13.8 | 4.9 | 0.8 | 12.2 |
| Dry, itching or tearing eyes | 52.0 | 14.6 | 2.4 | 1.6 | 29.3 |
| Strained eyes or focusing difficulties | 48.8 | 13.8 | 13.8 | 0.0 | 23.5 |
| Chest tightness | 79.7 | 10.6 | 0.8 | 0.0 | 9.0 |
| Unspecified hyper-sensitivity | 89.4 | 2.4 | 1.6 | 0.0 | 6.5 |
| Feeling heavy headed | 82.9 | 7.3 | 3.3 | 0.0 | 6.5 |
| Difficulty concentrating | 63.4 | 8.9 | 9.8 | 0.0 | 17.8 |
| Dry facial skin | 65.9 | 15.4 | 0.0 | 0.8 | 17.9 |
| Aching joints | 45.5 | 22.0 | 3.3 | 0.0 | 29.3 |
| Muscle twitching | 72.4 | 10.6 | 0.8 | 0.0 | 16.3 |
| Back pain | 40.7 | 22.8 | 3.3 | 0.0 | 33.3 |

## 11.2 General Health Questionnaire (GHQ)

The General Health Questionnaire (GHQ) is a self-administered screening questionnaire designed to detect current, diagnosable psychiatric disorders.[36] The tool does not identify severe illness, but can identify individuals who feel they are unable to carry out their normal daily functions, focusing on changes in normal functioning rather than lifelong traits. Respondents who report 12 or more complaints are considered to have a psychosomatic disorder.[37]

The mean sum of reported symptoms was 6.3. Eighty percent of the respondents scored lower than 12 (see Table 52); 16% scored between 12 and 24; and almost 4% scored over 25. Contrary to typical findings with this measure, female respondents were somewhat less likely to report experiencing complaints or difficulties than the male respondents were, although a larger percentage of women scored over 25 compared to men.

**Table 52: GHQ Score - Percentage of Respondents by Gender**

| Score | Percentage | | |
|---|---|---|---|
| | Males | Females | Total |
| 0 - 11 | 77.5 | 81.5 | 80.0 |
| 12 - 24 | 20.0 | 13.8 | 16.2 |
| 25 + | 2.5 | 4.6 | 3.8 |

## 11.3 Medical Outcomes Study Short Form (SF-36)

The Medical Outcomes Study, conducted by the Rand Corporation in the 1970's, developed a standard questionnaire intended to provide a general indicator of health status for use in population health surveys referred to as the 36 item Short Form (SF-36). The SF-36 includes a variety of questions designed to assess limitations in usual role activities due to physical or emotional problems, limitations in physical activities, limitations in social activities, general mental health, vitality, bodily pain, and general health perceptions. The questionnaire has been used extensively and has been proven reliable and valid.

Table 53 compares the mean scale score for Grande Prairie respondents with values from a reference population.[38] Differences in most cases are likely due to small sub-sample sizes. Differences between the sample population and the reference population were also not significant.

**Table 53: Role Limitations, Vitality, Pain, and General Health Perceptions**

| | Age Category | | | | | | |
|---|---|---|---|---|---|---|---|
| | 18-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65+ | Total |
| Role Limitations, Emotional Health: Males | | | | | | | |
| Grande Prairie | N/A | 66.7 (44.4) | 88.1 (24.8) | 81.5 (33.8) | 70.0 (42.9) | 66.7 (57.7) | 76.8 (37.1) |
| Reference | 82.9 (31.1) | 87.1 (27.9) | 86.0 (28.6) | 87.5 (29.5) | 85.8 (29.9) | N/A | N/A |
| Role Limitations, Emotional Health: Females | | | | | | | |
| Grande Prairie | 100.0 (0.0) | 82.4 (29.1) | 78.8 (36.4) | 85.7 (24.9) | 78.8 (27.0) | N/A | 81.9 (29.6) |
| Reference | 78.8 (33.0) | 80.6 (34.0) | 80.3 (33.6) | 80.8 (33.6) | 83.3 (32.5) | N/A | N/A |
| Role Limitations, Physical Health: Males | | | | | | | |
| Grande Prairie | N/A | 100.0(0.0) | 91.1 (21.0) | 40.0 (39.4) | 52.5 (43.2) | 50.0 (0.0) | 71.3 (39.0) |
| Reference | 91.8 (22.6) | 92.0 (23.2) | 89.5 (25.5) | 87.6 (28.3) | 78.8 (36.1) | N/A | N/A |
| Role Limitations, Physical Health: Females | | | | | | | |
| Grande Prairie | 100.0 (0.0) | 72.1 (40.4) | 78.4 (32.1) | 75.0 (37.1) | 50.0 (41.9) | N/A | 71.9 (37.5) |
| Reference | 88.6 (25.5) | 86.9 (29.2) | 84.0 (32.0) | 82.4 (32.0) | 76.6 (36.9) | N/A | N/A |
| Physical Functioning: Males | | | | | | | |
| Grande Prairie | N/A | 90.0 (9.7) | 96.1 (4.9) | 74.0 (25.3) | 74.5 (24.5) | 60.0 (39.7) | 83.0 (21.7) |
| Reference | 92.8 (16.8) | 93.9 (14.2) | 91.9 (14.5) | 87.9 (17.4) | 80.0 (22.1) | N/A | N/A |
| Physical Functioning: Females | | | | | | | |
| Grande Prairie | 95.0 (0.0) | 93.3 (9.5) | 90.0 (16.5) | 83.6 (16.7) | 74.0 (22.9) | N/A | 86.5 (17.0) |
| Reference | 90.1 (16.4) | 92.9 (13.3) | 89.4 (16.1) | 84.8 (18.3) | 74.8 (23.5) | N/A | N/A |

**Table 53: Role Limitations, Vitality, Pain, and General Health Perceptions (cont'd)**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Social Functioning: Males | | | | | | | |
| Grande Prairie | N/A | 46.2 (8.4) | 48.2 (6.7) | 47.5 (12.9) | 50.0 (10.2) | 50.0 (0.0) | 48.1 (9.0) |
| Reference | 90.2 (16.4) | 91.3 (16.3) | 90.5 (17.0) | 89.8 (18.7) | 86.9 (22.6) | N/A | N/A |
| Social Functioning: Females | | | | | | | |
| Grande Prairie | 50.0 (0.0) | 49.3 (6.9) | 49.4 (7.2) | 43.4 (12.3) | 53.4 (9.8) | N/A | 48.3 (9.7) |
| Reference | 85.7 (19.7) | 87.1 (18.9) | 86.7 (20.5) | 87.0 (20.8) | 85.9 (22.6) | N/A | N/A |
| Mental Health: Males | | | | | | | |
| Grande Prairie | N/A | 66.8 (6.0) | 67.1 (10.7) | 71.6 (7.4) | 65.4 (9.8) | 58.7 (12.8) | 6.71 (9.3) |
| Reference | 74.8 (15.4) | 75.8 (15.2) | 75.0 (16.1) | 76.0 (16.7) | 78.0 (17.3) | N/A | N/A |
| Mental Health: Females | | | | | | | |
| Grande Prairie | 68.0 (0.0) | 65.4 (9.9) | 65.4 (10.6) | 66.3 (8.7) | 665 (9.6) | N/A | 65.9 (9.5) |
| Reference | 70.2 (17.4) | 71.6 (15.2) | 71.6 (17.8) | 73.2 (18.2) | 74.4 (18.5) | N/A | N/A |
| Vitality – Males | | | | | | | |
| Grande Prairie | N/A | 56.5 (5.3) | 60.0 (14.3) | 62.5 (10.1) | 57.7 (5.2) | 53.3 (5.8) | 58.8 (9.7) |
| Reference | 66.4 (17.1) | 64.5 (17.3) | 63.5 (18.6) | 62.9 (19.9) | 62.9 (20.3) | N/A | N/A |
| Vitality – Females | | | | | | | |
| Grande Prairie | 55.0 (0.0) | 56.5 (10.4) | 50.2 (11.0) | 57.1 (10.7) | 58.6 (7.1) | N/A | 55.1 (10.5) |
| Reference | 59.8 (19.4) | 58.3 (19.5) | 58.2 (19.9) | 59.4 (20.3) | 59.0 (21.4) | N/A | N/A |
| Bodily Pain: Males | | | | | | | |
| Grande Prairie | N/A | 83.2 (13.5) | 85.1 (16.6) | 593 (22.9) | 67.6 (22.8) | 75.0 (30.5) | 74.8 (21.7) |
| Reference | 86.6 (17.9) | 87.5 (17.7) | 85.6 (19.7) | 81.8 (22.2) | 78.8 (23.6) | N/A | N/A |
| Bodily Pain: Females | | | | | | | |
| Grande Prairie | 100.0 (0.0) | 67.5 (19.2) | 71.3 (17.7) | 70.0 (21.6) | 52.6 (22.3) | N/A | 67.6 (20.8) |
| Reference | 81.7 (20.8) | 82.1 (21.1) | 79.4 (22.0) | 77.4 (22.3) | 75.0 (25.1) | N/A | N/A |
| General Health Perceptions: Males | | | | | | | |
| Grande Prairie | N/A | 55.7 (10.7) | 58.6 (7.4) | 52.7 (5.5) | 55.1 (9.0) | 43.0 (20.1) | 55.0 (9.5) |
| Reference | 72.0 (20.1) | 76.7 (17.7) | 74.1 (18.5) | 72.0 (20.1) | 68.1 (22.9) | N/A | N/A |
| General Health Perceptions: Females | | | | | | | |
| Grande Prairie | 52.0 (0.0) | 49.5 (11.0) | 54.9 (9.3) | 55.4 (7.3) | 53.2 (7.0) | N/A | 53.4 (9.0) |
| Reference | 72.1 (20.3) | 77.3 (18.5) | 74.1 (20.3) | 73.1 (19.9) | 68.0 (22.0) | N/A | N/A |

## 11.4 Previous Diagnoses

Study participants were asked to indicate which of a series of chronic diseases they have had diagnosed by a physician. Table 54 shows the percentage of the sample population who have been diagnosed with each specified chronic condition.

The proportion of the sample population diagnosed with each chronic condition in Grande Prairie is very similar to the proportion found in previous studies of Alberta populations. Differences were not statistically significant and may be due to bias introduced by the sampling method and the small sample size.

Back problems (35%) and allergies (34%) were diagnosed most frequently for Grande Prairie residents. Almost 20% of the respondents indicated they had been diagnosed with arthritis, and more than 10% had been diagnosed with food allergies, migraine, sinusitis and asthma. None of the respondents had been diagnosed with Alzheimer's disease, other forms of dementia, or effects of stroke and less than 1% of the respondents in Grande Prairie indicated that they had been diagnosed with epilepsy, glaucoma, or kidney failure. Two percent of respondents in Grande Prairie indicated that they had been diagnosed with heart disease, alcoholism, or a nervous system disease. Five percent of the Grande Prairie sample indicated they had been diagnosed with some form of cancer. A larger percentage of the Grande Prairie sample indicated they had been diagnosed with at least one of the chronic diseases compared to the reference sample.

**Table 54: Percentage of Respondents with Previously Diagnosed Condition**

| Diagnosis | Location | |
|---|---|---|
| | Grande Prairie | Fort McMurray |
| Food Allergies | 17.4 | 12.8 |
| Other Allergies | 33.9 | 33.2 |
| Asthma | 15.7 | 13.1 |
| Bronchitis/Emphysema | 6.6 | 3.6 |
| Sinusitis | 15.7 | 12.8 |
| Arthritis | 19.8 | 14.2 |
| Back Problems | 34.7 | 22.3 |
| Diabetes | 5.8 | 2.6 |
| Epilepsy | 0.8 | 2.2 |
| High Blood Pressure | 11.6 | 9.5 |
| Heart Disease | 2.5 | 1.1 |
| Effects of Stroke | 0.0 | 0.7 |
| Cancer | 5.0 | 1.8 |
| Alcoholism | 1.7 | 1.1 |
| Urinary Incontinence | 5.0 | 1.8 |
| Kidney Failure/Disease | 0.8 | 0.7 |
| Acne requiring medication | 7.4 | 5.5 |
| Cataracts | 4.1 | 0.4 |
| Glaucoma | 0.8 | 0.4 |
| Migraine | 16.5 | 10.9 |
| Head Injury | 6.6 | 5.8 |
| Alzheimer's Disease | 0.0 | 0.0 |
| Dementia | 0.0 | 0.0 |
| Emotional Illness | 6.6 | 4.0 |
| Mental Health Condition | 7.4 | 2.9 |
| Nervous System Disease | 2.5 | 1.5 |
| None of the Diagnoses | 13.0 | 21.5 |

## 12.0 Analysis of Health Records

One of the objectives of the Grande Prairie Community Exposure and Health Effects Assessment was to describe the distribution of human health outcomes potentially associated with exposure to airborne contaminants. This section compares residents of the Mistahia Health Region with the residents of the Chinook Health Region on selected morbidity and mortality measures derived from health records.

The analysis addressed two questions:

1. is there an increased health risk for residents of the Mistahia Health Region; and
2. were the health care services obtained by the study participants representative of the services obtained by the region's population?

A population cohort from the two health regions was created from the Alberta Health Care Insurance Plan (AHCIP) Stakeholder Registry. Records for the members of the cohort were added from January 1, 1995 or thereafter, until either December 31, 2000 or until the individual died or moved out of the area. Records from the Alberta Fee-For-Service (FFS) Claims File, the Alberta In-Patient Hospital Morbidity File, and the Alberta Vital Statistics Mortality File were linked to this file. The resulting database included demographic, socio-economic, and residential history information, linked by individual and geographic area to information about physician visits, hospital stays, and deaths. Overall, there were 124,235 people residing in Mistahia Health Region and 194,149 people residing in the Chinook Health Region between January, 1995 and December, 2000. Of these, 62,537 (50.3%) from the Mistahia Health Region and 114,407 (58.9%) from the Chinook Health Region were included for all six years.

A cohort design was used for morbidity measures, focusing on the period prevalence over 6 years (1995-2000) of measurement. A cross-sectional approach was applied for analysis of overall illness (January to December 2000) and mortality (1986-1999). The mortality rate was adjusted to reflect the age distribution of the 1996 Canadian census population. Multivariate logistic regression was used to control for the effects of potential confounding from age, sex, rural residence, First Nations status, and socio-economic status in the analysis of the morbidity measures. Since the residence was used as a proxy measure of exposure, the final assessment of the cohort study was limited to permanent residents only. This included 45,659 individuals from the Mistahia Health Region and 79,155 individuals from the Chinook Health Region.

### *Characteristics of the Population*

A comparison of the population living in the Mistahia Health Region with the population living in the Chinook Health Region indicates a number of differences (see Table 55). The Mistahia Health Region had more rural residents (19.0% compared to 17.2%), but fewer seniors (5.9% vs. 10.5%), fewer First Nations people (3.4% vs. 6.4%), and fewer individuals of low (2.7% vs. 3.4%) or lower (16.0% vs. 19.2%) socio-economic status. In addition, a smaller percentage of the population remained in the region for the complete six-year period compared to the Chinook Health Region.

**Table 55: Demographic and Socioeconomic Characteristics of Health Region Populations, 1995-2000**

| Demographic and Socioeconomic Factors | Category | Mistahia Health Region | | Chinook Health Region | | |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Sex | Male | 64,024 | 51.5 | 96,972 | 49.9 | p < 0.001 |
| | Female | 60,211 | 48.5 | 97,177 | 50.1 | |
| Age Group | 0-14 | 37,357 | 30.1 | 55,848 | 28.8 | |
| | 15-64 | 79,573 | 64.1 | 117,956 | 60.8 | p < 0.001 |
| | 65+ | 7,305 | 5.9 | 20,345 | 10.5 | |
| First Nation Status [1] | Yes | 4,228 | 3.4 | 12,426 | 6.4 | p < 0.001 |
| | No | 120,007 | 96.6 | 181,723 | 93.6 | |
| Socioeconomic Status (SES) Surrogate Indicator | Lower[2] | 3,315 | 2.7 | 6,559 | 3.4 | |
| | Low[3] | 19,906 | 16.0 | 37,201 | 19.2 | p < 0.001 |
| | Average[4] | 101,014 | 81.3 | 150,389 | 77.5 | |
| Rural Residence Status[5] | Yes | 23,594 | 19.0 | 33,405 | 17.2 | p < 0.001 |
| | No | 100,641 | 81.0 | 160,744 | 82.8 | |
| Complete 6-Year Observation[6] | Yes | 62,537 | 50.3 | 114,407 | 58.9 | p < 0.001 |
| | No | 61,698 | 49.7 | 79,742 | 41.1 | |
| Mobility Status, 1995-2000[7] | Moved[8] | 16,878 | 27.0 | 35,252 | 30.8 | p < 0.001 |
| | Not Moved | 45,659 | 73.0 | 79,155 | 69.2 | |

[1] Individuals registered with AHCIP had a First Nations status at the time of registration and/or updating.
[2] Lower: Receiving both social assistance and AHCIP subsidy.
[3] Low: Receiving AHCIP subsidy only.
[4] Average: Non-AHCIP subsidy and non-social assistance recipient.
[5] Individuals with a postal code of non-city or non-town by 1996 census; the first two digits of postal code is "T0".
[6] Entered into population cohort in January 1995 and still in region by December 31, 2000.
[7] Analysis was limited to individuals with a complete 6-year follow-up.
[8] Changed residence postal code over six years.

## 12.1 Morbidity of Respiratory Disorders

Respiratory disorders, particularly asthma, have received significant attention in studies of the potential impact of ambient air quality on human health. For example, several studies have reported a positive association between ambient air pollution and hospital admissions for asthma and other respiratory disorders.[39] For the purposes of this evaluation, the analysis focused on measures of morbidity due to asthma, bronchitis, COPD, and all respiratory disorders combined.

### *Proportion of Fee for Service Practitioner Visits, Hospitalizations, and Both*

The proportion of individuals who had visited a physician, had been hospitalized, or both were calculated for the permanent residents of the Mistahia Health Region and the Chinook Health Region during the 6-year period. As shown in Table 56, the proportion varied by diagnostic category and by study area.

**Table 56: Proportion of Residents Visiting a Physician and/or Hospital for Respiratory Disorders**

| Disease Group (ICD-9) | Visit Group[1] | Mistahia Health Region | | Chinook Health Region | | Group Comparison | |
|---|---|---|---|---|---|---|---|
| | | N | % | N | % | Ratio[2] | p-value[3] |
| Asthma (493) | Both | 351 | 0.77 | 806 | 1.02 | 0.75 | |
| | HV only | 69 | 0.15 | 128 | 0.16 | 0.93 | < 0.001 |
| | PV only | 3,376 | 7.39 | 8,592 | 10.85 | 0.68 | |
| | No visit | 41,863 | 91.69 | 69,629 | 87.97 | 1.04 | |
| Bronchitis (490, 491) | Both | 204 | 0.45 | 265 | 0.33 | 1.33 | |
| | HV only | 123 | 0.27 | 161 | 0.20 | 1.32 | < 0.001 |
| | PV only | 5,370 | 11.76 | 11,091 | 14.01 | 0.84 | |
| | No visit | 39,962 | 87.52 | 67,638 | 85.45 | 1.02 | |
| COPD (490-492,494, 496) | Both | 464 | 1.02 | 635 | 0.80 | 1.27 | |
| | HV only | 167 | 0.37 | 178 | 0.22 | 1.63 | < 0.001 |
| | PV only | 5,861 | 12.84 | 12,165 | 15.37 | 0.84 | |
| | No visit | 39,167 | 85.78 | 66,177 | 83.60 | 1.03 | |
| All Respiratory Disorders (460-519) | Both | 2,324 | 5.09 | 4,169 | 5.27 | 0.97 | |
| | HV only | 85 | 0.19 | 123 | 0.16 | 1.20 | < 0.001 |
| | PV only | 29,895 | 65.47 | 56,075 | 70.84 | 0.92 | |
| | No visit | 13,355 | 29.25 | 18,788 | 23.74 | 1.23 | |

[1] **Both:** An individual had at least one visit to a physician ***and*** one hospitalization for a given diagnosis **PV only:** an individual had visited a physician between January 1995 and December 2000, but had not been hospitalized; **HV only:** an individual had been hospitalized but had not visited a physician during the time period.
[2] Proportion for residents of Mistahia Health Region divided by proportion for residents of Chinook Health Region.
[3] Chi-square test for the difference in proportion between the residents of two areas.

A smaller proportion of the residents of the Mistahia Health Region visited a physician or a hospital for asthma compared to the residents of the Chinook Health Region. However, the residents of the Mistahia Health Region were more likely to be hospitalized but less likely to visit a physician for bronchitis, COPD, or all respiratory disorders. This difference may well be due to a difference in hospital admissions policies between the two regions.

### *Prevalence of Selected Respiratory Disorders by Frequency of Visit-Year*

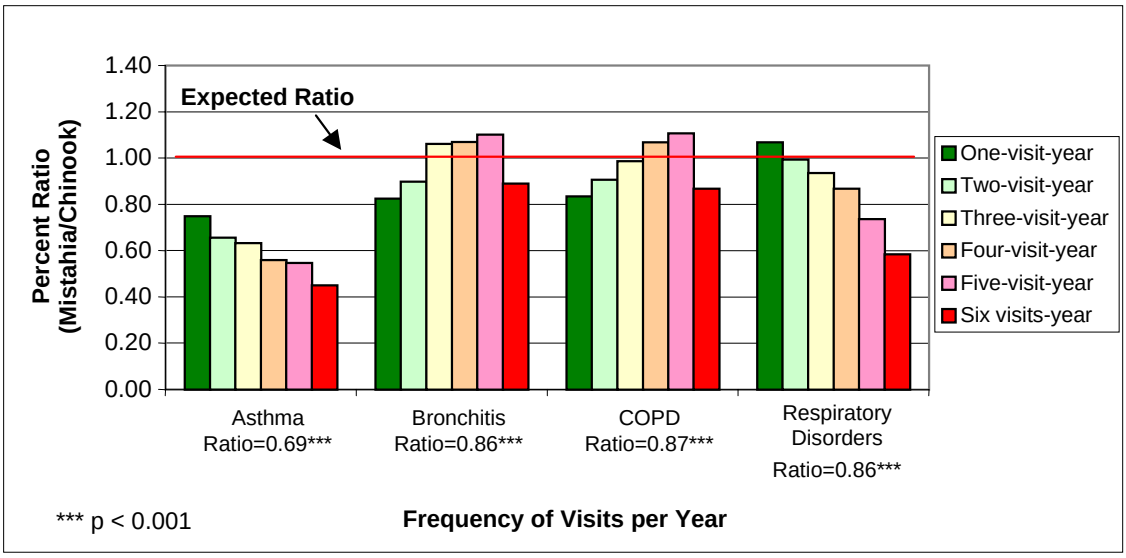
Seventy percent of the permanent residents of the Mistahia Health Region (32,304 people) visited a physician and/or were admitted to a hospital at least once for a respiratory disorder during the six year period, compared to 76.3% of the permanent residents of the Chinook Health Region (60,367 people). Only a small proportion of the residents had at least one visit every year (3.7% of Mistahia Health Region residents and 6.4% of Chinook Health Region residents).

Figure 102 shows the prevalence of visits for selected respiratory disorders for the two health regions. The first bar represents the ratio of Mistahia Health Region residents who visited a physician and/or were admitted to a hospital at least once, during only one year of the six year period compared to a similar measure for residents of the Chinook Health Region; the second bar represents the ratio of Mistahia Health Region residents who visited a physician and/or were admitted to a hospital at least once for two years during the six year period compared to Chinook Health region residents.

Overall, during the six year period, the prevalence of visits to a physician was lower for the residents of the Mistahia Health Region for asthma, bronchitis, COPD, and all respiratory disorders combined. The prevalence of visits for bronchitis for three, four and five years during the six year period was slightly

higher than expected compared to the Chinook Health Region population, but was much lower for visits for asthma. The prevalence of visits for all respiratory disorders combined was lower compared to the Chinook Health Region population, particularly for visits each of the six years.

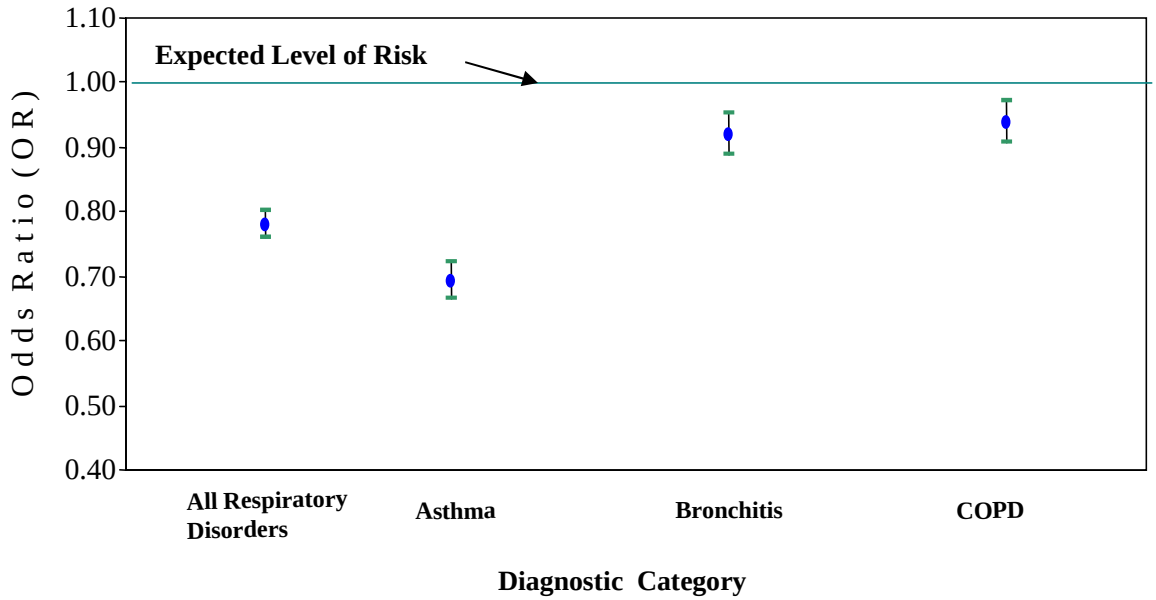
**Figure 102: Prevalence of Selected Respiratory Disorders**




### *Comparison of Prevalence of Asthma, Bronchitis, COPD, and Respiratory Disorders*

The prevalence of selected respiratory disorders for the entire six year period was estimated for the permanent residents of the Mistahia Health Region and the Chinook Health Region. The prevalence of each of these respiratory disorders was significantly lower for Mistahia Health Region residents: 8.3% of the population of the Mistahia Health Region compared to 12.0% of the population of the Chinook Health Region had asthma, 12.5% of the population the Mistahia Health Region compared to 14.5% of the population of the Chinook Health Region had bronchitis, 14.2% of the population of the Mistahia Health Region compared to 16.4% of the population of the Chinook Health Region had COPD, and 70.8% of the population of the Mistahia Health Region compared to 76.3% of the population of the Chinook Health Region had all respiratory disorders combined. This pattern remains consistent after adjusting for the potential confounding effects of age, sex, rural residence, First Nations status, and socio-economic status. Figure 103 shows the adjusted odds ratios.

**Figure 103: Estimated Relative Risk for Prevalence of Selected Respiratory Disorders: Mistahia Health Region Compared to Chinook Health Region, 1995-2000**



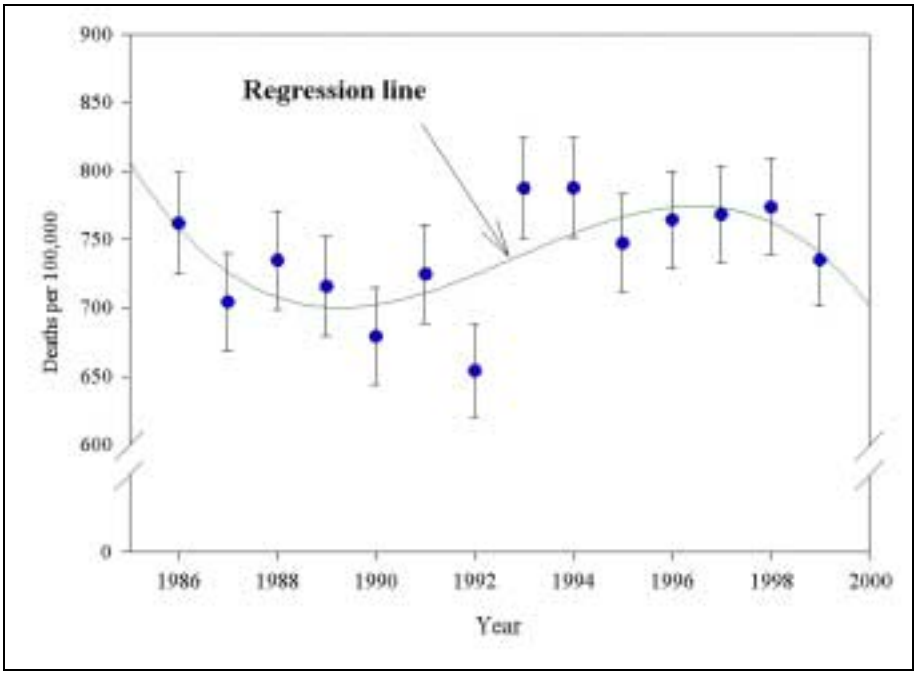

*Adjusted for the effects of sex, age, First Nations status, SES, and rural residence.*

### *Mortality*

Mortality rates have frequently been used as an outcome measure in many environmental epidemiological studies. Several studies have examined the relationship between the ambient air quality and the mortality of cardiovascular disease,[40, 41] lung cancer,[42] and death from any cause.[43, 44, 45.]

Figure 107 shows the age standardized mortality rate from all causes of death for the residents of the Mistahia Health Region. Overall, there was no increase in the standardized mortality rate for all causes of death (although the mortality rate was slightly lower in 1992 and higher in 1993 and 1994).

**Figure 104: Age-Standardized Mortality Rate of All Causes of Death, Mistahia Region, 1986-1999**

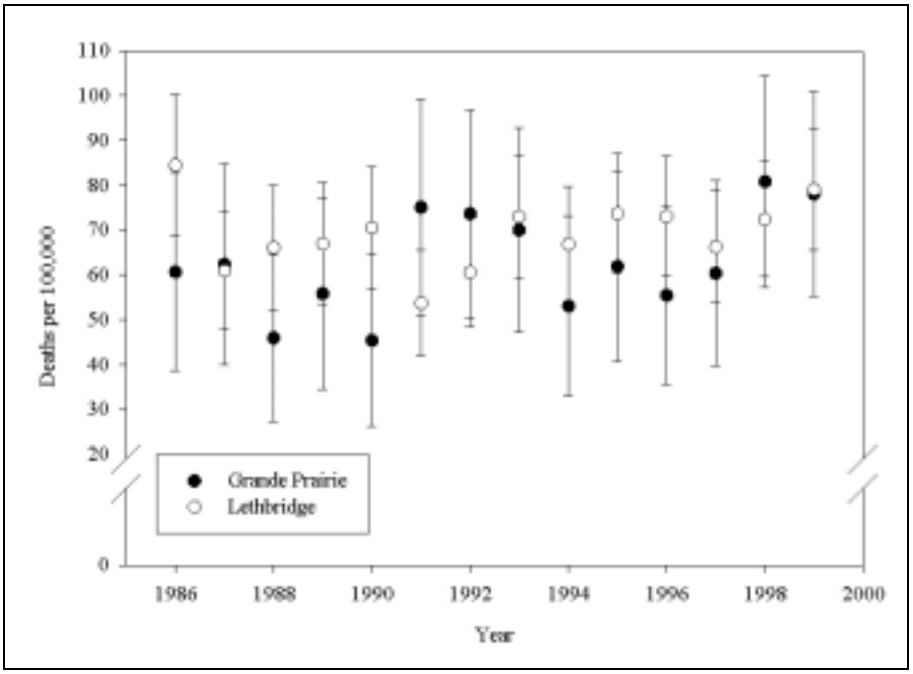



*Note: 1) Adjusted to 1996 Canadian Census age distribution.*
*2) All causes of death of underlying disease: ICD-9 = 001-999*
*Source: Health Surveillance, Alberta Health and Wellness. Epidemiological Measures Database, 2001 edition.*

The rate of mortality from respiratory disorders, from COPD and from all causes combined between the years 1986 to 1999 were compared for the residents of the Mistahia Health Region and the residents of the Chinook Health Region. There was no evidence of an increased risk of death for residents of the Mistahia Health Region from respiratory disorders, COPD or all causes of death combined for any of the 14 years compared (Figure 105: A, B, and C).

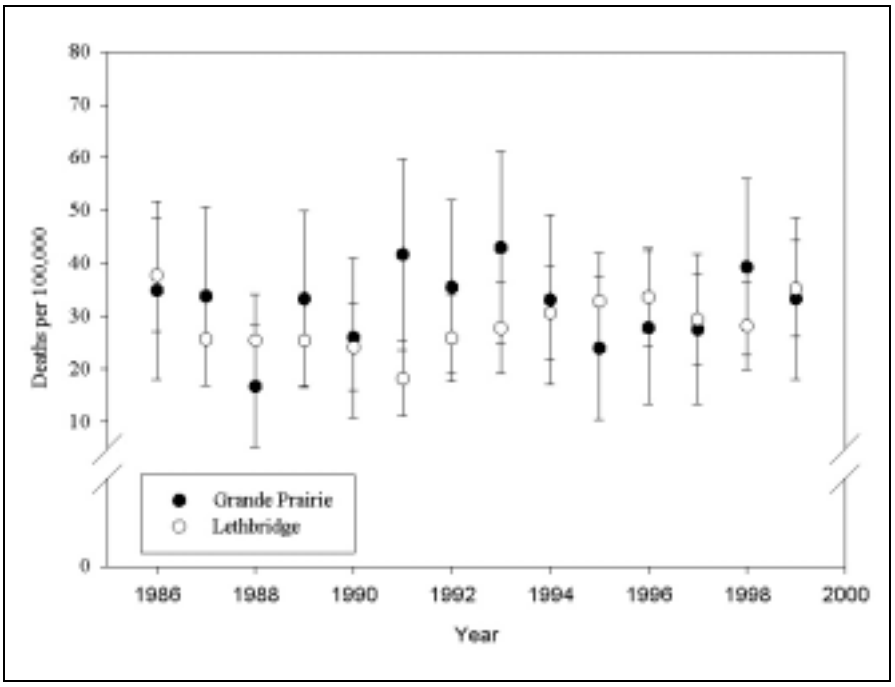
**Figure 105: Comparison of Age Standardized Mortality Rate Between Mistahia and Chinook Health Regions, 1986-1999**

**A. Respiratory Disorders**



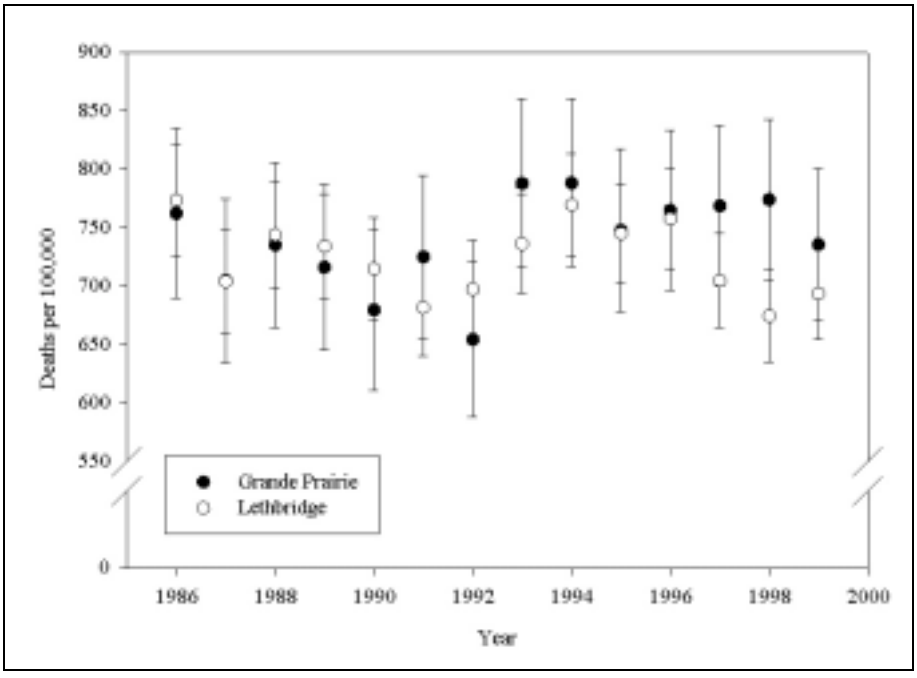

*Note: 1) Adjusted to 1996 Canadian Census age distribution.*
*2) All causes of death of underlying disease: ICD-9 = 001-999.*
*3) Respiratory Disorders: ICD-9 = 460-519.*
*Source: Health Surveillance, Alberta Health and Wellness. Epidemiological Measures Database, 2001 edition.*

**B. COPD**




*Note: 1) Adjusted to 1996 Canadian Census age distribution.*
*2) Chronic Obstructive Pulmonary Disorders (COPD): ICD-9 = 490-492, 494, 496.*
*Source: Health Surveillance, Alberta Health and Wellness. Epidemiological Measures Database, 2001 edition.*

**C. All Causes of Death**



## 12.2 Comparison of Overall Illness: Participants vs. Non-Participants

Health records for physician visits between January and December 2000 were used to compare study participants with the remaining population of the Mistahia Health Region to determine if the study participants accessed health care services differently than the other residents of the region. Of the 138 study participants, only 133 (96.4%) had provided complete information to enable accurate identification.

Among those identified, 91.7% (n=122) visited a physician between January and December 2000. This proportion is higher than the proportion (80.9%) for other residents of the region (see Table 57). After controlling for the potential confounding effects from age, sex, rural residence, First Nation status, and socio-economic status, this difference remains evident. The average number of visits per year was also higher for study participants (8 visits per year) compared to other residents of the health region (6 visits per year).

**Table 57: Proportion Visiting a Healthcare Provider and Average Number of Visits for Any Illness by Age Group, January to December 2000**

| Age Group | Participants (n=133) | | Other Residents (n=66,533) | |
|---|---|---|---|---|
| | Proportion[1] (%) | Average[2] Visits/Year | Proportion[1] (%) | Average[2] Visits/Year |
| 18-24 | 100.0 | 9.0 | 78.1 | 5.0 |
| 25-34 | 90.6 | 8.0 | 78.3 | 5.0 |
| 35-44 | 92.5 | 8.0 | 77.5 | 5.0 |
| 45-54 | 90.3 | 8.0 | 79.8 | 6.0 |
| 55-64 | 95.7 | 9.0 | 85.4 | 7.0 |
| 65+ | 100.0 | 10.0 | 94.7 | 11.0 |
| **Total** | **91.7** | **8.0** | **80.9** | **6.0** |

[1] The number of individuals who visited a Fee-For-Service practitioner at least once for any illness per 100 person-year under observation.
[2] The average number of visits per person with illness of a given age group, January to December, 2000.

## 12.3 Summary of Analysis of Health Records

Findings from the analysis of health records suggest the following:

- There is no evidence of either a significantly higher morbidity (period prevalence, frequency of visits) of asthma, bronchitis, COPD and all respiratory disorders combined in the Mistahia Health Region, nor an increased risk of death from all causes, respiratory disorders, and COPD in this area.
- There is a difference in the number of visits to a physician between the study participants and the rest of the residents of the health region non-participants. The study participants had more contact with the health care system than other residents of the Mistahia Health Region.

# 13.0 Conclusions

The goal of the Grande Prairie and Area Community Exposure and Health Effects Assessment Program was designed to explore the relationship between air quality and human health outcomes. The study collected a wide range of measures of health using both self-reported information and quantitative measures of health. Exposure levels to airborne chemicals and particulates were measured in a variety of locations and the relative contribution of various exposure sources and pathways to airborne chemicals was estimated. Finally, associations between the exposure data and human health effects were described. The key findings of the study are presented in the following sections.

## 13.1 The Study Sample

Despite an aggressive recruitment campaign, only 140 individuals volunteered for participation, and of these, only 121 provided a complete set of measures to complete the required analysis. The study sample reflected the population in terms of age, gender and education, but a larger percentage of the study sample had higher education and a greater average annual income compared to the rest of the population of the Mistahia Health Region. In addition, the study sample included fewer smokers than expected.

Study participants indicated that they consumed fewer than the recommended number of servings of grain products, and more than the recommended number of non-nutritious foods each day. The sample also had a higher average body mass index (27.3) compared to the Canadian average of 25.4, indicating more obesity in the sample population than expected. In addition, the sample population indicated that they did not get the recommended amount of exercise compared to recommendations from Health Canada.

## 13.2 Measures of Exposure

An exposure model was developed to describe the effects of nine factors on personal exposure. These nine factors were: 1) gender; 2) urban-rural location; 3) housing characteristics; 4) ownership of a garage; 5) job status; 6) smoking characteristics; 7) time activity pattern; 8) outdoor concentration levels; and 9) indoor concentration levels.

The following describes the major findings of the air quality investigation both in terms of the concentrations measured and the factors affecting the variations in personal exposure.

### *Nitrogen Dioxide ($NO_2$)*

Levels were low compared to existing guidelines and were comparable to other similar studies. Median concentrations were personal (11.6 $\mu g/m^3$), indoor (9.1 $\mu g/m^3$), and outdoor (4.7 $\mu g/m^3$). The final model predicted about 64% of the variation in personal $NO_2$ exposure across individuals. Indoor variation accounted for over one-half of the variation in personal $NO_2$ exposure described by the model. Time activity was also an important driver of personal exposure while smoking and housing characteristics had more minor effects. The most important factor within time activity appears to be the amount of time spent indoors at work; higher exposure is associated with more indoor work time.

### *Sulfur Dioxide ($SO_2$)*

Indoor and outdoor levels were very low compared to existing guidelines. Median concentrations were outdoor (0.86 $\mu g/m^3$), personal (0.37 $\mu g/m^3$), and indoor (0.17 $\mu g/m^3$). The final model predicted about 52% of the variation in personal $SO_2$ exposure across individuals. Overall, variations of outdoor levels

accounted for roughly one-half of the variation in personal $SO_2$ exposure explained by the model. Time activity was also an important factor affecting personal exposure.

### *Ozone ($O_3$)*

Indoor and personal levels of ozone were very low. Outdoor levels were an order of magnitude higher. This suggests that ambient measures are an inadequate measure of personal exposure. Median concentrations were outdoor (51.2 $\mu g/m^3$), personal (4.7 $\mu g/m^3$), and indoor (2.2 $\mu g/m^3$). The final model predicted about 69% of the variation in personal $O_3$ exposure across individuals. The variation in personal $O_3$ exposure described by the model was due to outdoor levels and time activity acting directly and indirectly through indoor levels. Indoor concentrations were also an important factor and housing characteristics were found to be of relatively minor importance.

### *Volatile Organic Compounds (VOCs)*

Levels of all measured VOCs were very low. The final models for each VOC contaminants predicted between 25-55% of variation in personal VOC exposure across individuals. For example, the benzene model predicted about 35% of variation and median concentrations were personal (1.45 $\mu g/m^3$), indoor (0.89 $\mu g/m^3$), and outdoor (0.52 $\mu g/m^3$). For the rest of the VOCs, variation in indoor concentrations is the predominant factor affecting personal exposure; the other factors were of only minor relative importance. Outdoor concentrations did not have a significant direct effect on personal exposure.

### *Particulate Matter 2.5µm ($PM_{2.5}$)*

$PM_{2.5}$ outdoor concentrations measured in Grande Prairie were lower than other communities and well below guidelines. They were not important as either a driver or a pathway of personal exposure. Median concentrations were personal (19.9 $\mu g/m^3$), indoor (8.7 $\mu g/m^3$), and outdoor (4.4 $\mu g/m^3$). Although the small number of samples preclude detailed analysis, it appears that smoking is a major contributor to influences in personal exposure through its effects on particulate concentration in indoor air.

## 13.3 Measures of Health

### *Biomarkers of Exposure*

The biomarkers of exposure were included to provide evidence of exposure to a variety of contaminants. Biomarkers for benzene, toluene, and nicotine were measurable (i.e., above detection limits), but all levels were unassociated with measures of exposure.

### *Biomarkers of Effect*

No statistically significant differences in neurocognitive functioning were found between the samples or in comparison to reference populations.

### *Self-Reported Health*

Several standardized questionnaires were included to obtain measures of the participant's perceived health, as well as measures of mental and psychosocial health. No statistically significant differences between the samples on any of the self-reported health questionnaires were identified. The most common diagnoses in Grande Prairie included back problems (35%) and allergies (34%). There is no difference in

overall illness between people who participated in the study and those who did not, although the frequency of physician visits in some groups of participants appears to be higher.

### *Health Records*

Construction and analysis of health records provided insight as to the degree to which the study samples were representative of their populations in terms of overall illness and allowed for a comparison of rates of illness and death from selected diseases.

The prevalence of asthma, bronchitis, COPD, and all respiratory disorders for Mistahia Health Region residents was compared to that of Chinook Health Region residents. The prevalence of all four disease groups is significantly lower for Mistahia residents. There is no evidence of significantly higher morbidity (incidence, prevalence, number of physician visits) of asthma or COPD or of an increased risk of death from all causes, respiratory disorders, or COPD in Mistahia compared to Chinook. Study participants had more contact with the health care system than other residents of the Mistahia Health Region.

## 14.0 Discussion

> "A series of new studies over the past decade have demonstrated a link between ambient air pollution and several adverse human health effects…"[46]
>
> "It is critical to our understanding of health and the environment that we have credible information. Continuing to improve our exposure assessment is the key to understanding this relationship … the goal of such studies (i.e., personal exposure) is to gather sound scientific evidence based on the best possible technology." (Gabos, 1998)

There is ample evidence in the peer-reviewed literature that epidemiological studies (i.e., ecological studies) have been used to establish a correlation between ambient air quality and human health outcomes. However, there is little evidence of a causal relationship.[2] Furthermore, there is very little conclusive evidence that demonstrates the contribution to personal exposure from indoor and outdoor sources.

Many previous exposure studies have relied on data from static ambient air monitoring stations to represent population exposure contaminants. It is clear from the Alberta Oil Sands Community Exposure and Health Effects Assessment Program and the Grande Prairie and Area Community Exposure and Health Effects Assessment experience that air quality data recorded at static ambient air monitoring stations does not represent, and should not be interpreted as representing, personal exposure to the contaminants being monitored. If we are to better understand the relationship between air quality and human health outcomes, it is clear that personal exposure monitoring must become part of an enhanced long-term air monitoring strategy.

This approach (i.e., personal exposure monitoring) has been recognized by the Clean Air Strategic Alliance* (CASA), and by the Alberta Multi-Stakeholder Group on Particulate Matter and Ozone** (MSG-PM/$O_3$). CASA's Human Health Project Team developed a comprehensive air quality and human health monitoring framework that recognizes and supports establishing a long-term, systematic approach to data gathering, focused on improving our knowledge about the link between air quality and human health. The components of the comprehensive human health and air quality monitoring system include:

[2] The strength of these ecological studies is that they provide evidence of an association between ambient air quality and human health. However, their weakness relates to judgements regarding causality; they lack the direct link between personal exposure to a contaminant and the resulting human health outcome. They also fail to tell us anything about individual exposure or individual risk.

- Symptoms and public health complaints;
- Known human health effects of air contaminants;
- Information about relevant event occurrences;
- Ambient air quality monitoring data; and
- Human health effects monitoring data.

Recommendations from the MSG-PM/$O_3$ to Alberta Environment included the recommendation that: "personal exposure monitoring should become part of a long-term air monitoring strategy in any region within the country and these efforts should be encouraged and supported. Personal exposure monitoring data will help us better understand the relationship between air quality and human health outcomes."

These initiatives, together with the Community Exposure and Health Effects Assessment Programs completed in Fort McMurray and Grande Prairie, recognize that data gaps currently exist that limit our understanding of the relationship between air quality and human health outcome. These include:

- Identification of the responsible component(s) of air quality that is/are causally associated with adverse health effects;
- A description of the population and personal distribution of exposure to airborne chemicals and particulates; and
- An understanding of the relative contribution of various exposure sources and pathways to airborne chemicals (i.e., the relative contribution of outdoor and indoor air to the total exposure).

Recently, the National Environmental Respiratory Center (NERC) indicated support for addressing these data gaps. It states that, "environmental air quality research and regulatory strategies have focused largely on single pollutants and sources, but it is unlikely that the health effects observed in individuals or populations are caused solely by single pollutants or sources. Indeed, as levels of most air pollutants are reduced, it is unlikely that the residual effects observed in populations are attributable to a single pollutant species or sources. There is an increasing need to know more about the contributions of individual pollutants and families of pollutants to the total effects of exposure to complex mixtures of air contaminants from man-made and natural sources. There is also a great need to better understand the health risks caused by interactions between exposures to environmental pollutants and to airborne materials encountered in the home and workplace."[47]

There is clearly a need to encourage others to develop and participate in activities that are consistent with the terms of reference and experience of the Community Exposure and Health Effects Assessment Programs completed in Fort McMurray and Grande Prairie:

- Describe the population and personal distribution of exposure to airborne chemicals and particulates:
  - estimate the population distribution of selected airborne chemicals and particulates;
  - estimate the seasonal variation of exposure; and
  - characterize the personal variation of exposure as a function of individual activity patterns.
- Quantify the relative contribution of various exposure sources and pathways to airborne chemicals:
  - quantify the relative contribution of outdoor and indoor air to the total exposure.
- Describe associations between exposure to airborne chemicals and human health effects:
  - analyze occurrence relationships between selected exposures, biomarkers and health outcomes.

***** The mandate of the Clean Air Strategic Alliance is to bring together diverse stakeholder groups to solve air quality problems on a consensus, rather than adversarial, basis.

****** The Multi-Stakeholder Group on Particulate Matter and Ozone provided recommendations to Alberta Environment related to the Canada Wide Standard process addressing Particulate Matter and Ozone.

## 15.0 Abbreviations

**AEP** – Alberta Environmental Protection

**AHCIP** – Alberta Health Care Insurance Plan

**BTEX** – benzene, toluene, ethylbenzene, and xylenes

**BMI** – body mass index

**CASA** – Clean Air Strategic Alliance

**COPD** – chronic obstructive pulmonary disease

**DNA** – deoxyribonucleic acid

**FFS** – Fee-for-Service

**GHQ** – General Health Questionnaire

**I/O** – ratio of indoor exposure to outdoor exposure

**L** – litres

**MDL** – method detection limit

**MSG-PM/$O_3$** – Alberta Multi-Stakeholder Group on Particulate Matter and Ozone

**N** – number of cases overall

**NES2** – Neurobehavioral Evaluation System

**NIS** – Neuropsychological Impairment Scale

**$NO_2$** – nitrogen dioxide

**$O_3$** – ozone

**P/I** – ratio of personal exposure to indoor exposure

**$PM_{2.5}$** – particulate matter of 2.5 microns or less (approximately 1/20 the diameter of a human hair); also called fine particles

**P/O** – ratio of personal exposure to outdoor exposure

**RHA** – Regional Health Authority

**SD** – standard deviation

**SES** – socioeconomic status

**$SO_2$** – sulfur dioxide

**TEAM** – Total Exposure Assessment Methodology

**UK** – United Kingdom

**USEPA** – United States Environmental Protection Agency

**VOCs** – volatile organic compounds

**WMS-R** – Wechsler Memory Scale – Revised

## 16.0 Definitions

**Benzene**[48]

- a water-soluble volatile organic compound (VOC) which at normal temperatures is a liquid, but readily evaporates and small amounts are detectable in the atmosphere.
- important sources are the combustion of petroleum fuels by motor vehicle engines and emissions associated with many industrial activities such as ore mining, wood processing, coal mining, textile manufacture, and processes used in the oil and gas industry.
- other sources, of which cigarette smoking is a major one, make important contributions to the exposure of individuals.
- benzene is a known carcinogen and appears on Health Canada's First Priority Substances List.

**Biomarker**

- a specific biochemical in the body which has a particular molecular feature that makes it useful for indicating environmental exposure, the progress of disease, or the effects of treatment.

**Body mass index (BMI)**

- a measure of body mass; it has the highest correlation with skinfold thickness or body density.

**BTEX compounds**

- the BTEX chemicals (benzene, toluene, ethylbenzene, and xylenes) are volatile organic compounds (VOCs) which are commonly found together in crude petroleum and petroleum products such as gasoline.
- they are also produced as bulk chemicals for industrial use as solvents and starting materials for the manufacture of pesticides, plastics, and synthetic fibres.

**Empirical**

- based on observation and experiment.

**Ethylbenzene**

- a water-soluble volatile organic compound (VOC)
- ethylbenzene is used primarily in the production of styrene; other uses include solvents in paints and varnishes, as products in synthetic rubber, household cleaning products, gasoline, pesticides, carpet glues, asphalt, and tobacco smoke.
- ethylbenzene will enter the atmosphere primarily from emissions and exhaust connected with its use in gasoline; more localized sources will be emissions, waste water, and spills from its production and industrial use.

**Median**

- the value halfway through an ordered data set, below and above which there lies an equal number of samples.

**Method detection limit (MDL)**

- the minimum concentration that can be measured and reported with confidence that the value is above zero -- that is, that the contaminant is actually present
- in this study, three standard deviations above the mean method blank levels were used as the MDL.

**Morbidity**

- the condition of being diseased or sick; a state of ill-health.

**Nitrogen dioxide ($NO_2$)**[49]

- for the purposes of air quality monitoring, oxides of nitrogen ($NO_X$) is considered to be the sum of nitric oxide and nitrogen dioxide; most oxides of nitrogen are emitted in the form of nitric oxide which will rapidly react with ozone in the atmosphere to form nitrogen dioxide.
- in Alberta, about 43% of oxides of nitrogen emissions are produced by transportation (primarily by vehicles), while 37% are due to industrial sources (oil and gas industries) and 18% as a result of power plants (based on 1990 emission estimates).
- smaller sources of oxides of nitrogen include natural gas combustion, heating fuel combustion, and forest fires.

**Ozone ($O_3$)**[50, 51]

- ozone is both a naturally occurring gas, generated in the higher layers of the atmosphere and a major constituent of photochemical smog.
- unlike other pollutants, ground-level ozone is not emitted directly by man's activities, but is generated by a photochemical reaction of oxides of nitrogen ($NO_X$) and volatile organic compounds (VOCs) in the presence of sunlight.
- in Alberta, ozone concentrations are generally lower at urban locations than at rural locations due to the destruction of ozone by nitric oxide which is emitted by vehicles.
- in Alberta, maximum ozone values are generally recorded during the spring and summer months.

**Particulate Matter (PM)**[52]

- particulate matter consists of a mixture of particles of varying size and chemical composition.
- most man-made particles are in the range of 1 to 10 microns in diameter; particles less than 10 micrometers in diameter ($PM_{10}$) are considered to be inhalable particulates and are suspended in the air for an indefinite period of time.
- $PM_{10}$ sources, which can be inhaled into the nose and throat but do not normally penetrate into the lungs, include windblown soil, road dust, dust resulting from other activities (e.g. harvest), and industrial processes, generally created during burning processes, consisting of fly ash from power plants, carbon black from diesel and gasoline engines, and soot from wood-burning.
- the fine particles ($PM_{2.5}$ and less), which can penetrate into the lungs (respirable particulates), are typically secondary aerosols that form when chemical reactions occur between sulfate (from power plants) or nitrate (from motor vehicles and industry such as oil and gas plants) and ammonia or from sources such as compressor stations, household heating appliances, and forest fires.

**Relative Risk**

- ratio of at-risk individuals to those not at risk in a group; ratio of a disease rate in the study population to the rate in the reference population.
- **adjusted relative risk:** ratio of a disease rate in the study population to the rate in the reference population when effects of confounding are taken into consideration.

**Sulfur dioxide ($SO_2$)**[53,54]

- a water-soluble irritant gas and a major pollutant in the atmosphere formed during the processing and combustion of fossil fuels containing sulfur, for example from gas plant flares, oil refineries, pulp and paper mills, fertilizer plants, coal-fired power plants, power generating stations, metal smelters, and heating boilers.
- sulfur dioxide (along with $NO_X$) has a number of other environmental effects including lake acidification due to acid rain, and associated corrosion of stone and metalwork.
- sulfur reacts in the atmosphere to form sulfuric acid and acidic aerosols which contribute to acid rain; combines with other gases to produce aerosols which may reduce visibility causing haze over large regions.
- in Alberta, it is estimated that 42% of sulfur dioxide emissions are emitted by natural gas processing plants while oil sands and power plants produce 26% and 18%, respectively, based on 1990 emission inventory.

**TEAM**

- method developed by the USEPA to determine exposures of the general population to certain pollutants.

**Toluene**

- a water-soluble volatile organic compound (VOC).
- the largest chemical use for toluene is in the production of benzene and urethane; also used as a solvent, gasoline additive, and in the manufacture of explosives, dyes, cements, spot removers, cosmetics, antifreezes, asphalt, and detergent.
- toluene is released into the atmosphere principally from the volatilization of petroleum fuels and toluene-based solvents and thinners, and from motor vehicle exhaust.
- toluene appears on Health Canada's First Priority Substances List.

**Volatile organic compounds (VOCs)**

- VOCs are chemicals that contain the element carbon.
- VOCs produce vapors readily; at room temperature and normal atmospheric pressure, vapors escape easily from volatile liquid chemicals.
- VOCs include gasoline, industrial chemicals such as benzene, solvents such as toluene and xylene, and tetrachloroethylene (perchloroethylene, the principal dry cleaning solvent).
- VOCs can be emitted naturally or as by-products of industrial processes.

**Xylene**

- a water-soluble volatile organic compound (VOC)
- found in coal and wood tar, and crude wood spirit; used primarily as solvents for which their use is increasing as a replacement for benzene and in gasoline.

- major environmental releases of xylenes are due to emissions from petroleum refining, gasoline, and diesel engines.
- xylene appears on Health Canada's First Priority Substances List.

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# Measuring Exposure to Polycyclic Aromatic Hydrocarbons:

# A Pilot Study in Grande Prairie, Alberta

March 2002

By
Health Surveillance, Alberta Health and Wellness,
Mistahia Health Region

## 1.0 Executive Summary

Public concern over the health and environmental effects of products of incomplete combustion has been increasing. In addition to outdoor anthropogenic sources of incomplete combustion including gas flaring in the oil industry, “teepee” burners in the forest industry, automobile exhaust in urban centers, controlled burning on farmland, and controlled or natural forest fires, there are also many indoor sources including fireplaces, cigarette smoking, and cooking. Understanding personal exposures to products of incomplete combustion is important to adequately address public health concerns associated with the emission of these pollutants. Information on the levels of personal exposure to products of incomplete combustion is needed to understand the human health implications. Additional information on the relative contribution of the various emission sources is valuable in identifying high impact emissions and can provide evidence for decision making in support of public policies affecting emissions of health concern.

Health Surveillance has undertaken a pilot study to measure exposure to polycyclic aromatic hydrocarbons (PAHs) as an indicator of products of incomplete combustion.

There were two main study objectives: 1) to evaluate techniques for measuring exposures to PAHs (products of incomplete combustion); and 2) to gather preliminary data on the levels of PAHs in Grande Prairie, Alberta. The two measurement techniques evaluated were “integrated sampling” and “real-time monitoring”. Integrated sampling involves collecting PAHs on filter media over a period of time, shipping of the filters to the laboratory where the samples are extracted and analyzed using gas chromatography and mass spectrometry (GC/MS). Real-time monitoring captures and analyzes samples in the field using a device that provides moment to moment results.

The real-time PAS 2000 CE PAH monitor has demonstrated the potential to be an effective tool in monitoring PAHs. This real-time monitor differentiates between outdoor and indoor sources of PAHs and can characterize the temporal variations in air quality. The monitor effectively differentiated between relative levels changing over time at one site but was only predictive of absolute measures compared at different sites when high PAH levels, due to indoor smoking, were encountered. The real-time PAH measures combined with wind speed and direction may prove effective in apportioning outdoor PAH levels between local and regional sources.

Integrated samples were effective in comparing 7-day average absolute levels indoors, outdoors, and personally. The impact of indoor and outdoor levels on the personal exposure to PAHs was characterized by using the “fingerprint” of individual PAH compounds in the personal, indoor, and outdoor samples. These “fingerprints” can be used to identify the sources of the PAHs (Khalili *et al.,* 1995), but was not attempted in this preliminary study. Further research investigating this option is warranted.

The levels of PAHs measured in this study were similar to or lower than levels reported in other similar studies. Outdoor levels and sources drive exposure to the heavier (larger molecular weight) PAHs when there is no apparent or known contact with cigarette smoking. Smoking was the only indoor source of the heavier group of PAH compounds identified in the study. Indoor levels and sources drive exposure to the lighter group of PAH compounds and while smoking

appears to be a source of these compounds, there are other sources that appear to be more significant. Significant impacts of regional sources of PAHs on the city of Grande Prairie were not found, however, this result is based on limited data.

This study has demonstrated the capability of measuring exposures to PAHs. These techniques are currently available to be used to address concerns over exposures to products of incomplete combustion, in general, or PAHs, in particular.

## Table of Contents

## 2.0 Introduction

Public concern over the health and environmental effects of products of incomplete combustion has been increasing. In addition to outdoor anthropogenic sources of incomplete combustion including gas flaring in the oil industry, “teepee” burners in the forest industry, automobile exhaust in urban centers, controlled burning on farm land, and controlled or natural forest fires, there are also many indoor sources including fireplaces, cigarette smoking, and cooking. Understanding personal exposures to products of incomplete combustion is important to adequately address public health concerns associated with the emission of these pollutants. Information on the levels of personal exposure to products of incomplete combustion is needed to understand the human health implications. Additional information on the relative contribution of the various emission sources is valuable in identifying high impact emissions and can provide evidence for decision making in support of public policies affecting emissions of health concern.

Health Surveillance has undertaken an investigation of methods capable of characterizing exposure to products of incomplete combustion. This study investigated levels of exposure to incomplete combustion products by measuring the concentration of polycyclic aromatic hydrocarbons (PAHs). PAHs are multi-ringed compounds that can be formed by incomplete combustion processes. Some PAH compounds exhibit carcinogenic effects in humans (IARC, 1983-1985), the earliest documentation dates back over 200 years, when an increase in scrotal cancer was noted among chimney sweeps in London (Pott, 1963).

The current study involved measuring PAH levels indoors, outdoors, and in the personal breathing zone of 14 individuals, each for one consecutive 7-day period. The two complementary measurement techniques used were integrated samples and real-time monitoring. Integrated samples with laboratory analysis can provide concentrations of individual PAH compounds, but the short-term fluctuations in the levels cannot be identified. Real-time monitoring identifies short-term fluctuations in total PAHs but no information on individual PAH compounds is provided. These techniques have been widely used and are recommended by others to investigate air contamination due to products of incomplete combustion (Chuang *et al.,* 1999).

## 3.0 Objective

There were two main study objectives: 1) to evaluate techniques for measuring exposures to PAHs (products of incomplete combustion); and 2) to gather preliminary data on the levels of PAHs in Grande Prairie, Alberta.

## 4.0 Methods

Volunteers in Grande Prairie, Alberta were monitored over a consecutive 7-day period for personal, indoor, and outdoor air concentrations of PAHs between April and September 2000. Measurements of PAHs were taken using 7-day integrated samples and real-time monitoring using the PAS 2000 CE PAH monitor (Ecochem Analytics Inc.). In addition to the PAH samples, 7-day integrated samples of $SO_2$, $NO_2$, VOCs, and $O_3$, were also collected outdoors, indoors, and in the participants breathing zone (see Grande Prairie and Area Community Exposure and Health Effects Assessment Program: Final Report for results). The study involved

fourteen participants, ten from the community of Grande Prairie (population 36,000) and four from surrounding areas. The maps in Figures 1 and 2 show the locations of the participants.

### 4.1 Real-time Monitored PAH

PAHs were monitored nearly continuously indoors and outdoors at the residence of each participant for the 7-day period. The sampling method consisted of a PAS 2000 CE PAH monitor that estimates the total concentration of PAHs with four or more aromatic rings using a photo-ionization detector. The monitor was connected to a manifold and valve system that alternated, continuously drawing a sample from the indoors for a five minute period and then from the outdoors for a five minute period. A data logger was used for data collection and for timing the alterations of the valve. Average readings over a 30-second time period were recorded with the first reading of each five minute-sampling interval discarded, resulting in nine usable measures of both indoor and outdoor air every ten-minute cycle. The data plots developed from this data used one hour running averages to minimize the noise in the data.

The detection of PAH by the PAS 2000 CE monitor is based on the measurement of small electric charges induced by the photoelectric ionization of PAH adsorbed on the surface of carbon aerosols (Agnesod *et.al*, 1996). The monitor has a demonstrated ability to detect PAH compounds containing four or more aromatic rings like benzo(a)pyrene (Ramamurthi *et al.*, 1997). The PAS 2000 was not considered to be specific for individual PAH compounds but has proved effective as a total PAH aerosol monitor for a given combustion source (Wall, 1996). Evaluations of the performance of this monitor have recommended its use in estimating human exposure related to various activities that may generate PAH (Wilson *et al.*, 1994). This monitor provided a good comparison of the changes in particulate-bound PAH over time, although caution should be used when interpreting absolute levels as they may vary depending on combustion sources.

The instrument output showed a positive bias and was adjusted downwards by a value of 8 $ng/m^3$ so that the background readings would coincide with zero.

### 4.2 Integrated PAH Samples

Seven-day integrated samples of PAHs in the personal, indoor, and outdoor environments were collected at the residence of each volunteer. The sample method for the personal, indoor, and outdoor samples consisted of a particulate matter (PM) sampler to capture particulate-bound PAH followed by a polyurethane foam (PUF) filter to capture PAH in the vapor or gaseous phase. Generally, under normal temperatures the heaviest PAHs (least volatile) occur in the environment bound to the fine particulates in air while the lightest PAHs (most volatile) occur entirely in the vapor phase with the compounds between (semi-volatile) occurring in both media. There were duplicate indoor and outdoor samples collected at each site with the duplicates sent to two separate laboratories for analysis. A size selective impactor head with a cut-size of 2.5 μm (MSP Corporation and Airmetrics) containing a glass fiber filter was used as the PM sampler. The samples were analyzed for the list of PAHs in Table 1 at the Centre for Toxicology at the University of Calgary, Alberta and Axys Analytical Services in Victoria, British Columbia. Axys Analytical Services is a respected commercial laboratory and the Centre for Toxicology is a public health laboratory. Axys analyzed a complete set of personal, indoor, and outdoor samples

from each participant using a GC/MS method. The Centre for Toxicology analyzed the duplicate indoor and outdoor samples from each participant also using GC/MS. While the results for both labs correlated well, the majority of the analysis in this report uses the results from Axys because this data included the personal samples.

# 5.0 Results

The results of the two measurement techniques are provided with a brief discussion demonstrating the insights gained from the different data.

## 5.1 Real-time PAHs

Eleven of the 14 participants were successfully monitored for PAHs using the PAS 2000 CE. Patterns of PAH levels from the houses monitored are shown in Figures 3a to 3k. In these figures a blue line and a pink line represent the concentration of PAHs measured with the PAS 2000 CE indoors and outdoors, respectively. Generally, the PAH level indoors followed closely to the outdoor levels, except when indoor exposure sources occur, mainly cigarette smoking indoors, as shown in Figures 3e and 3i to 3k. Table 2 shows the average levels, a ratio of indoor over outdoor levels, and the fraction of time indoor levels exceeded outdoor levels. Clearly, the highest ratios of indoor over outdoor levels and the largest portion of the time where indoor PAH levels exceeded outdoor levels, occurred in the three (of the four) houses where smoking took place.

These data suggest that outdoor levels of PAHs have an important impact on indoor levels. Increases in outdoor levels lead to increases in indoor levels after a brief lag period. Important information on the time of day the outdoor increases occur can be used to speculate on the exposure sources. For example, participant 4 (Figure 3b), located in a smaller community with a highway running through, shows an interesting daily pattern of outdoor PAH levels that may be related to the traffic volume along the highway. Participant 3 (Figure 3a) shows lower outdoor levels through the night that may also have been related to traffic volume.

### 5.1.1 Local and regional sources

Studies have shown that weak air movements or calm conditions over cities are correlated with poorer air quality due to local pollution sources not being effectively dispersed (Delaney *et al*, 1998). A study in Fort McMurray, Alberta revealed that the relative impact of pollution sources in the city (local) compared with an industry 50km away (regional) could be quantified by plotting hourly contaminant levels with wind speed and direction (see The Alberta Oil Sands Community Exposure and Health Effects Assessment: Summary Report). A plot of the PAH data collected in Grande Prairie is shown in Figure 4 that includes an interpolated surface representing the average. The surface is based on only 11 weeks of monitoring, which is not adequate to draw firm conclusions. Thus, the results presented here are preliminary findings. Regional sources of PAH are not detected as the figure shows the highest average levels of PAHs measured occurred in the calm or low wind speed conditions and PAH levels decrease with increasing wind speed in all directions.

### 5.2 Integrated Sampling PAHs

The results of the integrated samples of PAHs in the outdoor, indoor, and personal air is shown in Tables 3a to 3e. The table presents the air concentrations ($ng/m^3$) of the PAHs measured and lists the PAH compounds measured in order of ascending molecular weights (lightest to heaviest). The numbers beside the PAH compounds will be used for reference in later figures. Participants 7 and participants 12 to 14 indicated that smoking occurred indoors during the sampling period. As expected, the average levels measured in Grande Prairie are lower than the minimum levels measured in other studies in larger centers (Brown *et al.,* 1996; Chuang *et al.*, 1991). The outdoor levels are similar to levels measured in other rural areas of Alberta and lower than the larger centres (Alberta Environmental Protection, 1998).

The concentrations of the PAHs measured are shown pictorially for outdoor, indoor, and personal air in Figures 5 to 7. Note that the scale on the outdoor figure is much different than those for indoor and personal figures. The figures show the lighter PAH compounds were in higher concentration than the heavier compounds. In all cases the compound with the highest concentration was phenanthrene.

Figure 8 compares the indoor, outdoor, and personal air concentrations of the sum of the seven lighter compounds (1 to 7 in Table 1). The figures show a similar pattern for smoking and non-smoking households with similar indoor and personal levels that were much higher than outdoor levels. The explanation for higher indoor and personal levels in one of the smoking households is not clear since it does not appear to be related to the amount of smoking. This figure suggests that there are significant indoor sources of these more volatile PAH compounds. These indoor sources were not identified in this study, although it appears that smoking is not the most important source.

Similarly, Figure 9 compares the indoor, outdoor, and personal air concentrations of the sum of the eight heaviest (9 to 17 in Table 1). The figure shows that the highest levels were measured outdoors and the lowest measured indoors at the non-smoking households. Indoor and personal levels were significantly higher at two of the three smoking households and are related to the amount of smoking during sampling. Figure 10 shows the positive correlation between indoor and outdoor levels at the non-smoking houses. These results point to outdoor concentrations as being the driver of indoor and personal levels in non-smoking households. The only indoor source of these PAH compounds identified was smoking, with the levels increasing with the amount smoked.

#### 5.2.1 Analysis of the PAH Fingerprint

A summary of these findings are elegantly displayed in a biplot (Figure 11) which allows a comparison of the distribution of the PAH compounds measured at each sample location relative to the average distribution. The blue numbers represent the PAH compounds numbered 1 to 17 (smallest to largest), while the pink, green, and red numbers represent the personal, indoor, and outdoor sample locations of each participant, respectively. The diagram allows the comparison by sample location of the differences in the pattern and magnitude of air concentrations of the set of PAH compounds. To determine the nature of these differences, follow this basic procedure for each sample location: mentally draw a line from its co-ordinate through the '+' located on the

graph at the 0,0 point (the origin). Consider this line as a new dimension with the positive direction being from the origin outward to the sample location number. The magnitude of the deviation from the average PAH compound distributions are ordered along this dimension. Notice that the actual PAH compound distribution will differ for sample location numbers located in different quadrants of the space. Additionally, a global mode of interpretation is possible by combining all of this information as follows: sampling locations in the same radial sector have similar distributions of PAH compounds; those farther from the origin (the '+' point) have less scatter than those nearer the origin. Compounds that are very close together on the plot have similar patterns of influence and similar levels of scatter.

The figure shows two distinct clusters of the blue PAH compound markers with numbers 9 through 17 and number 2 tightly grouped, while the others are more loosely grouped in another location. These results are similar with earlier analysis, which showed that the lighter PAHs occur very differently than the heavier PAHs, although here, the lighter compound number 2, acenaphthylene is grouped with the heavier PAHs.

The figure shows that all of the outdoor sampling locations (indicated by red numbers) are in the same radial quadrant to the lower left of the origin. This suggests a similar distribution of the PAH compounds at all sites with lower levels than the average of both the heavy and light PAH compounds. Just above the cluster of outdoor sites but still roughly in the same quadrant is a group of indoor sites including participant numbers 1, 3, 4, 5, 6, 8, 9, and 10 (in green). The location of this cluster indicates that the indoor levels are similarly low in the heavy PAH group but higher in the lighter group compared to the outdoor sites. The personal sites for participants 3, 4, 5, 6, 8, 9, and 10 (pink numbers) are all in this same lower left quadrant mixed between the indoor and outdoor clusters. The location of these points indicates a similar pattern of PAH compounds in the personal air of these participants. It is interesting to note that for these participants, the pattern of the PAH compounds is the same indoors, outdoors, and personal but the outdoor levels of the lighter fraction were roughly one quarter the indoor and personal level.

Except for the personal number 1 (pink), the location of the other personal (pink) and indoor (green) sites on the figure is explained by the smoking patterns of the participants. The participants numbered 2, 7, 12, 13, and 14 all allowed smoking in the home and 7, 13, and 14 indicated that they smoked. There seem to be two exposure patterns among this group with participants 12 and 13 showing very high levels of the heavier PAHs and participants 2, 7, and 14 showing higher levels of the lighter PAHs. The figure shows that the personal levels of the smokers exceeded the indoor levels while the non-smokers living in a smoking environment showed lower personal levels compared to indoor levels.

This confirms earlier predictions that smoking was an important indoor exposure source for both heavier and lighter PAH groups. The elevated indoor levels of the lighter PAH compounds at the non-smoking households indicates that there must be other unidentified indoor exposure sources that appear more important than smoking.

### 5.2.2 Comparison of Laboratories

As described in the methods section, duplicate indoor and outdoor samples were collected with one set of samples sent to Axys Analytical Services (Axys) and one set to the Centre for Toxicology (CFT) for analysis. A scatter plot correlating the lightest (1 to 7) and heaviest (9 to 17) PAH compounds is show in Figure 12. As the figure shows there appears to be fairly good agreement between the two laboratories.

### 5.3 Comparison of the PAH measurement techniques

The two measurement techniques for PAHs provided different perspectives of the concentrations of the contaminants. The integrated samples provide the individual concentrations of the PAH compounds averaged over the sampling period which allows comparison of the changes in the profile of the PAH compounds as well as the average levels for different locations. The PAS 2000CE provides real-time readings of the total concentration of the PAH compounds with four or more aromatic rings which allows identification of short-term spikes and may indicate whether the sources were indoors or outdoors.

A plot of the correlation coefficients of the average PAS 2000 CE measures versus the different PAH compounds of the integrated samples is shown in Figure 13. The figure shows there is good correlation between the two techniques for the PAH compounds of benzo(a)anthracene and higher in molecular weight. Figure 14 shows a scatter plot of the sum of the heavier PAH compounds (8 to 17) versus the average PAS 2000 CE measures which illustrates the correlation. The figure shows that both sampling techniques identified the high levels at the houses where smoking occurred. Replotting this data in Figure 15, excluding these elevated points, demonstrates that there is no correlation between the two sampling techniques at the lower levels. The literature on the PAS 2000 advises that the instrument output is a good relative measure of PAHs, useful for comparing changing levels at one site, but may not be a good absolute measure comparable from one site to another. This data shows that the PAS instrument predicted the relatively higher levels well, but as expected, was not predictive of the lower levels at the different sites.

Similarly, the two measurement techniques for PAHs did agree when comparing the relative indoor and outdoor levels at the same location. The ratios of the indoor/outdoor PAH concentrations predicted by the two techniques are correlated in Figure 16. The figure shows that both techniques similarly identified the three participants with much higher indoor than outdoor levels. The two systems did not correlate well below a ratio of 1 with the PAS 2000 CE showing ratios between 0.85 and 1 and the integrated samples falling between 0.1 and 0.55.

## 6.0 Discussion

This study has pilot tested methods of measuring personal exposure to PAHs that can be used to characterize exposure to products of incomplete combustion. The real-time PAS 2000 CE PAH monitor has demonstrated the potential to be an effective tool in monitoring PAHs. The monitor has demonstrated the ability to differentiate between outdoor and indoor sources of PAHs and to characterize the temporal variations in indoor air quality. The monitor effectively differentiated between relative levels of both indoor and outdoor PAHs at the same site, but was only predictive

of absolute measures at different sites when measuring high levels due to indoor smoking. The real-time measure of PAHs plotted against wind speed and direction may prove effective in apportioning outdoor PAH levels between local and regional sources.

Integrated samples were effective in comparing 7-day average absolute levels indoors, outdoors, and personally. The levels measured for the non-smokers were generally low compared to other similar studies. The impact on the personal exposure to PAH originating indoors and outdoors was characterized by using the fingerprint of individual PAH compounds in the personal, indoor, and outdoor samples. Using these fingerprints to identify the sources of the PAHs has been demonstrated by others (Khalili *et al.,* 1995) but was not attempted in this preliminary study. Further research investigating this option is warranted.

## 7.0 Recommendations and Conclusions

The current study has demonstrated the capability to measure exposures to PAHs. These techniques should be used in the future to address concerns over exposures to products of incomplete combustion, in general, or PAHs, in particular.

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**Table 1: Individual PAH Compounds Analyzed in Integrated Samples**

| | |
|---|---|
| 1 | Naphthalene |
| 2 | Acenaphthylene |
| 3 | Acenaphthene |
| 4 | Fluorene |
| 5 | Phenanthrene |
| 6 | Anthracene |
| 7 | Fluoranthene |
| 8 | Pyrene |
| 9 | Benzo(a)anthracene |
| 10 | Chrysene |
| 11 | Benzofluoranthenes |
| 12 | Benzo(e)pyrene |
| 13 | Benzo(a)pyrene |
| 14 | Perylene |
| 15 | Dibenzo(a.h)anthracene |
| 16 | Indeno(1,2,3-cd)pyrene |
| 17 | Benzo(ghi)perylene |

**Table 2: Summary of Real-time PAH Data from Houses in Study**

| Participant Number | Indoor Average | Outdoor Average | In/out Ratio | Time in>out |
|---|---|---|---|---|
| 3 | 11.0 | 11.7 | 94% | 32% |
| 4 | 19.5 | 20.6 | 95% | 26% |
| 5 | 11.0 | 11.4 | 97% | 37% |
| 7 | 13.8 | 14.9 | 93% | 31% |
| 8 | 13.5 | 13.7 | 99% | 47% |
| 9 | 14.2 | 16.3 | 88% | 36% |
| 10 | 15.3 | 15.6 | 98% | 48% |
| 11 | 15.9 | 18.4 | 86% | 40% |
| 12 | 32.3 | 10.8 | 298% | 85% |
| 13 | 34.1 | 12.6 | 272% | 63% |
| 14 | 21.8 | 16.0 | 136% | 60% |

## Table 3a: Results of Integrated PAH Samples

| Compound Name | Grande Prairie #1 | | | | | Grande Prairie #2 | | | | | Grande Prairie #3 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | |
| | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor |
| 1 Naphthalene | 3.88 | 2.96 | 1.41 | 16.61 | 0.97 | 10.46 | 8.84 | 1.10 | 3.87 | 0.66 | 2.48 | 2.40 | 0.36 | 1.71 | 0.26 |
| 2 Acenaphthylene | 0.39 | 0.18 | 0.05 | 2.00 | 0.06 | 0.38 | 0.42 | 0.09 | 0.36 | 0.09 | 0.44 | 0.17 | 0.01 | 0.25 | ND |
| 3 Acenaphthene | 0.44 | 0.34 | 0.27 | ND | ND | 0.43 | 0.44 | 0.24 | ND | ND | 0.47 | 0.34 | 0.08 | ND | ND |
| 4 Fluorene | 2.23 | 1.36 | 1.21 | 4.39 | 0.98 | 2.09 | 2.21 | 1.60 | 1.18 | 0.79 | 2.17 | 1.64 | 0.18 | 1.20 | 0.23 |
| 5 Phenanthrene | 12.69 | 19.36 | 3.23 | 9.52 | 2.24 | 6.98 | 16.87 | 4.21 | 9.00 | 2.68 | 11.23 | 10.00 | 2.75 | 5.94 | 1.87 |
| 6 Anthracene | 12.17 | 0.53 | 0.17 | ND | ND | 0.24 | 0.60 | 0.29 | ND | 0.23 | 0.34 | 0.34 | 0.18 | ND | ND |
| 7 Fluoranthene | 0.49 | 0.79 | 0.61 | 0.59 | 0.37 | 0.43 | 1.00 | 0.88 | 0.42 | 0.56 | 0.52 | 0.72 | 0.45 | 0.28 | 0.29 |
| 8 Pyrene | 0.70 | 1.84 | 0.64 | 1.34 | 0.38 | 0.40 | 1.99 | 1.10 | 0.77 | 0.55 | 0.84 | 1.76 | 0.45 | 0.69 | 0.36 |
| 9 Benzo(a)anthracene | 0.02 | 0.01 | 0.01 | ND | 0.02 | 0.03 | 0.03 | 0.04 | 0.02 | 0.04 | 0.03 | ND | 0.01 | ND | ND |
| 10 Chrysene | 0.06 | 0.02 | 0.10 | ND | 0.02 | 0.08 | 0.08 | 0.13 | ND | 0.02 | ND | ND | 0.03 | ND | ND |
| 11 Benzofluoranthenes | 0.13 | 0.08 | 0.19 | ND | 0.12 | 0.18 | 0.12 | 0.19 | 0.09 | 0.15 | 0.03 | ND | 0.03 | ND | ND |
| 12 Benzo(e)pyrene | 0.07 | 0.02 | 0.08 | ND | 0.05 | 0.10 | 0.06 | 0.07 | 0.04 | 0.05 | ND | ND | ND | ND | ND |
| 13Benzo(a)pyrene | 0.04 | ND | ND | ND | ND | 0.05 | 0.03 | 0.02 | 0.04 | 0.03 | ND | ND | ND | ND | ND |
| 14 Perylene | 0.01 | ND | ND | ND | ND | 0.02 | 0.01 | ND | ND | ND | 0.08 | ND | ND | ND | ND |
| 15 Dibenzo(ah)anthracen | 0.03 | ND | ND | ND | ND | 0.04 | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| 16 Indeno(1,2,3-cd)pyren | 0.13 | 0.05 | 0.08 | ND | ND | 0.10 | 0.06 | 0.06 | ND | 0.01 | 0.04 | ND | ND | ND | ND |
| 17 Benzo(ghi)perylene | 0.20 | 0.07 | 0.13 | ND | 0.07 | 0.10 | 0.09 | 0.07 | 0.04 | 0.06 | ND | ND | ND | ND | 0.02 |
| Total | 33.7 | 27.6 | 8.2 | 34.5 | 5.3 | 22.1 | 32.9 | 10.1 | 15.8 | 5.9 | 18.7 | 17.4 | 4.5 | 10.1 | 3.0 |
| Total # 3 to 8, 1 | 32.60 | 27.18 | 7.53 | 32.46 | 4.93 | 21.03 | 31.95 | 9.43 | 15.24 | 5.48 | 18.05 | 17.19 | 4.45 | 9.82 | 3.02 |
| Total # 9 to 17, 2 | 1.09 | 0.44 | 0.64 | 2.00 | 0.34 | 1.09 | 0.91 | 0.67 | 0.59 | 0.45 | 0.62 | 0.17 | 0.08 | 0.25 | 0.02 |

## Table 3b: Results of Integrated PAH Samples

| Compound Name | Grande Prairie #4 | | | | | Grande Prairie #5 | | | | | Grande Prairie #6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | |
| | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor |
| 1 Naphthalene | 5.06 | 3.22 | 0.30 | 1.97 | 0.18 | 2.72 | 1.67 | 0.32 | ND | 0.24 | 2.68 | 1.24 | 1.49 | 1.01 | 0.51 |
| 2 Acenaphthylene | 0.25 | 0.09 | 0.02 | 0.15 | 0.06 | 0.18 | 0.10 | 0.03 | ND | 0.10 | 0.29 | 0.13 | 0.27 | 0.10 | 0.15 |
| 3 Acenaphthene | 0.89 | 0.62 | 0.05 | ND | ND | 0.44 | 0.32 | 0.08 | ND | ND | 0.43 | 0.16 | 0.10 | ND | ND |
| 4 Fluorene | 4.11 | 3.02 | 0.28 | 1.88 | 0.25 | 3.81 | 2.95 | 0.34 | ND | 0.23 | 2.14 | 0.51 | 0.14 | 0.43 | 0.17 |
| 5 Phenanthrene | 10.76 | 9.06 | 1.83 | 4.19 | 1.12 | 14.97 | 14.14 | 0.21 | ND | 1.49 | 8.21 | 9.52 | 1.59 | 5.36 | 0.95 |
| 6 Anthracene | 0.51 | ND | 0.12 | ND | ND | 0.14 | ND | 3.54 | ND | ND | 0.06 | 0.62 | 0.07 | ND | ND |
| 7 Fluoranthene | 0.47 | 0.38 | 0.27 | 0.18 | 0.11 | 0.20 | 0.80 | 0.43 | 0.04 | 0.15 | 0.54 | 1.52 | 0.34 | 0.39 | 0.12 |
| 8 Pyrene | 0.60 | 0.91 | 0.42 | 0.43 | 0.13 | 0.35 | 1.88 | 0.75 | 0.06 | 0.18 | 1.00 | 3.50 | 0.31 | 0.89 | 0.14 |
| 9 Benzo(a)anthracene | 0.04 | ND | 0.01 | ND | ND | ND | ND | 0.01 | ND | ND | 0.04 | 0.02 | 0.03 | ND | 0.01 |
| 10 Chrysene | ND | ND | 0.02 | ND | ND | ND | ND | 0.02 | ND | ND | 0.08 | 0.05 | 0.08 | ND | 0.01 |
| 11 Benzofluoranthenes | 0.21 | 0.00 | 0.01 | ND | ND | 0.01 | 0.01 | 0.02 | 0.10 | 0.02 | 0.05 | 0.03 | 0.08 | 0.05 | 0.05 |
| 12 Benzo(e)pyrene | ND | ND | ND | ND | ND | ND | ND | ND | 0.05 | 0.01 | 0.05 | 0.03 | 0.04 | 0.02 | 0.02 |
| 13Benzo(a)pyrene | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | 0.02 | 0.02 | 0.02 | ND | 0.02 |
| 14 Perylene | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | 0.02 | ND | 0.01 | ND | ND |
| 15 Dibenzo(ah)anthracen | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | 0.01 | ND | ND | ND | ND |
| 16 Indeno(1,2,3-cd)pyren | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | 0.05 | 0.04 | 0.04 | ND | ND |
| 17 Benzo(ghi)perylene | ND | ND | ND | ND | 0.00 | ND | ND | ND | 0.03 | 0.00 | 0.06 | 0.03 | 0.03 | 0.01 | 0.01 |
| Total | 22.9 | 17.3 | 3.3 | 8.8 | 1.9 | 22.8 | 21.9 | 5.8 | 0.3 | 2.4 | 15.7 | 17.4 | 4.6 | 8.3 | 2.2 |
| Total # 3 to 8, 1 | 22.40 | 17.22 | 3.27 | 8.65 | 1.79 | 22.64 | 21.76 | 5.67 | 0.10 | 2.29 | 15.06 | 17.07 | 4.03 | 8.07 | 1.88 |
| Total # 9 to 17, 2 | 0.50 | 0.09 | 0.06 | 0.15 | 0.07 | 0.19 | 0.12 | 0.08 | 0.18 | 0.13 | 0.66 | 0.35 | 0.60 | 0.18 | 0.28 |

## Table 3c: Results of Integrated PAH Samples

| Compound Name | Grande Prairie #7 | | | | | Grande Prairie #8 | | | | | Grande Prairie #9 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | |
| | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor |
| 1 Naphthalene | 5.14 | 2.61 | 0.94 | 3.45 | 0.32 | 2.00 | 1.75 | 1.60 | 0.97 | 1.21 | 2.19 | 1.65 | 0.95 | 1.16 | 0.65 |
| 2 Acenaphthylene | 1.11 | 0.44 | 0.09 | 0.81 | 0.08 | 0.13 | 0.12 | 0.20 | ND | 0.14 | 0.28 | 0.20 | 0.31 | 0.26 | 0.29 |
| 3 Acenaphthene | 1.05 | 0.48 | 0.12 | ND | ND | 0.33 | 0.28 | 0.14 | ND | ND | 0.58 | 0.42 | 0.12 | ND | ND |
| 4 Fluorene | 3.86 | 2.01 | 0.58 | 2.54 | 0.26 | 2.13 | 2.62 | 0.31 | 1.32 | 0.34 | 2.29 | 1.88 | 0.54 | 1.23 | 0.40 |
| 5 Phenanthrene | 22.89 | 19.86 | 4.68 | 52.88 | 2.42 | 7.84 | 12.49 | 3.30 | 6.85 | 2.23 | 12.32 | 12.89 | 3.40 | 10.83 | 2.05 |
| 6 Anthracene | 0.95 | 0.74 | 0.52 | ND | ND | 0.51 | 0.64 | 0.33 | ND | 0.21 | 0.78 | 0.73 | 0.33 | ND | 0.28 |
| 7 Fluoranthene | 1.08 | 1.44 | 0.74 | 2.55 | 0.23 | 0.35 | 1.01 | 0.71 | 0.38 | 0.37 | 0.45 | 0.48 | 0.55 | 0.59 | 0.35 |
| 8 Pyrene | 1.47 | 2.41 | 1.15 | 3.18 | 0.25 | 0.38 | 2.01 | 0.71 | 0.87 | 0.41 | 0.58 | 1.07 | 0.89 | 0.97 | 0.42 |
| 9 Benzo(a)anthracene | 0.08 | 0.03 | 0.03 | ND | 0.01 | 0.02 | 0.05 | 0.08 | 0.03 | 0.06 | 0.03 | ND | 0.04 | ND | 0.04 |
| 10 Chrysene | 0.12 | 0.06 | 0.07 | ND | ND | 0.04 | 0.06 | 0.14 | ND | 0.01 | 0.07 | 0.02 | 0.05 | ND | 0.04 |
| 11 Benzofluoranthenes | 0.08 | 0.03 | 0.04 | 0.09 | 0.02 | 0.06 | 0.09 | 0.17 | 0.05 | 0.10 | 0.02 | 0.02 | 0.06 | ND | 0.09 |
| 12 Benzo(e)pyrene | 0.10 | 0.05 | 0.03 | 0.05 | 0.01 | 0.02 | 0.02 | 0.06 | 0.02 | 0.04 | 0.02 | ND | 0.03 | ND | 0.04 |
| 13Benzo(a)pyrene | 0.08 | 0.03 | ND | 0.06 | ND | 0.02 | 0.02 | 0.03 | ND | 0.03 | 0.01 | ND | ND | ND | 0.01 |
| 14 Perylene | 0.02 | 0.01 | ND | ND | ND | 0.01 | ND | 0.01 | ND | ND | 0.00 | ND | ND | ND | ND |
| 15 Dibenzo(ah)anthracen | 0.01 | ND | ND | ND | ND | 0.01 | ND | 0.01 | ND | ND | 0.01 | ND | ND | ND | ND |
| 16 Indeno(1,2,3-cd)pyren | 0.13 | 0.06 | 0.03 | ND | ND | 0.04 | 0.05 | 0.07 | 0.01 | 0.01 | 0.03 | 0.02 | 0.05 | ND | 0.01 |
| 17 Benzo(ghi)perylene | 0.19 | 0.07 | 0.04 | 0.07 | 0.02 | 0.05 | 0.05 | 0.07 | 0.02 | 0.04 | 0.05 | 0.05 | 0.07 | 0.03 | 0.08 |
| Total | 38.4 | 30.3 | 9.1 | 65.7 | 3.6 | 13.9 | 21.3 | 7.9 | 10.5 | 5.2 | 19.7 | 19.4 | 7.4 | 15.1 | 4.7 |
| Total # 3 to 8, 1 | 36.44 | 29.54 | 8.71 | 64.59 | 3.48 | 13.54 | 20.81 | 7.09 | 10.40 | 4.76 | 19.19 | 19.11 | 6.79 | 14.77 | 4.15 |
| Total # 9 to 17, 2 | 1.93 | 0.79 | 0.34 | 1.07 | 0.15 | 0.39 | 0.46 | 0.84 | 0.13 | 0.42 | 0.52 | 0.30 | 0.62 | 0.28 | 0.59 |

## Table 3d: Results of Integrated PAH Samples

| Compound Name | Grande Prairie #10 | | | | | Grande Prairie #11 | | | | | Grande Prairie #12 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | |
| | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor |
| 1 Naphthalene | 3.53 | 2.81 | 0.40 | 2.10 | 0.20 | 2.63 | - | 0.66 | 1.98 | 0.51 | 1.11 | 0.96 | 0.52 | 0.75 | 0.40 |
| 2 Acenaphthylene | 0.22 | 0.07 | 0.05 | 0.11 | 0.04 | 0.31 | - | 0.36 | 0.39 | 0.23 | 0.54 | 0.47 | 0.15 | 0.40 | 0.12 |
| 3 Acenaphthene | 0.49 | 0.32 | 0.13 | ND | ND | 0.48 | - | 0.18 | ND | ND | 0.41 | 0.38 | 0.08 | ND | ND |
| 4 Fluorene | 1.33 | 1.02 | 0.40 | 0.73 | 0.11 | 1.26 | - | 0.43 | 1.31 | 0.42 | 2.11 | 0.94 | 0.38 | 1.34 | 0.37 |
| 5 Phenanthrene | 10.87 | 13.63 | 4.47 | 12.75 | 1.30 | 5.88 | - | 2.55 | 3.68 | 1.28 | 11.85 | 10.25 | 3.39 | 9.47 | 2.35 |
| 6 Anthracene | 0.33 | 1.68 | 0.19 | ND | ND | 0.25 | - | 0.16 | ND | ND | 0.98 | 0.88 | 0.51 | ND | 0.27 |
| 7 Fluoranthene | 0.38 | 0.86 | 0.64 | 0.48 | 0.18 | 0.31 | - | 0.32 | 0.23 | 0.20 | 0.72 | 1.41 | 0.55 | 0.65 | 0.32 |
| 8 Pyrene | 0.49 | 1.82 | 1.32 | 1.38 | 0.23 | 0.31 | - | 0.43 | 0.43 | 0.23 | 0.80 | 2.78 | 0.52 | 1.49 | 0.32 |
| 9 Benzo(a)anthracene | 0.01 | ND | 0.04 | ND | 0.03 | 0.02 | - | 0.03 | ND | 0.03 | 0.10 | 0.13 | 0.04 | 0.06 | 0.02 |
| 10 Chrysene | 0.04 | 0.01 | 0.10 | ND | 0.01 | 0.03 | - | 0.05 | ND | 0.03 | 0.19 | 0.30 | 0.09 | 0.03 | ND |
| 11 Benzofluoranthenes | 0.03 | 0.02 | 0.06 | ND | 0.06 | 0.08 | - | 0.09 | ND | 0.08 | 0.16 | 0.18 | 0.03 | 0.30 | 0.05 |
| 12 Benzo(e)pyrene | 0.02 | ND | 0.03 | ND | 0.03 | 0.06 | - | 0.04 | 0.02 | 0.04 | 0.21 | 0.26 | 0.04 | 0.14 | 0.02 |
| 13Benzo(a)pyrene | 0.00 | ND | ND | ND | 0.02 | 0.01 | - | ND | ND | 0.02 | 0.18 | 0.19 | 0.01 | 0.12 | 0.01 |
| 14 Perylene | 0.00 | ND | ND | ND | ND | 0.01 | - | ND | ND | ND | 0.04 | 0.04 | 0.00 | ND | ND |
| 15 Dibenzo(ah)anthracen | 0.01 | ND | ND | ND | ND | 0.02 | - | ND | ND | ND | 0.01 | 0.05 | ND | ND | ND |
| 16 Indeno(1,2,3-cd)pyren | 0.04 | 0.02 | 0.03 | ND | 0.01 | 0.04 | - | 0.05 | ND | 0.01 | 0.18 | 0.19 | 0.04 | 0.03 | 0.01 |
| 17 Benzo(ghi)perylene | 0.05 | 0.01 | 0.04 | 0.01 | 0.03 | 0.04 | - | 0.11 | 0.05 | 0.10 | 0.19 | 0.21 | 0.06 | 0.12 | 0.03 |
| Total | 17.9 | 22.3 | 7.9 | 17.6 | 2.2 | 11.8 | - | 5.5 | 8.1 | 3.2 | 19.8 | 19.6 | 6.4 | 14.9 | 4.3 |
| Total # 3 to 8, 1 | 17.43 | 22.15 | 7.55 | 17.45 | 2.02 | 11.13 | - | 4.74 | 7.63 | 2.64 | 17.97 | 17.60 | 5.96 | 13.70 | 4.03 |
| Total # 9 to 17, 2 | 0.43 | 0.13 | 0.35 | 0.12 | 0.23 | 0.63 | - | 0.72 | 0.46 | 0.54 | 1.80 | 2.02 | 0.47 | 1.21 | 0.26 |

## Table 3e: Results of Integrated PAH Samples

| Compound Name | Grande Prairie #13 | | | | | Grande Prairie #14 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Ayxs Analytical | | | Centre for Toxicology | | Ayxs Analytical | | | Centre for Toxicology | |
| | Personal | Indoor | Outdoor | Indoor | Outdoor | Personal | Indoor | Outdoor | Indoor | Outdoor |
| 1 Naphthalene | 3.91 | 4.15 | 1.14 | ND | ND | 3.96 | 0.93 | 0.55 | 1.66 | 0.20 |
| 2 Acenaphthylene | 2.11 | 1.01 | 0.35 | ND | ND | 0.47 | 0.24 | 0.05 | 0.33 | 0.06 |
| 3 Acenaphthene | 1.22 | 0.91 | 0.47 | ND | ND | 13.32 | 4.57 | 0.11 | 2.68 | ND |
| 4 Fluorene | 2.87 | 2.21 | 0.59 | ND | ND | 14.76 | 7.15 | 0.76 | 6.65 | 0.28 |
| 5 Phenanthrene | 15.63 | 10.13 | 3.62 | ND | ND | 30.97 | 39.75 | 4.89 | 36.34 | 1.37 |
| 6 Anthracene | 2.61 | 0.75 | 0.27 | ND | ND | 3.96 | 5.17 | 1.27 | ND | ND |
| 7 Fluoranthene | 2.27 | 0.60 | 0.36 | ND | ND | 1.98 | 1.99 | 0.68 | 0.97 | 0.18 |
| 8 Pyrene | 2.11 | 0.49 | 0.34 | ND | ND | 1.12 | 2.38 | 1.59 | 0.93 | 0.22 |
| 9 Benzo(a)anthracene | 0.96 | 0.16 | 0.02 | ND | ND | 0.05 | 0.04 | 0.02 | 0.03 | 0.02 |
| 10 Chrysene | 2.08 | 0.39 | 0.04 | ND | ND | 0.08 | 0.05 | 0.05 | 0.02 | 0.04 |
| 11 Benzofluoranthenes | 1.75 | 0.36 | 0.05 | ND | ND | 0.09 | 0.10 | 0.03 | 0.08 | 0.08 |
| 12 Benzo(e)pyrene | 0.63 | 0.18 | 0.02 | ND | ND | 0.05 | ND | 0.01 | 0.04 | 0.04 |
| 13Benzo(a)pyrene | 0.94 | 0.15 | 0.01 | ND | ND | 0.01 | ND | ND | ND | ND |
| 14 Perylene | 0.15 | 0.02 | ND | ND | ND | 0.01 | ND | ND | ND | ND |
| 15 Dibenzo(ah)anthracen | 0.11 | 0.02 | ND | ND | ND | 0.02 | ND | ND | ND | ND |
| 16 Indeno(1,2,3-cd)pyren | 0.57 | 0.14 | 0.03 | ND | ND | 0.05 | ND | 0.01 | ND | ND |
| 17 Benzo(ghi)perylene | 0.52 | 0.13 | 0.03 | ND | ND | 0.03 | ND | 0.01 | 0.02 | 0.03 |
| Total | 40.4 | 21.8 | 7.4 | 0.0 | 0.0 | 70.9 | 62.4 | 10.1 | 49.7 | 2.5 |
| Total # 3 to 8, 1 | 30.61 | 19.24 | 6.79 | - | - | 70.07 | 61.95 | 9.86 | 49.23 | 2.26 |
| Total # 9 to 17, 2 | 9.82 | 2.56 | 0.56 | - | - | 0.87 | 0.43 | 0.20 | 0.52 | 0.26 |

Figure 1: Map of locations of urban volunteers' residences

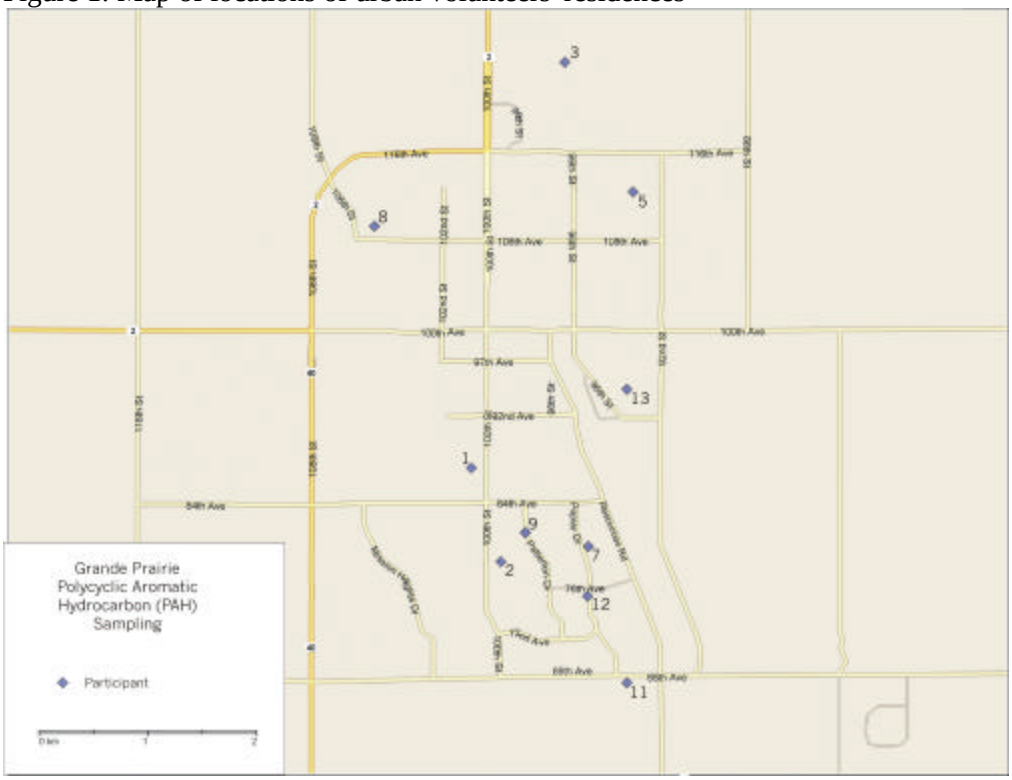


Figure 2: Map of locations of rural volunteers' residences

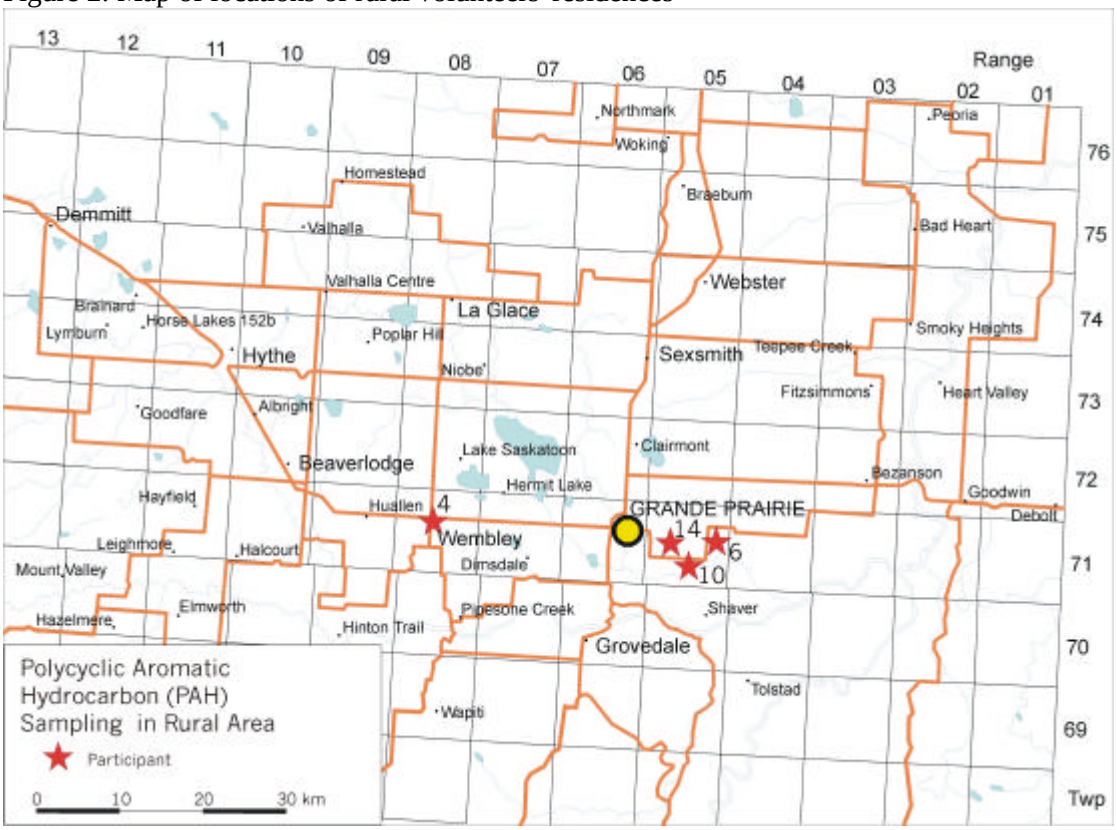

**Figure 3a: Real-time measures of PAH levels indoors and outdoors at the house of participant #3**

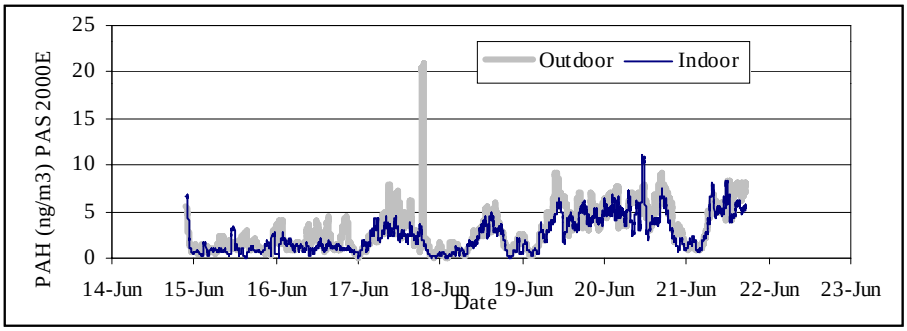


**Figure 3b: Real-time measures of PAH levels indoors and outdoors at the house of participant #4**

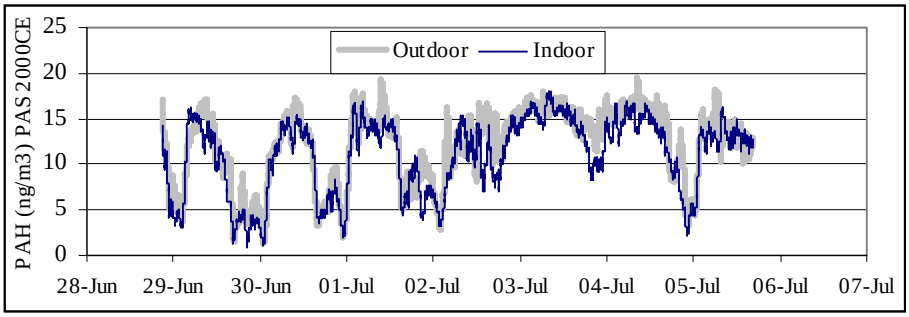


**Figure 3c: Real-time measures of PAH levels indoors and outdoors at the house of participant #5**

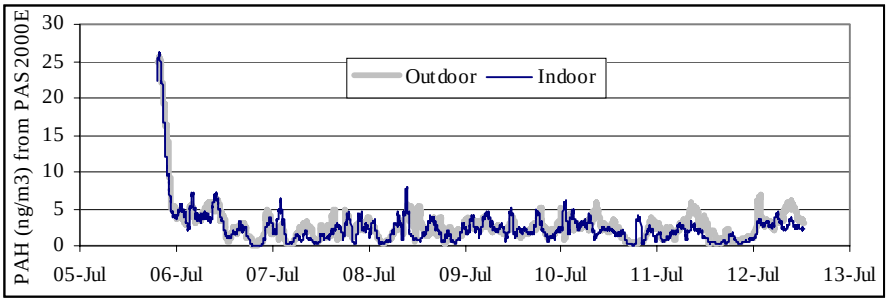


**Figure 3d: Real-time measures of PAH levels indoors and outdoors at the house of participant #7**

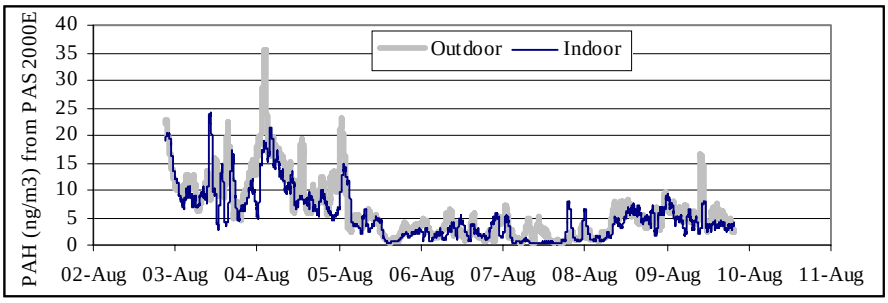

**Figure 3e: Real-time measures of PAH levels indoors and outdoors at the house of participant #8**

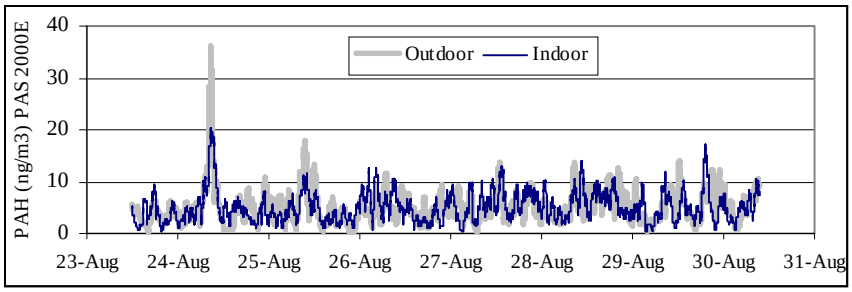


**Figure 3f: Real-time measures of PAH levels indoors and outdoors at the house of participant #9**

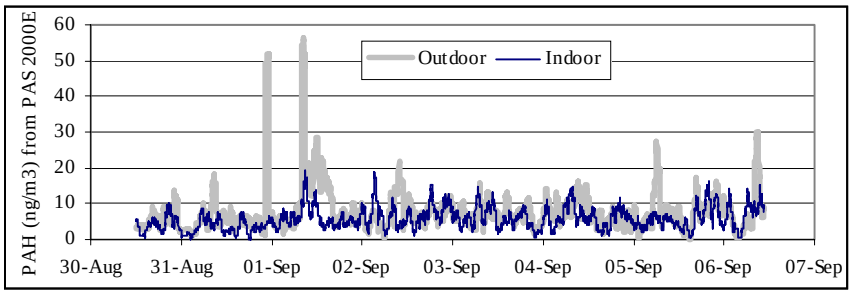


**Figure 3g: Real-time measures of PAH levels indoors and outdoors at the house of participant #10**

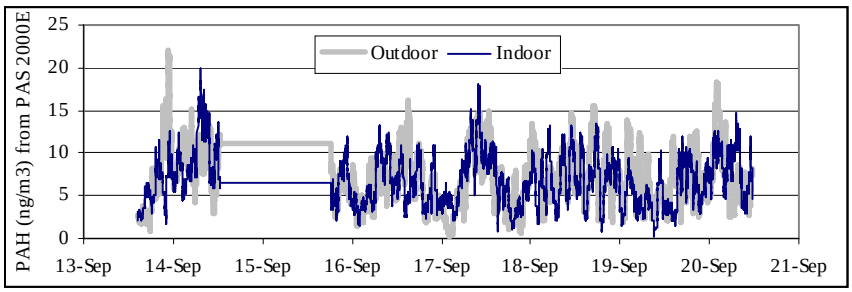


**Figure 3h: Real-time measures of PAH levels indoors and outdoors at the house of participant #11**

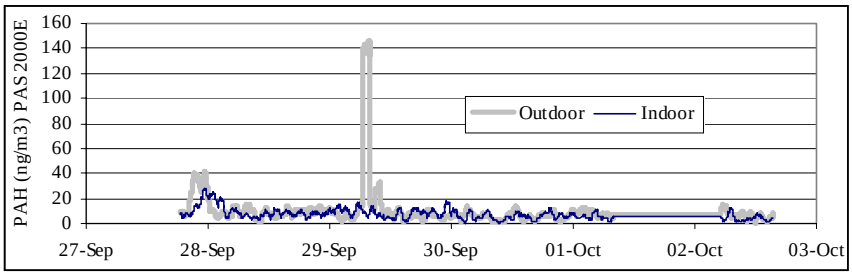

**Figure 3i: Real-time measures of PAH levels indoors and outdoors at the house of participant #12**

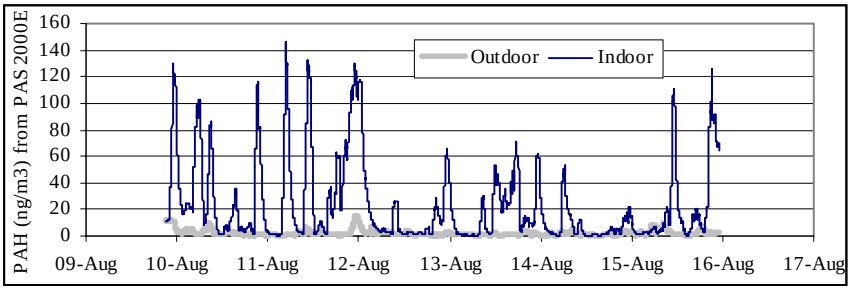


**Figure 3j: Real-time measures of PAH levels indoors and outdoors at the house of participant #13**

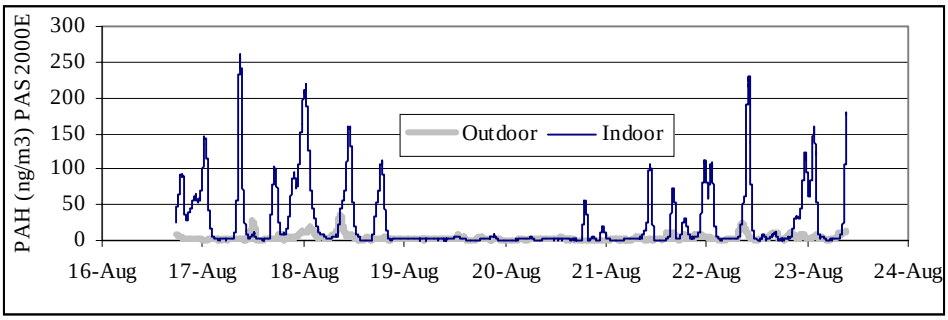


**Figure 3k: Real-time measures of PAH levels indoors and outdoors at the house of participant #14**

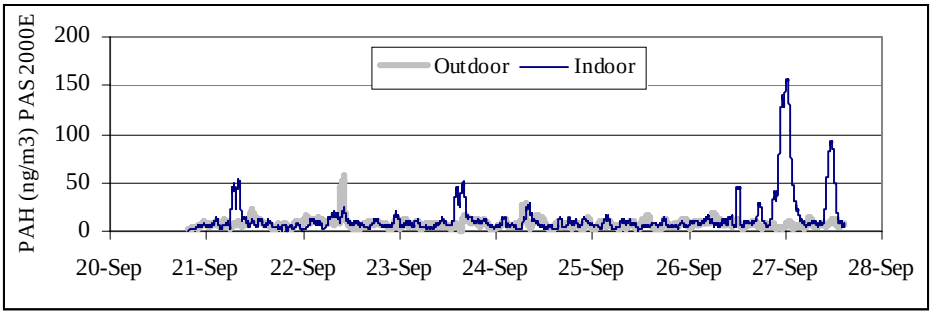

**Figure 4: Real-time measures of PAH levels outdoors compared to wind speed and direction**

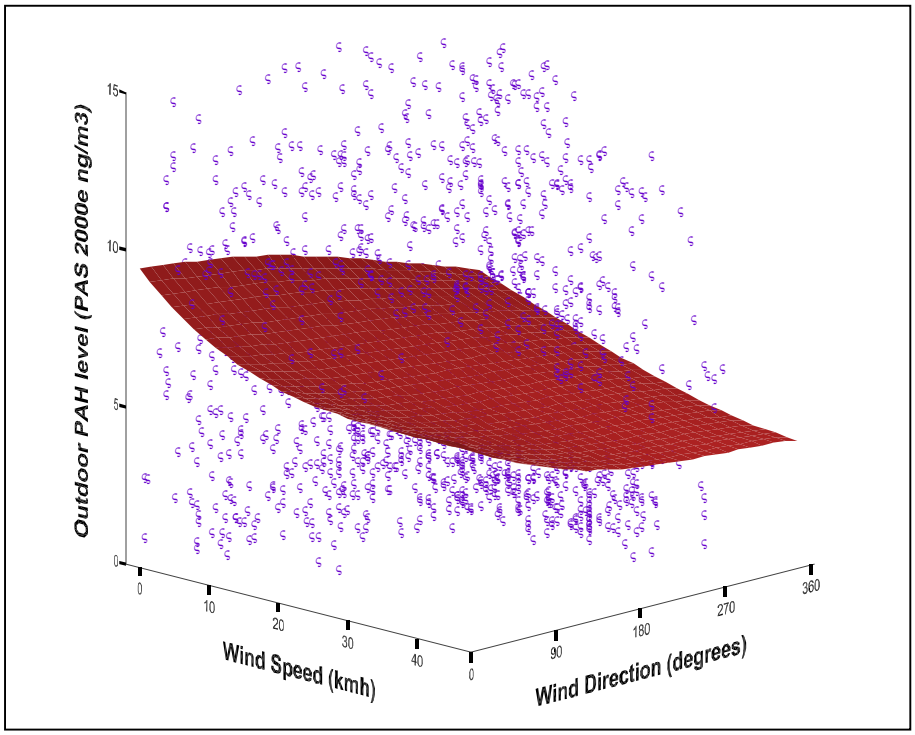

**Figure 5: Summary of PAH levels measured with personal integrated samplers**

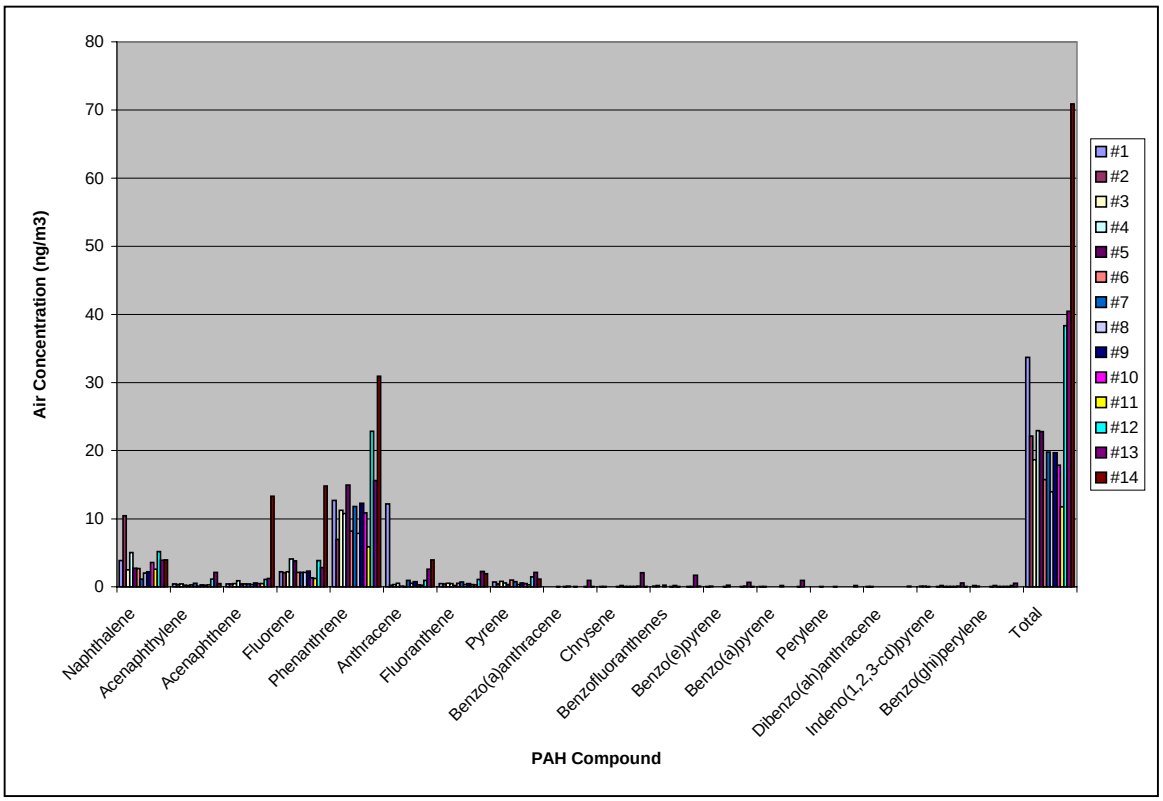


**Figure 6: Summary of PAH levels measured with indoor integrated samplers**

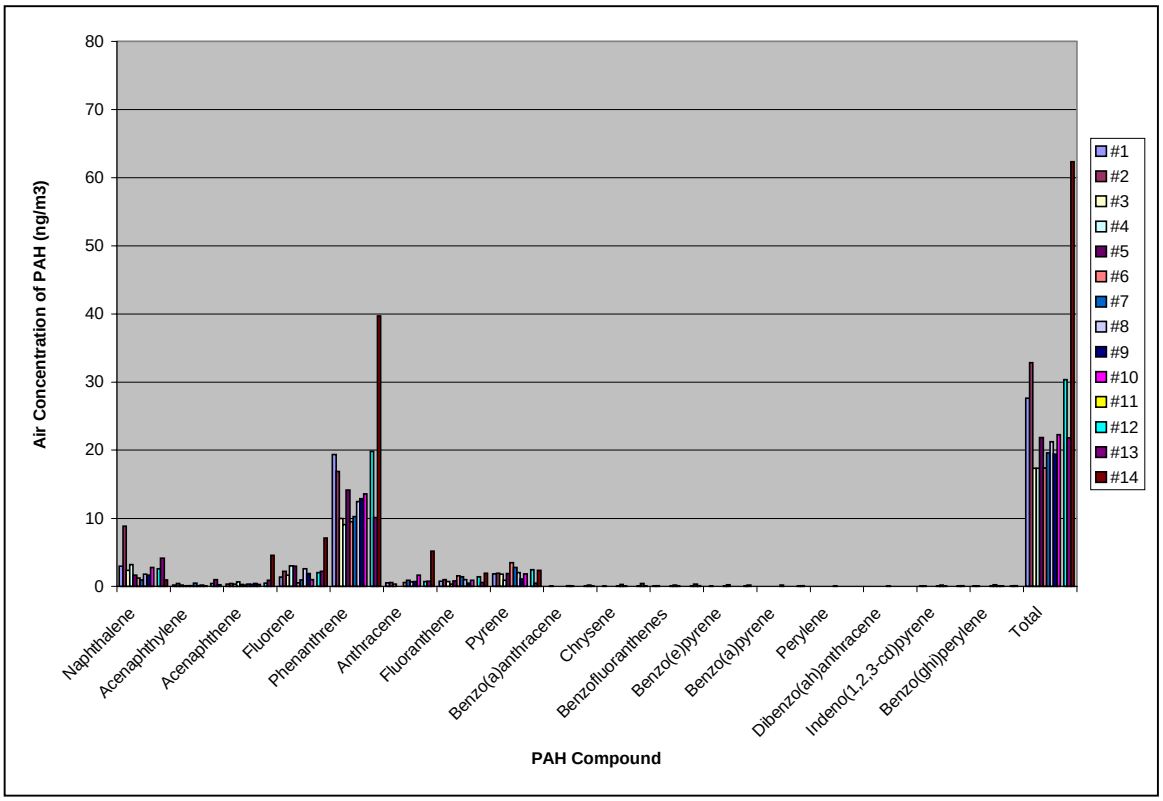

**Figure 7: Summary of PAH levels measured with outdoor integrated samplers**

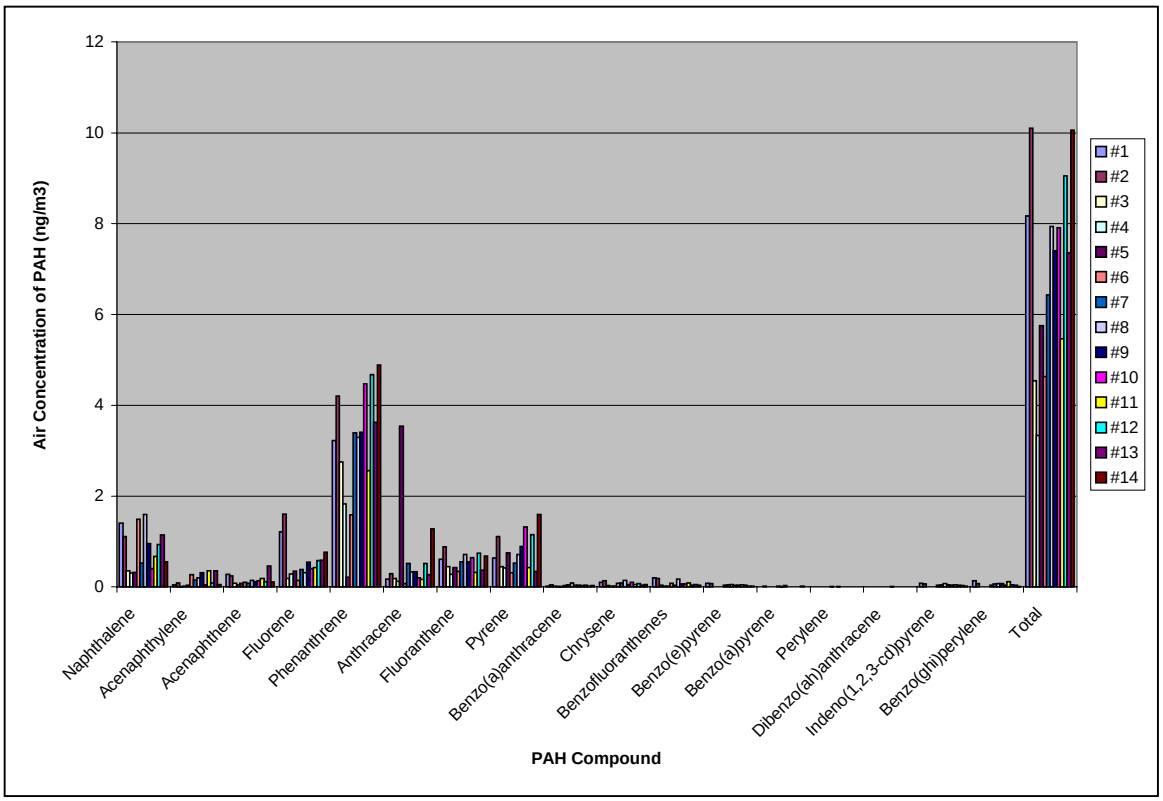


**Figure 8: Comparison of personal, indoor, and outdoor levels of the lightest PAHs measured**

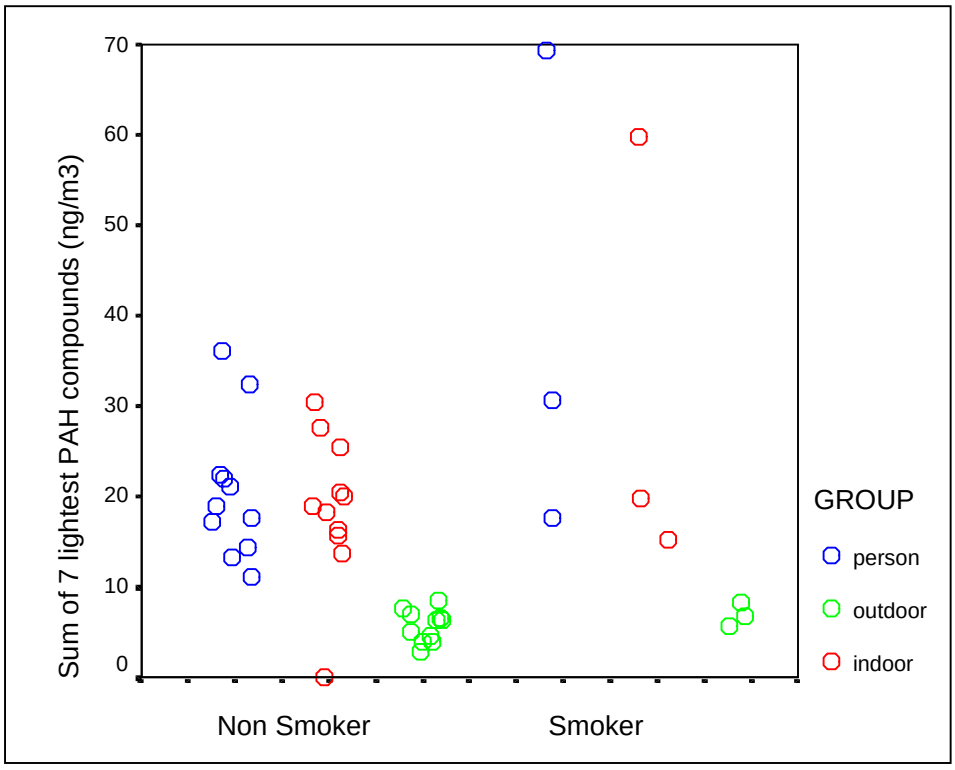

**Figure 9: Comparison of personal, indoor, and outdoor levels of the heaviest PAHs measured**

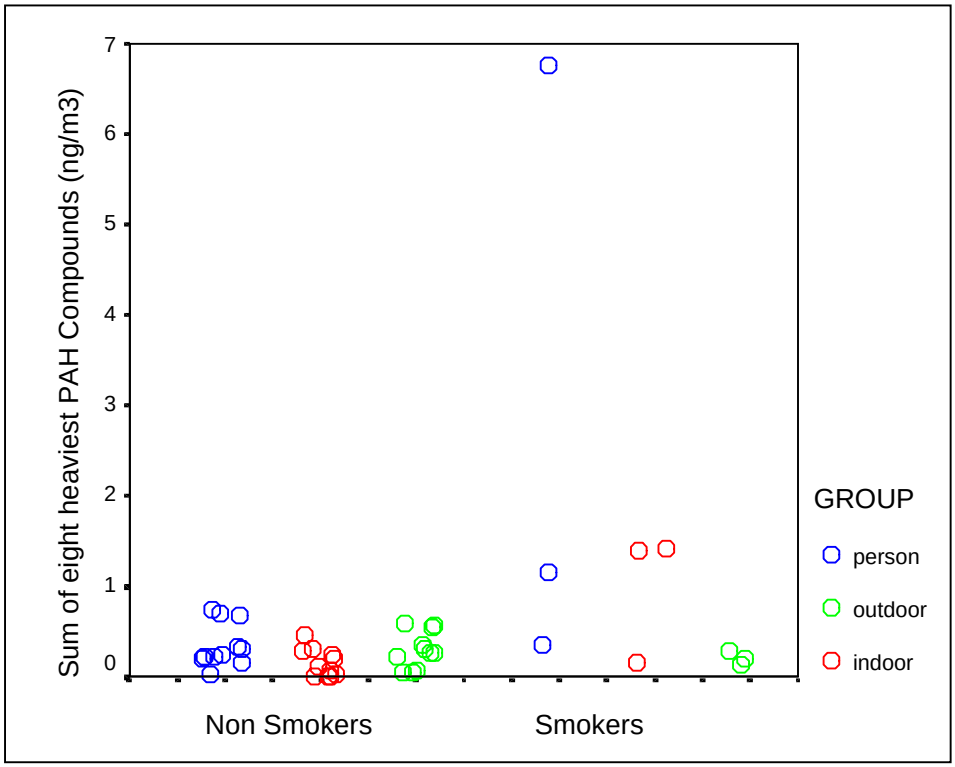


**Figure 10: Correlation of personal, indoor, and outdoor levels (non-smokers only)**

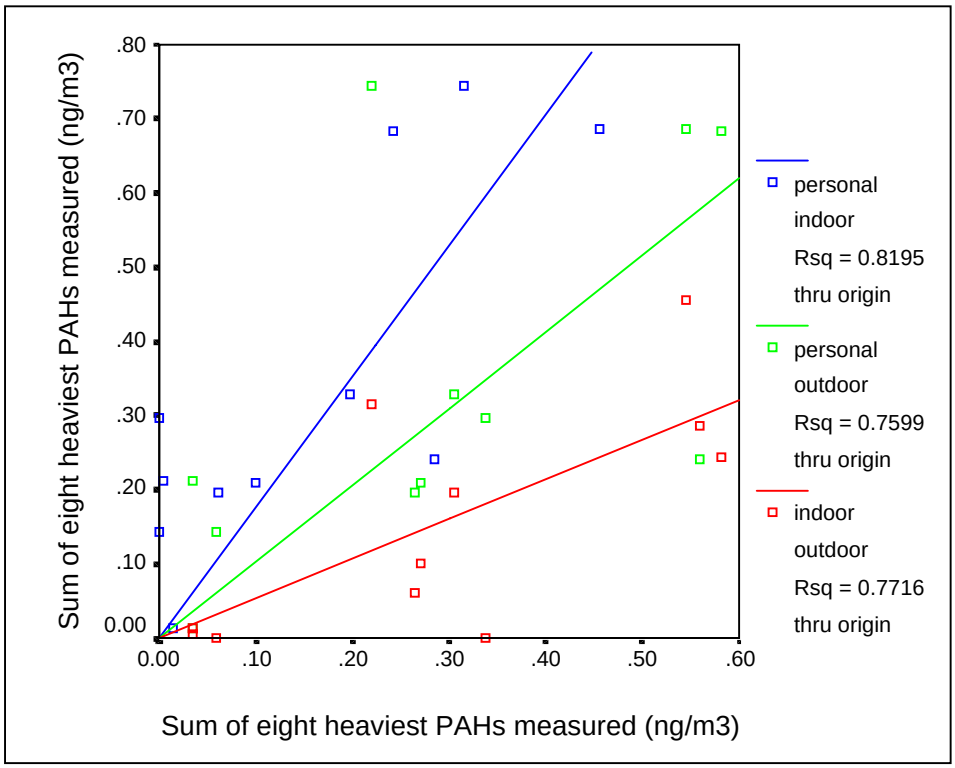

**Figure 11: Biplot showing distribution of PAH compounds at the sampling sites**

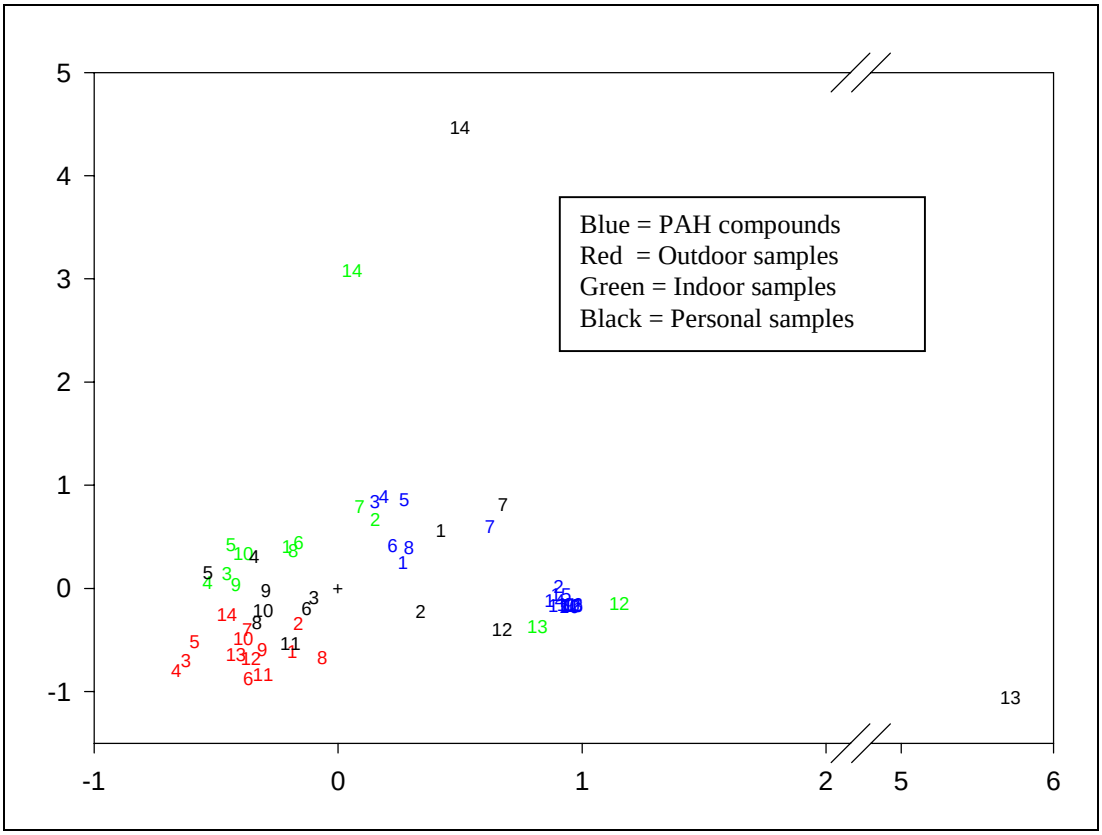


**Figure 12: Comparison of the two laboratories**

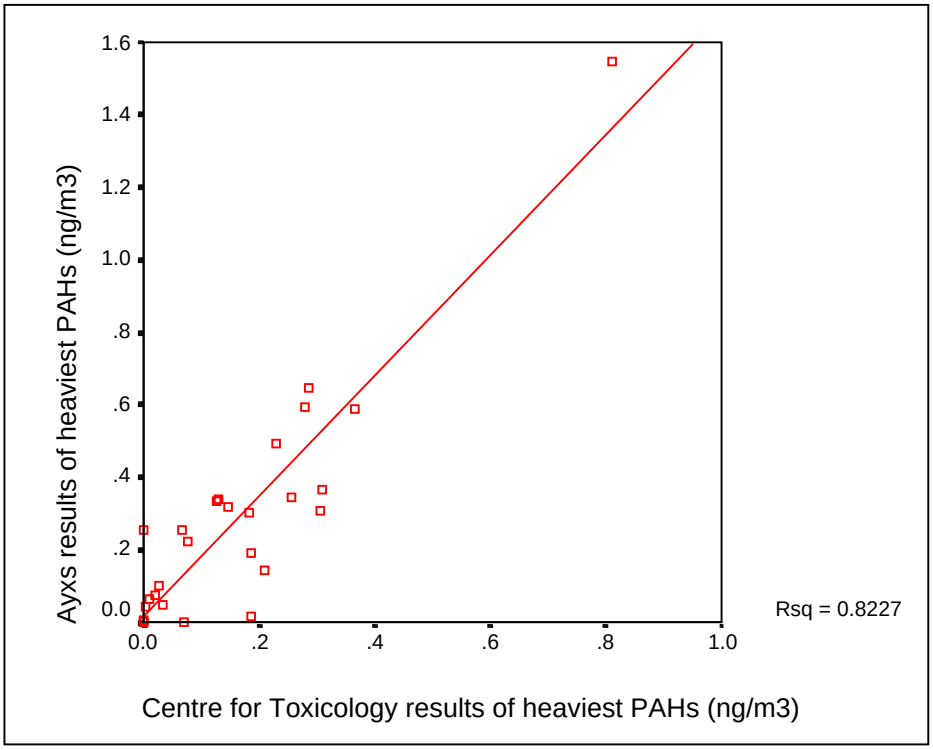

**Figure 13: Correlation coefficients comparing the PAS measures to individual PAH compounds**

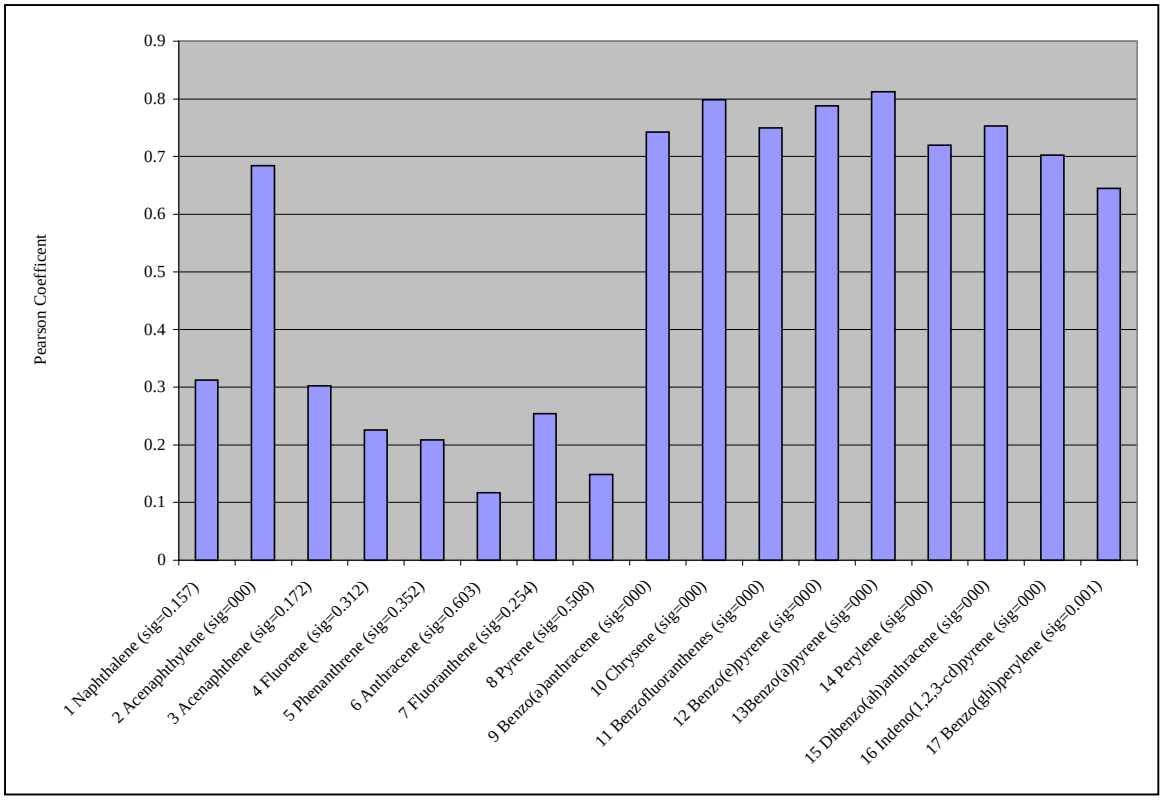


**Figure 14: Comparison of PAS 2000 CE and laboratory measures**

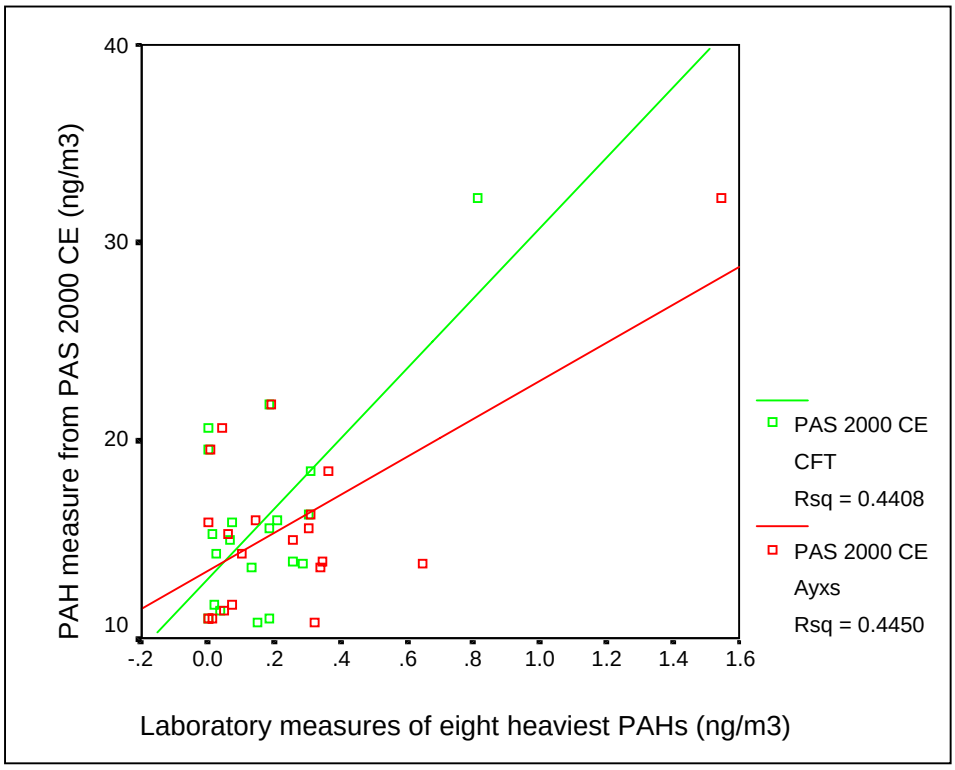

**Figure 15: Comparison of PAS 2000 CE and laboratory measures excluding highest point**

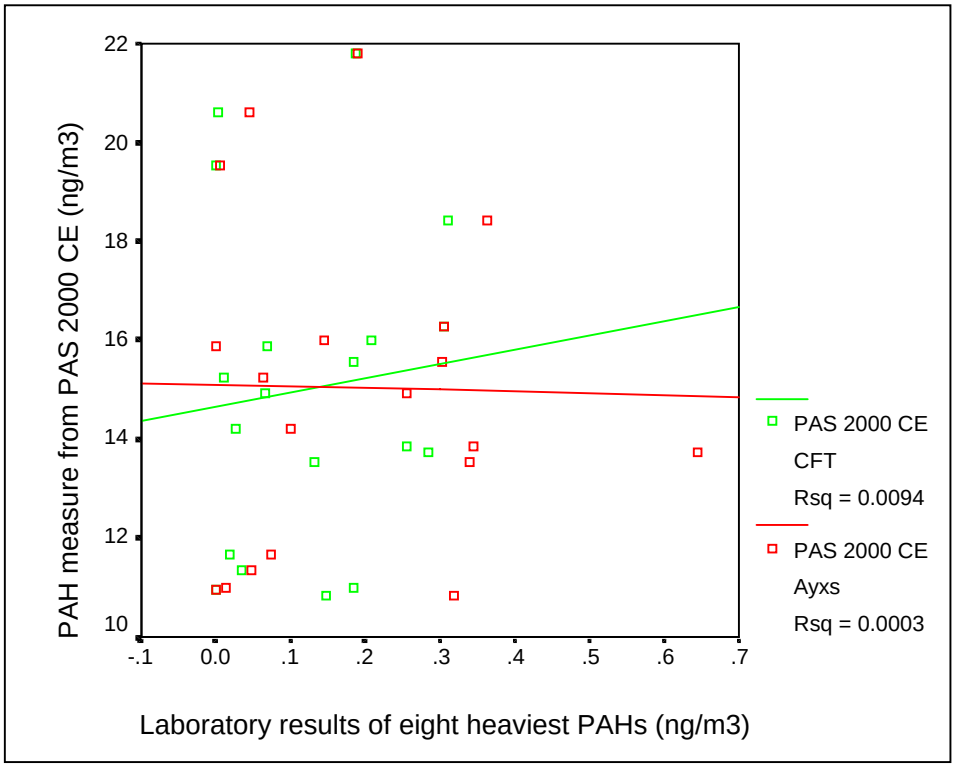


**Figure 16: Comparison of indoor / outdoor PAH ratios measured with real time and integrated methods**