Comparing Indoor Air Quality in Newer "Green" Low-Income Housing with Traditional Housing

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THESIS
Submitted in partial fulfillment of the requirements
for the degree of Master of Science in Public Health Sciences
in the Graduate College of the
University of Illinois at Chicago, 2011

Chicago, Illinois

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ACKNOWLEDGMENTS

This research and researcher were supported, in part, by the U.S. Department of Housing and Urban Development, Office of Healthy Homes and Lead Hazard Control, Grant # ILHH0191-08 and NIOSH Grant #T42/OH008672. We also wish to thank the residents who graciously permitted access to their homes to carry out the air sampling.

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LIST OF ABBREVIATIONS AND NOMENCLATURE

ACGIH	American Conference of Governmental Industrial Hygienists
AIHA	American Industrial Hygiene Association
ASHRAE	American Society of Heating, Refrigerating, and Air Conditioning Engineers
ATSDR	United States Agency for Toxic Substances and Disease Registry
BEI	Biological Exposure Indices
C	Ceiling Exposure Limit
CDC	United States Centers for Disease Control and Prevention
CO	Carbon monoxide
CO_2	Carbon dioxide
EPA	United States Environmental Protection Agency
GM	Geometric Mean
IARC	International Agency for Research on Cancer
LC_{50}	The lethal concentration that will kill 50 percent of a certain organism after an exposure at that concentration for a specific amount of time
LEED	Leadership in Energy and Environmental Design
MIGHHTY	Moving Into Green Healthy Homes: The Yield in health benefits study
MRL	Minimum Risk Level
ND1	New Development 1
ND2	New Development 2
OD	Old Development
PEM2.5	Personal Exposure Monitor pre-collector

LIST OF ABBREVIATIONS AND NOMENCLATURE (CONTINUED)

$PM_{2.5}$	Particulate matter less than 2.5 μm
ppm	Parts of contaminant of interests (by volume) per million parts of air (by volume)
PVC	Polyvinyl chloride
STEL	Short Term Exposure Limit
TLV	Threshold Limit Values
TWA	Time-weighted Average
USGBC	United States Green Building Council
VOC	Volatile Organic Compound
WHO	World Health Organization

SUMMARY

This study was conducted to determine if there are differences in air quality between older traditional low income housing and newer low income housing built with green design specifications. This research was conducted as a part of the larger Moving Into Green Healthy Homes: The Yield in health benefits (MIGHHTY) study. The MIGHHTY study is looking at the possible health benefits of moving into green housing through interviews with residents, visual inspections, and examination of Medicaid data related to health-care system usage, at three low-income housing developments. The study presented here examined the level of indoor air contaminants in a sample of non-smoking units in each of the three developments. Two developments were designed with green design specifications and built within the last 10 years and one was built traditionally over 30 years ago.

Participants were recruited into the larger MIGHHTY study through an initial survey process, in which a visual assessment of the home was taken, Medicaid data recorded, and a health interview completed using an adaptation of the Centers for Disease Control and Prevention (CDC) National Health Interview Survey. Survey responses revealed homes where cigarette smoke was not allowed, and these homes were then recruited to further participate in the air sampling portion of the study.

Once households agreed to participate in the air sampling, an investigator set up the air samplers in the home and returned approximately 24 hours later to collect the samplers. Samplers were typically located in the living room of the homes, though another location was occasionally chosen due to furniture arrangements and preferences of study participant.

Carbon monoxide (CO) and carbon dioxide (CO_2) were simultaneously measured using a data logging Q-Trak Indoor Air Quality Monitor made by TSI, Inc. then uploaded to a personal computer. Formaldehyde was measured using a UMEX-100 Passive Formaldehyde Sampler made by SKC, Inc. then sent to a laboratory for analysis. The laboratory is accredited by the American Industrial Hygiene Association (AIHA).

Particulate matter less than 2.5 μm ($PM_{2.5}$) was collected on pre-weighed polyvinyl chloride (PVC) filters in a SKC PEM2.5 pre-collector, using a pump that was pre- and post-calibrated with a bubble tube. The filters were sent to the laboratory, where they were post-weighed for analysis. Samples were blank corrected and concentrations were determined using the recorded volume of air sampled.

Whole air samples were collected in a vacuum-sealed canister with a flow regulator calibrated to draw air for 24 hours. These canisters were sent to an AIHA-accredited laboratory where the air sample was analyzed for 60 volatile organic compounds (VOCs) using Environmental Protection Agency (EPA) Compendium Method TO-15 (Center for Environmental Research Information 1999). To narrow down the results, a literature review was conducted using Toxicological Profiles from the Agency for Toxic Substances and Disease Registry (ATSDR), Documentation for Threshold Limit Values from the American Conference of Governmental Industrial Hygienists (ACGIH), the Monographs on the Evaluation of Carcinogenic Risks to Humans, and from other available literature.

The literature review identified individual VOCs commonly found in homes. Of these, VOC measurement results were further examined if at least 80% of the measurements for that contaminant were above the limit of detection. The VOCs chosen for study were , propene,

acrolein, acetone, methyl ethyl ketone, benzene, toluene, 1,4-dichlorobenzene, vinyl acetate, heptane, ethylbenzene, m/p-xylene, o-xylene, styrene, 1,2,4-trimethylbenzene and total VOCs.

The geometric mean (GM) concentration of CO_2 concentration in new development 1 (ND1) is significantly higher than the GM CO_2 concentration in the old development (OD). New development 2 (ND2) had a GM CO_2 concentration higher than OD but not significant at $p<0.05$. A higher level of CO_2 in the home is an indicator that less outdoor air is being introduced into the indoor air environment (Norbäck 1995). Significantly higher geometric mean or mean concentrations of acrolein, methyl ethyl ketone, benzene, toluene, heptane, and xylenes were measured in ND1 than in OD. Accordingly, total VOCs are higher in ND1 than OD. Motor vehicle exhaust is a source for these contaminants, it is possible that differing traffic levels on roads and highways near the developments could explain the differences seen in this study.

Three contaminants have been identified as above the Minimum Risk Level (MRL) and potential health risks in this study: $PM_{2.5}$, formaldehyde, and acrolein. These pollutants were of concern for all three developments and were similar to concentrations found in homes from other studies. While the higher CO_2 level of ND1 compared to OD may indicate a lower amount of fresh air entering the home from the outdoors, the health impacts of the air in the homes in the three developments appear to be similar.

1. Literature Review

1.1. Healthy Homes

The relationship between housing and public health has been the subject of study and policy for many years. Florence Nightingale reportedly stated "The connection between health and the dwellings of the population is one of the most important that exists (Lowry, 1989)."

The industrial revolution in the 1800s led to increased populations in cities, resulting in crowded, substandard housing and increasing the flow of municipal, industrial, and human waste that proved to be a large vector of disease. It was thought at the time that diseases were caused by miasma, the theory that bad smells carried disease. Although incorrect, this theory led to the sanitation movement and resulted in several interventions, slum clearance, housing ventilation and lighting, rubbish collection, and increased capabilities of waste containment and control, that did lead to a reduction in transmission of communicable disease in cities (Krieger 2002; Ormandy 1987).

The National Center for Healthy Housing (http://www.nchh.org/What-We-Do/Healthy-Homes-Principles.aspx) describes seven principles of healthy housing. Housing should be dry, clean, pest-free, safe, contaminant-free, ventilated, and maintained. Krieger and Higgins (2002) add neighborhood as an important determinant of health.

Dampness and dirtiness in the home is associated with several adverse health effects. Buildings that are not dry provide an environment that is conducive to growth of molds and bacteria (Andersson et al. 1997) as well as dust mites and other pests (Brunekreek et al. 1989). Presence of mold in homes is associated with increased asthma rates and increased respiratory

illness (Jedrychowski et al.2011, Perzanowski et al. 1998, Taskinen 2007) and decreased cognitive development in young children (Jedrychowski et al.2011). Children exposed to dust mite, mouse, dog, cat, and cockroach allergens are at in increased risk to develop asthma (Lin et al. 2011).

Safety of the home is also an important characteristic of healthy homes. From 1992 to 1998, there was an average of 18,048 unintentional injury deaths in the home per year (Runyan et al. 2005). Falls are the most common source of home injury, most frequently with the elderly but in other age groups as well (Kool et al. 2006, Alptekin et al. 2007, Runyan et al. 2005, Williams et al. 2011). Poisoning and fire/burn injuries are also important contributors to home injuries, while suffocation and drowning were high among young children (Runyan et al. 2005).

The neighborhood of a home can have impacts on the health of its residents. Elevated rates of intentional injury, certain communicable diseases, cardiovascular issues, birth issues, lack of exercise, and all-cause mortality have been assocaited with the neighborhood of a home (Krieger and Higgins 2002).

Proper ventilation of a building is important to protect health. A study of office buildings demonstrated that when ventilation rates were increased, the prevalence of sick building syndrome decreased (Seppänenn et al. 1999). Sick building syndrome is the occurrence of several symptoms including eye, nose and throat irritation, fatigue, or headache when in a specific building (Fisk et al. 2009). Most studies of sick building syndrome have been conducted in offices with mechanical ventilation systems that are different from those in homes. An appropriate ventilation system in the home is likely to be an effective way to improve health in the home, however, more research is needed in the subject (Sundel 2010).

Chemical exposures in homes can have impacts on health. Many chemical concentrations are higher in indoor air than outdoor air as both exposures from outdoors and exposures from products within the building contribute to the atmosphere (Edwards et al. 2001). The long-term exposure to chemicals in the home can result in a wide variety of health outcomes, both non-cancerous and cancerous in nature (Dawson and McAlary 2009).

The construction of environmentally friendly, or "green," housing is becoming more commonplace in the United States, with over one million homes in the country qualifying as ENERGY STAR homes (EnergyStar 2011) and over 9,500 homes certified by the more stringent Leadership in Energy and Environmental Design (LEED) certification standards (Watson 2010). Leadership in Energy and Environmental Design is a program of the U.S. Green Building Council (USGBC) and provides "a framework for identifying and implementing practical and measurable green building design, construction, operations and maintenance solutions. (http://www.usgbc.org/DisplayPage.aspx?CMSPageID=1988)." Leadership in Energy and Environmental Design certification rates building construction and operation in key areas including: sustainable sites, water efficiency, energy and atmosphere, materials and resources, indoor environmental quality, innovation and design, and regional priority (http://www.usgbc.org/DisplayPage.aspx?CMSPageID=1989). Enterprise Green Communities has eight families of criteria that need to be met in order to receive certification from their organization. These include: integrated design, locations and neighborhood fabric, site improvements, water conservation, energy efficiency, materials beneficial to the environment, healthy living environment, and operations and maintenance (http://www.greencommunitiesonline.org/tools/criteria/).

1.2. Indoor Air Quality

People spend large amounts of time in their homes, making the home an environment of particular health interest. Young people are at significant risk at home, since they are more susceptible to the possible effects of pollution. In addition, young people spend large quantities of time at home; infants average 19.3 hours per day in the home (Farrow 1997). Indoor air contaminants can cause a wide range of health outcomes including sore eyes, headaches, fatigue, worsening allergies and asthma, cancer, or even death from carbon monoxide asphyxiation (U.S. EPA 2010).

One focus of "green" homes is to create an energy efficient environment that reduces consumption of fossil fuels that contribute to global climate change and reduces costs of living. One method of reducing the energy impact of homes is to reduce leakage from outdoor air into the home to prevent the need to either heat or cool the outdoor air. The energy crisis in the 1970s resulted in changes to new construction and retrofitting of existing building to significantly reduce leakage. Many of these tight buildings had no mechanism for introducing outdoor air and others reduced the mechanical ventilation of outdoor air in order to reduce energy consumption causing increased prevalence of sick building syndrome (Letz 1990).

Several VOCs and aldehydes are associated with the symptoms of sick building syndrome (Takigawa et al. 2010). Poorly ventilated buildings have higher incidence rates of sick building syndrome, correlated with the concentrations of CO_2 in the rooms (ASHRAE 2010). Individual VOCs are duscussed in greater detail in the Air Contaminants section.

As outdoor pollution rates continue to decrease and outdoor-to-indoor air exchange rates are lowered, indoor sources of air pollutants are becoming a more important concern in pollutant

exposure (Logue 2010). Though the energy reduction capabilities of a new green housing are well documented, the effects of these homes on human health have not been well established. There is however, limited evidence that interventions in housing have been shown to improve health (Jacobs, et al. 2010, Krieger et al. 2010, Sandel et al. 2010, DiGuiseppi et al. 2010)

1.2.1. Volatile Organic Compounds (VOCs)

VOCs are a wide range of compounds that are emitted from liquids or solids that are easily evaporated. These compounds can be man-made, natural, or, in many cases, both. VOCs are associated with health effects ranging from being a mere annoyance to being carcinogenic or immediately dangerous to life and health, depending on the specific chemical and the exposure level. This study used the Environmental Protection Agency Compendium Method TO-15 (EPA 1999) to determine the concentrations of VOCs in homes. Though this technique will be discussed in greater detail in the methods section, it is important to note that for this study this technique provides data on 60 different volatile organic compounds. Table I organizes information on the sources of generation, established toxicity levels, and health effects for each contaminant. From this information, VOCs were selected for further analysis, based on the likelihood of the contaminant to be in a home, its health effects, and the results of the sampling in the homes.

The primary sources of information for this table come from the American Conference of Governmental Industrial Hygienists (ACGIH), the United States Agency for Toxic Substances and Disease Registry (ATSDR), and the International Agency for Research on Cancer (IARC).

ACGIH is a member-based organization focused on occupational health (acgih.org 2008). Each year the organization develops and publishes threshold limit value (TLVs) and biological

exposure indices (BEIs). Table I often cites TLVs in the toxicity levels section; TLVs are health-based guidelines referring to airborne concentrations of a pollutant at which most workers can repeatedly be exposed for a normal 8-hour shift, without experiencing health effects. TLVs with the notation "TWA" after it refer to "time-weighted averages." In this case, time weighted averages are average concentrations that should not be exceeded during an 8 hour work-day in a conventional 40 hour work week. TLVs with the notation "STEL" after it refer to "short term exposure limits." Short term exposure limits are average concentrations that occur in a period of 15 minutes. TLVs with the notation "C" after it refer to a "ceiling." Ceilings are concentrations that should never be exceeded in a workday at any point. The TLVs reported in the "Toxicity Levels" column of Table I come from the 2010 edition of "TLVs and BEIs: Based on the Documentation of the Threshold Limit Values for Chemical and Physical Agents and Biological Exposure Indices." ACGIH also publishes documentation outlining the research and explaining the reasoning behind the threshold limit values. Information for Table I about sources and health effects comes from the 1991 edition of this book.

TLVs are typically not appropriate guidelines for indoor air quality in the home. These guidelines are intended to be used under the specific circumstances of a healthy worker working in the time parameters established by the TWA or STEL notation. These are not appropriate for the home because a very different exposure occurs in the home. The home does not exclusively feature healthy workers; occupants of homes include every possible demographic, including those susceptible to disease such as infants/ children, the immunologically compromised, those with other health conditions or respiratory issues, and the elderly. The length of exposure in the home greatly differs from the time spent in the workplace. Large amounts of time can be spent in the home, with some of the most vulnerable, infants and the elderly, spending the most time in

the house (Farrow 1997). Threshold limit values do, however, provide insight on the toxicity of different chemicals that are found in the home. Though the exact concentration of a TLV may not be of particular utility in indoor air, noting that one contaminant has a TLV-TWA of 1 ppm and another contaminant has a TLV-TWA of 1000 ppm reveals that, based on current research, the first contaminant is much more toxic than the second one.

ATSDR is a United States governmental body established to confront the public health issues associated with hazardous substances in the environment, specifically at waste sites (atsdr.cdc.gov 2011). "ATSDR is charged under the Superfund Act to assess the presence and nature of health hazards at specific Superfund sites, to help prevent or reduce further exposure and the illnesses that result from such exposures, and to expand the knowledge base about health effects from exposure to hazardous substances (http://www.atsdr.cdc.gov/about/congress.html)." Among other duties, one responsibility of the ATSDR is to publish toxicological profiles on specific substances. Toxicological profiles provide detailed information about a substance such as its sources, its concentrations and doses that cause health effects, its chemical and physical properties, its health effects through different routes of exposure, and its potential to compromise public health. Table I frequently refers to a LC_{50} of a contaminant in the "Toxicity Levels" column. An LC_{50} is the lethal concentration that will kill 50 percent of a certain organism after an exposure at that concentration for a specific amount of time. The LC_{50} is of similar usefulness to the TLV in that it can provide a good comparison of the toxicities of different substances.

The ATSDR also publishes minimal risk levels, or MRLs, for certain substances. An MRL is a concentration of a pollutant at which there is no appreciable risk of non-cancerous health effects resulting (ATSDR 2010). The ATSDR sets MRLs for acute exposure, between 1 and 14 days; intermediate exposure, between 14 and 365 days; and chronic exposure, a year or

longer. Minimum Risk Levels use human data when available and animal data when needed. As toxicological data will not account for the most at-risk populations, MRLs are significantly lower than the lowest concentration seen to have health effects to ensure that it is protective of all populations. Minimum Risk Levels are useful when examining pollutants of the home, because they are intended to be protective for all people and assume constant exposure. Since it is unlikely that many people will spend all of their time in the home, the home occupants will not be truly experiencing constant exposure. Assuming constant exposure, however, errs on the side of caution, making MRLs a very conservative and protective figure to reference.

The Internaitonal Association for Research on Cancer is an international, interdisciplinary group dedicated to researching causes and prevention of cancer. The Internaitonal Association for Research on Cancer regularly develops monographs that summarize all the most current data and information about potential carcinogens. These monographs categorize agents into different groups based on the available evidence of their carcinogenicity, as seen in the "Health Effects" column of Table I. The categories are explained as follows (monographs.iarc.fr 2011): Group 1- Agent is carcinogenic to humans- there is sufficient evidence of carcinogenicity in humans or there is less than sufficient evidence in humans with sufficient animal evidence and strong evidence of a cancerous mechanism in humans; Group 2A- Agent is probably carcinogenic to humans- there is limited evidence of carcinogenicity in humans but there is sufficient evidence that the substance is carcinogenic to animals; Group 2B- Agent is possibly carcinogenic to humans- there is limited evidence of carcinogenicity in humans and less than sufficient evidence that the substance is cancerous to animals, or there is inadequate evidence of carcinogenicity in humans but there is sufficient evidence that the substance is cancerous to animals; Group 3- The agent is not classifiable as to its carcinogenicity to humans-

evidence of carcinogenicity in humans is inadequate and evidence of carcinogenicity in animals is inadequate or limited; Group 4- The agent is probably not carcinogenic to humans- there is evidence suggesting a lack of carcinogenicity in humans and animals.

These categorizations are useful because the carcinogenicity of a substance is of great importance when determining the potential danger of a contaminant. Keeping long-term exposures to potential carcinogens to a minimum, even at low concentrations, is an important goal for public health professionals.

Of the sixty chemicals included in the TO-15 analysis, the following chemicals were selected for consideration in this study. The determination was based on the likelihood of the contaminant to be found in the home as reported in the literature and shown in Table I, and at least 80% of the measurements for that chemical exceeding the limit of detection.

1.2.1.1. Propene (Analyte #2)

Propene is a colorless gas used in gasoline as an octane improvement agent. It is also a byproduct of combustion of organic materials (Canada OEHHA 1999).

Propene is an upper respiratory tract irritant (ACGIH 1991). Chronic exposures in rats resulted in damage to the nasal cavity (Canada OEHHA 1999). Propene is a group 3 carcinogen, not classifiable as a human carcinogen. This is because there is insufficient evidence of carcinogenicity in both humans and animals (IARC 1998). The TLV-TWA for propene is 500 ppm (ACGIH 2010).

1.2.1.2. Acrolein (Analyte #10)

Acrolein is a colorless or yellowish liquid with a choking odor, and is used as an intermediate in the manufacture of polyurethane resins, polyester resins, pharmaceuticals, and herbicides (ACGIH 1991). Acrolein can be produced from the incomplete combustion of organic materials, indoor sources include heated cooking oil, cigarette smoke, burning incense, and wood burning fireplaces (Seaman 2009).

Single and repeated exposure of 3-4 ppm of acrolein to monkeys and rats resulted in death before the 10^{th} day of exposure (ATSDR 2007). Acrolein exposure has been reported to cause intense eye and upper respiratory tract irritation, possibly causing pulmonary edema and tracheobronchitis (ACGIH 1991). Exacerbation of asthma in children has resulted from acrolein exposure (Leikauf, 2002). The irritation threshold of this contaminant has been reported to be 0.25 ppm (ACGIH 1991). It is designated as a group 3 carcinogen because there is inadequate evidence of carcinogenicity in both humans and animals (IARC 1995). The TLV-Ceiling for acrolein is 0.1 ppm (ACGIH 2010). The acute MRL is 0.003 ppm and the intermediate is 0.00004 ppm (ATSDR 2010).

Winter acrolein concentrations in non-smoking homes in Saskatchewan, Canada had a geometric mean of 0.23 ppb while summer concentrations had a geometric mean of 0.44 ppb (Heroux 2010). In 4 studies of homes in industrialized countries, mean acrolein levels were 1.0 ppb (Logue 2010).

The studies show that the concentrations of acrolein that are typical in urban homes are above the chronic MRL. This indicates that there is a possibility of public health impacts from acrolein in homes.

1.2.1.3. Acetone (Analyte #11)

Acetone is a colorless liquid with a unique taste and smell. It is used as a solvent, chemical intermediate, and cleaner (ATSDR 1994). It is also used as an ingredient in some paints, a potential indoor air source (ACGIH 1991).

Acetone is a respiratory system irritant, with complaints about nose, trachea, throat and lung irritation at exposures greater than 901 ppm (ATSDR 1994). Central nervous system effects of acetone exposure include headache and lightheadedness at exposures of 1006 ppm for a day, workers that were exposed to 12,000 ppm or greater of acetone experienced unconsciousness, dizziness, confusion, and headache (ATSDR 1994). The TLV-TWA for acetone is 500 ppm and the TLV-STEL is 750 ppm (ACGIH 2010). The acute MRL is 26 ppm, the intermediate MRL is 13 ppm, and the chronic MRL is 13 ppm (ATSDR 2010).

In 6 studies of homes in industrialized countries, mean acetone levels were 16.8 ppb (Logue 2010). Acetone levels in homes are typically well below the chronic MRL set by ATSDR. This could indicate that the potential for health impacts from acetone would be minimal in the homes being examined in this study. Its prevalence in many substances that are frequently found in homes indicates that it could provide insight on VOCs in the home.

1.2.1.4. Vinyl Acetate (Analyte #20)

Vinyl acetate is a colorless liquid with a sweet odor. It is used to produce polyvinyl acetate emulsions and polyvinyl alcohol. Polyvinyl emulsions are used in adhesives, paints, textiles, and paper products, all of which can be found inside the home environment (ACGIH 1991).

Vinyl acetate exposure causes irritation or the upper respiratory tract, and it has damaged the respiratory tract in lab animals (ATSDR 1992). Vinyl acetate vapors have been reported to be an eye irritant at 21.6 ppm (ACGIH 1991). Vinyl acetate is a group 2B carcinogen, possibly carcinogenic to humans. It is listed this way because there is inadequate human evidence of human carcinogenicity and there is limited evidence of cancer in animals (IARC 1995). The TLV-TWA for vinyl acetate is 10 ppm and the TLV-STEL is 15 ppm (ACGIH 2010). The intermediate MRL is 0.01 ppm (ATSDR 2010).

1.2.1.5. Methyl Ethyl Ketone (Analyte #21)

Methyl Ethyl Ketone is a colorless, flammable liquid that smells similar to acetone (ACGIH 1991). It is used in paints, glues, finishes and a solvent. It is also found in motor vehicle exhaust (ATSDR 1992).

A four-hour exposure of 11,700 ppm to methyl ethyl ketone in rats caused death in half the rats (ATSDR 1992). Workers exposed to 100 ppm reported slight nose and throat irritation at 100 ppm, mild eye irritation at 200 ppm, and found these symptoms to be offensive at 300 ppm (ACGIH 1991). A group of cable factory workers exposed to concentrations varying from 50-117 ppm of methyl ethyl ketone had an increased prevalence of upper respiratory tract irritation and gastrointestinal symptoms compared to a control group (ATSDR 2010). Case studies have demonstrated chronic exposures to methyl ethyl ketone led to neurological effects including dizziness, anorexia, and body weakness (ATSDR 2010). The TLV-TWA for methyl ethyl ketone is 200 ppm and the TWA-STEL is 300 ppm (ACGIH 2010).

In a review of 5 studies of homes in industrialized countries, an average methyl ethyl ketone concentration was reported to be 6.68 ppb (Logue 2010).

The lack of an appropriate guideline (MRL) for methyl ethyl ketone makes it difficult to assess its potential for health outcomes in home exposure. Literature on the levels of the contaminant suggest that typical home concentrations (Logue et al. 2010) are far below the TLV, but there is need for further study in realm of green housing.

1.2.1.6. Benzene (Analyte #30)

Benzene is a colorless, flammable, non-polar liquid that is used in the production of styrene, cyclohexane, and other organic compounds (ACGIH 1991). Benzene is also emitted in the burning of coal and oil (ATSDR 2007). Motor vehicle exhaust is a major source of benzene pollution, particularly in high traffic urban areas (Massolo, et al. 2010). Other indoor sources can include building materials, furniture, attached garages, cooking systems, and heating and cooking systems (WHO 2010).

It is estimated that 5-10 minutes exposure of 20,000 ppm of benzene is usually fatal (ATSDR 2007). Fifty percent of rats die after a 13,700 ppm exposure to benzene for 4 hours (ATSDR 2007). Benzene exposures of 50-100 ppm for 30 minutes can cause fatigue and headaches while 30-minute exposures to 250-300 ppm can cause dizziness, faintness, and nausea (WHO 2010). Extended exposure to benzene can damage the hematopoietic system, including aplastic anemia, lymphopenia, leukemia, thrombocytopenia, and pancytopenia (WHO 2010). Chronic benzene exposures are associated with respiratory symptoms and diseases such as upper respiratory tract irritation, laryngitis, and bronchitis (ATSDR 2007). Immunological effects such as decreased white blood cell counts and changes in bone marrow activity are associated with exposure (WHO 2010). Muscle pain, eye irritation, skin irritation, decreased fertility, and central nervous system damage have also been associated with chronic benzene exposure (ATSDR

2007). A study of pregnant Spanish women showed no relationship between home benzene concentration and birth outcomes including birth head circumference, length, and weight (Estarlich 2011). Benzene is classified as a group 1 carcinogen. It is classified this way because there is sufficient evidence that chronic exposure to benzene results in an increased risk of developing leukemia (IARC 1999). The TLV-TWA for benzene is 0.5 ppm with a TLV-STEL of 2.5 ppm (ACGIH 2010). The acute MRL is 0.009 ppm, the intermediate MRL is 0.006 ppm, and the chronic MRL is 0.003 ppm (ATSDR 2010).

Indoor concentrations of benzene in Argentinean urban areas had a mean of 1.12 ppb (Massolo, et al. 2010). Winter benzene concentrations in non-smoking homes in Saskatchewan, Canada had a geometric mean of 0.41 ppb while summer concentrations had a geometric mean of 0.37 ppb (Heroux 2010). A study of homes in Spanish urban areas demonstrated a mean concentration of 0.5 ppb (Estarlich 2011). A United Kingdom study of non-smokers showed a mean benzene concentration in the home of 0.62 ppb (Delgado-Saborit 2010). In 16 studies of homes in industrialized countries, mean benzene levels were 0.78 ppb (Logue 2010).

Since benzene is a carcinogen, it warrants study for its concentrations in homes. Studies have found that benzene concentrations in homes are below the chronic MRL, although they are still only one order of magnitude lower. There could be potential for adverse health outcomes from benzene exposures in the homes.

1.2.1.7. Heptane (Analyte #37)

Heptane (C_7H_{16}) is a VOC with nine isomers. It is used commonly in gasoline, rubber solvent, and other petroleum solvents (ACGIH 1991). Indoor sources of heptane contamination can come from numerous sources including: combustion products, cooking, furnishings, paints, varnishes and solvents, adhesives and caulks, office equipment and consumer products (Massolo,

et al. 2010). Exposure to mice from 10,000 to 15,000 ppm resulted in narcosis within 30 to 50 minutes and exposures 15,000 to 20,000 ppm caused convulsions and death within 30-60 minutes. Human exposure to concentrations of 1000-5000 ppm of heptane can cause health effects including dizziness, nausea, or loss of appetite. Chronic nervous system effects have not been solely from heptane exposure, but studies of rats have found neurotoxic effects from heptane exposure. Another study of workers exposed to a solvent of 80% pentane, 14% heptane, and 5% hexane experienced polyneuropathy, concluding that all of these ingredients may be neurotoxins (ACGIH 1991). Based on these findings, the ACGIH-TLV for heptane is TWA-400 ppm (ACGIH 2010).

Indoor concentrations of heptane were sampled in the La Plata region of Argentina, and mean indoor rural concentrations were 0.64 ppb while mean indoor concentrations in an urban area 0.78 ppb (Massolo 2010). Another study of Hong Kong homes found mean concentrations of heptane to be 5.41 ppb (Guo 2009). In 5 studies of homes in industrialized countries, mean heptane levels were 2.68 ppb (Logue 2010).

Heptane lacks appropriate guidelines with which to compare home concentrations. Studies found concentrations in homes were well below the TLV, though its potential to harm health is not ruled out due to this.

1.2.1.8. Toluene (Analyte #43)

Toluene is a colorless liquid with a smell that is typical of hydrocarbons. It is used as a high octane blending stock for gasoline, a solvent, a chemical intermediate, in household aerosols, paints, paint thinners, varnishes, rust inhibitors, solvent based cleaners, and nail polish (ACGIH 1991). In addition to the numerous household products that contain toluene, toluene

can also enter the home as a constituent of motor vehicle exhaust, especially in high traffic urban areas (Massolo, et al. 2010).

Mice have a 7-hour LC_{50} for toluene at 5320 ppm (ATSDR 2000). Irritation of the respiratory tract and decreased lung function have been noted after toluene exposure (ATSDR 2000). Depression of the central nervous system with symptoms such as headache, impaired coordination, depression, and impaired reaction time (ACGIH 1991). Toluene is an irritant of the eye and it can cause deficits in color vision, with workers exposed to 100 ppm or greater experiencing color confusion (ATSDR 2000). Changes in sperm count and ovarian structure have occurred after exposure, with reports of increased spontaneous abortion levels in exposed pregnant women (ATSDR 2000). Toluene is a group 3 carcinogen, not classifiable as to its carcinogenicity to humans. It is listed this way because there is inadequate evidence that it causes cancer in humans and there is evidence that it does not cause cancer in animals (IARC 1999). It has a TLV-TWA of 20 ppm (ACGIH 2010). Its acute MRL is 1 ppm and its chronic MRL is 0.08 ppm (ATSDR 2010).

A study of 3 year old homes in Tokyo found a mean toluene concentration of 2.9 ppb, while in older homes it found a mean of 2.4 ppb (Park 2006). A study of indoor air in urban areas of Argentina noted a mean concentration of 4.0 ppb (Massolo, et al. 2010). A study of non-smoking homes in Saskatchewan, Canada demonstrated geometric means of 3.0 ppb in the summer and 2.1 ppb in the winter (Heroux 2010). A study of Hong Kong homes found a mean concentration of 4.1 ppb (Guo 2009). A United Kingdom study of non-smokers found a mean concentration of 4.7 ppb (Delgado-Saborit 2011). In 17 studies of homes in industrialized countries, mean toluene levels were 3.98 ppb (Logue 2010).

Studies indicate that toluene in typical urban homes is less that the chronic MRL by one order of magnitude. Its ubiquity in products that can be in the home warrant its investigation in this study.

1.2.1.9. Ethyl benzene (Analyte #49)

Ethyl benzene is a colorless liquid with an aromatic odor (ACGIH 1991). It is largely used as an intermediate in the production of styrene, but it is also found in consumer products including: gasoline, carpet glues, paints, inks, varnishes, automotive products, and tobacco products (ATSDR 2010). Many of these consumer products that feature ethyl benzene are found in the home and can cause exposure there. Motor vehicle exhaust can be an important source of ethyl benzene in the home, particularly in high-traffic urban areas (Massolo, et al. 2010).

A 4-hour exposure of 4000 ppm of ethyl benzene to rats resulted in the death of 50 percent of the rats (ACGIH 1991). A 6-minute, 2000 ppm exposure in men resulted in reports of upper respiratory irritation and chest constriction (ATSDR 2010). Workers chronically exposed to ethyl benzene experienced decreased lymphocyte values, though these workers were also exposed to several other pollutants while working (ATSDR 2010). Central nervous system effects experienced by exposed workers have included headaches, irritability, and fatigue (ACGIH 1991). Kidney damage in rats has been noted from inhaled ethyl benzene exposure (ATSDR 2010). Ethyl benzene is listed as a group 2B carcinogen, possibly carcinogenic to humans. Though there is inadequate evidence for human carcinogenicity, there is sufficient evidence of carcinogenicity in the lungs and kidneys of rats and mice (IARC 2000). The TLV-TWA is 100 ppm with a notice of intended change to 20 ppm for next year's edition (ACGIH

2010). The acute MRL is 5 ppm, the intermediate MRL is 2 ppm, and the chronic MRL is 0.06 ppm (ATSDR 2010).

A study in Tokyo of 3 year old homes showed a 2.1 ppb mean concentration for ethyl benzene, while older homes in the same study showed a mean of 1.38 ppb (Park 2006). An Argentinean study found urban indoor air samples had a mean value of 0.31 ppb (Massolo, et al. 2010). A study of homes in Hong Kong showed a mean ethyl benzene concentration of 0.94 ppb (Guo 2009). A study of non-smokers from the United Kingdom showed a mean concentration of 0.40 ppb in their homes (Delgado-Saborit 2010). In 15 studies of homes in industrialized countries, mean ethyl benzene levels were 0.90 ppb (Logue 2010).

Reported ethyl benzene concentrations in homes are below the MRL. This pollutant, however, is in many household products and building materials and could reveal differences in air quality between new green housing and old traditional housing.

1.2.1.10. M/P-Xylene (Analyte #50)

Xylenes are clear, flammable liquids with an odor typical of aromatic hydrocarbons. Mixed xylene is in gasoline and aviation fuel, and a solvent in paints. M-xylene is used as an intermediate in isophthalic acid production and p-xylene is used as an intermediate in the production of pharmaceuticals, insecticides, and terephthalic acid. P-xylene is the most frequently used of the three xylene isomers as it is used in the production of polyester (ACGIH 1991). Xylenes are also found in the indoor environment as a part of exhaust from motor vehicles, which is more prominent in urban areas with high traffic (Massolo, et al. 2010).

Rats have a 4-hour LC_{50} for mixed xylene of 6350 ppm, a 6-hour LC_{50} for m-xylene of 5267 ppm, and a 6-hour LC_{50} for p-xylene of 3907 ppm (ACGIH 1991). Exposure to mixed xylene, m-xylene, and p-xylene has caused respiratory tract irritation, with chronic exposure causing difficulty breathing and decreased pulmonary function (ATSDR 2007). Nausea, vomiting, and gastric discomfort have been noted in workers exposed to xylenes (ATSDR 2007). Xylene vapor can be irritating to the eyes (ACGIH 1991). Exposures to mixed xylene and m-xylene caused neurological effects including slower reaction time, impaired short-term memory, and lack of balance. Exposure to p-xylene did not show these effects (ATSDR 2007). Long-term mixed xylene exposures demonstrated impairments to the central nervous system including anxiety, forgetfulness, and inability to concentrate (ATSDR 2007). Xylenes are listed as a group 3 carcinogen, unclassifiable as to their carcinogenicity to humans due to inadequate evidence of carcinogenicity in both animals and humans (IARC 1999). Mixed xylene and all of its isomers have a TLV-TWA of 100 ppm and a TLV-STEL of 150 ppm (ACGIH 2010). The acute MRL for xylenes is 2 ppm, the intermediate MRL is 0.6 ppm, and the chronic MRL is 0.05 ppm (ATSDR 2010).

A study in Tokyo of 3 year old homes, showed a 3.2 ppb mean concentration for p-xylene, while older homes in the same study also showed a mean of 3.2 ppb (Park 2006). An Argentinean study showed urban indoor air to have a mean m/p-xylene concentration of 1.5 ppb (Massolo, et al. 2010). A study of Hong Kong homes showed a mean p-xylene concentration of 0.69 ppb (Guo 2009). A study of UK non-smokers had a home p-xylene concentration of 0.39 ppb and a m-xylene concentration of 0.95 ppb (Delgado-Saborit 2011). In 15 studies of homes in industrialized countries, mean m/p-xylene levels were 1.9 ppb (Logue 2010).

Xylene isomers were found to be lower than health guidelines in urban homes, though the possibility of xylenes to be introduced to the home environment is high due to its high level of use in products.

1.2.1.11. Styrene (Analyte #52)

Styrene is a colorless to yellow oily liquid with a sweet smell. It is used in the production of plastic and rubber, in foam packaging, pipes, flooring, insulation, disposable cups, plates and bowls, and it is found in motor vehicle exhaust (ACGIH 1991).

Styrene has a 4-hour LC_{50} of 4940 ppm in rats (ATSDR 2010). Styrene is a throat and nose irritant, with throat irritation noted as taking place after exposure for 4 hours to 800 ppm (ATSDR 2010). Its vapors also can be irritating to the eye (ACGIH 1991). Nausea has occurred in humans exposed to styrene (ATSDR 2010). Liver dysfunction has been reported in several studies after prolonged styrene exposure (ATSDR 2010). The nervous system is most severely impacted by styrene, with workers exposed feeling inebriated after exposure. Also, an international cohort found that exposure to styrene increases the risk of mortality from a disease to the central nervous system (ATSDR 2010). Styrene is a group 2B carcinogen, possibly carcinogenic to humans. It has limited evidence of carcinogenicity in both humans and animals (IARC 2002). The TLV-TWA for styrene is 20 ppm and the TLV-STEL is 40 ppm (ACGIH 2010). The acute MRL is 5 ppm and the chronic MRL is 0.2 ppm.

A study in Tokyo revealed a mean concentration of 1.4 ppb in 3 year old homes and a mean concentration of 1.2 ppb in older homes (Park 2008). A study of urban indoor areas in Argentina had a mean of 0.05 ppb for styrene (Massolo, et al. 2010). A study of Hong Kong homes demonstrated a mean concentration of 2.6 ppb (Guo 2009). A study of non-smokers in

the United Kingdom had a mean styrene concentration of 0.2 ppb (Delgado-Saborit 2011). An examination of 13 studies of homes in industrialized countries had a mean of 1.4 ppb.

1.2.1.12. O- Xylene (Analyte #54)

O-xylene is similar to m/p-xylene, except it is used in the production of phthalic anhydride (ACGIH 1991). Its health effects are similar to m/p xylene and it has a 6-hour LC_{50} for rats of 4595 ppm (ACGIH 1991).

An Argentinean study found that mean urban indoor air concentration of o-toluene was 1.1 ppb (Massolo, et al. 2010). A study of UK non-smokers had a mean o-xylene concentration in the home of 0.46 ppm (Delgado-Saborit 2011). In 11 studies of homes in industrialized countries, mean o-xylene levels were 1.9 ppb (Logue 2010).

1.2.1.13. 1,2,4-Trimethyl Benzene (Analyte #57)

1,2,4-trimethyl benzene is a colorless liquid with a pleasant odor. It is naturally present in oil and coal tar and is used as a raw material in chemical synthesis and used as an ultraviolet stabilizer in plastics (ACGIH 1991).

1,2,4-trimethyl benzene damages the central nervous system, with exposed workers complaining of anxiety, nervousness, and tension (ACGIH 1991). It is also an eye, nose, and respiratory irritant (ACGIH 1991). Birth weight of rats was significantly reduced when exposed gestationally to 1,2,4-trimethyl benzene at levels of 600 ppm for two weeks (Saillenfait 2005). The TLV-TWA of all isomers of trimethyl benzene is 25 ppm (ACGIH 2010).

A study of Hong Kong homes found a mean 1,2,4-trimethyl benzene concentration of 1.0 ppb (Guo 2009). A United Kingdom study of non-smokers revealed a mean concentration of 0.5 ppb of 1,2,4-trimethyl benzene (Delgado-Saborit 2011). In 8 studies of homes in industrialized countries, the mean 1,2,4-trimethyl benzene concentration was 0.85 ppb (Logue 2010).

The lack of acceptable guidelines (MRL) for this pollutant in homes makes it difficult to assess the health implications of the findings of previous studies of urban housing. The concentrations in the homes were indeed well below the TLV, but its presence in homes could lead to insight on the use of VOCs in homes.

1.2.1.14. 1,4-Dichlorobenzene/ para-dichlorobenzene (Analyte #60)

1,4-Dichlorobenze (p-DCB) is a white crystalline solid with a strong odor. It is used as an insecticide and for moth control (ACGIH 1991). Rats, guinea pigs, and rabbits died when exposed to 1600 ppm p-DCB for 30 minutes per day for 5 to 9 days. Rabbits exposed to p-DCB concentrations of 770-800 ppm for 8 hours per day for 62 days experienced tremors, weakness, and death. When ingested at a rate of 150 and 300 mg p-DCB/kg/day for two years, male rats had an increased incidence of kidney tumors, as well as hepatocellular carcinomas. In humans, eye and nose irritation has been reported at levels of 80-160 ppm, with difficulty breathing at levels above 160 ppm. Other effects reported from high p-DCB exposure include weakness, dizziness, headache, and vomiting. Chronic exposures have led to jaundice or cirrhosis of the liver (ACGIH 1991). 1,4-dichlorbenzene is listed as group 2B carcinogen, possibly carcinogenic to humans. It is listed this way because there is inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in the livers of rats and mice (IARC 1999).

The ACGIH TLV-TWA is 10 ppm (ACGIH 2010). The acute MRL is 2 ppm, the intermediate MRL is 0.2 ppm, and the chronic MRL is 0.01 ppm (ATSDR 2010).

Moth repellents commonly used in home emitted para-dichlorobenzene at rates from 0.033 to 0.011 g/h depending on the manufacturer (Shinohara 2008). In a study of homes in Hong Kong, the mean concentration of p-DCB was 0.96 ppb (Guo 2009). Another study of home in Shizuoka, Japan showed geometric mean levels of p-DCB to be 2.01 ppb in the living room and 11.9 ppb in the bedroom (Olansandan 1999). In 2 studies of new homes in industrialized countries, mean p-DCB levels were 9.15 ppb (Logue 2010).

p-DCB concentrations in homes in Japanese bedrooms were above the chronic MRL, and other studies have indicated p-DCB concentrations near the MRL. This indicates a potential for health to be negatively impacted by this pollutant in the home.

1.2.2. Carbon Dioxide

Carbon dioxide (CO_2) is a colorless, odorless gas that comes from the combustion of organic materials or from respiration (ACGIH 1991). Outdoor levels of carbon dioxide tend to be between 300 and 500 ppm, and the largest contributor to excess indoor carbon dioxide, in non-industrial settings, is human respiration (ASHRAE 2010).

Men exposed to carbon dioxide concentrations of 15,000 ppm for 42 days did not experience any physiological dysfunction (ACGIH 1991). At 30,000 ppm, workers that had adequate oxygen content of 18% did not complain of adverse effects until oxygen levels were reduced to 15-17% (ACGIH 1991). Another study of men exposed to 30,000 ppm carbon

dioxide, found that this concentration was irritating to workers during hard exercise (ACGIH 1991). The TLV-TWA is 5000 ppm and the TLV-STEL is 30,000 ppm (ACGIH 2010).

A study found, that in indoor air, when concentrations of CO_2 were increased with respect to the outdoor air concentration, symptoms indicative of sick building syndrome, including upper and lower respiratory irritation, increased (Apte 2000). The American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) notes that when indoor air are more than 700 ppm higher than outdoor air concentration, people will begin to feel uncomfortable and notice body odor from the people in the room. Since outdoor air is usually 300-500 ppm, ASHRAE sets its carbon dioxide indoor air guideline at 1000 ppm (ASHRAE 2010).

In Finnish apartments built specifically for residents with respiratory illness, the mean carbon dioxide concentration was 490 ppm three years after construction; whereas for an apartment building built with traditional construction techniques, the mean concentration was 673 ppm three years after construction (Tuomainen 2003). A Swedish study found that in homes where residents complained of nocturnal chest tightness, the mean carbon dioxide concentration was 1020 ppm while in houses with no such complaint the mean concentration was 850 ppm (Norbäck 1995). A study of green rehabilitation of low-income housing found that the average CO_2 level over a one-year period was 982 ppm (Breysse et al. 2011)

1.2.3. Carbon Monoxide

Carbon monoxide is a colorless, tasteless, scentless gas that comes from the incomplete combustion of a fuel (ACGIH 1991). Carbon monoxide exposure occurs in the home from outdoor sources such as vehicle exhaust infiltrating indoors, from incorrectly installed, poorly

maintained, or poorly ventilated cooking and heating appliances that burn fossil fuels (WHO 2010).

Carbon monoxide's toxicity in humans is centered around its ability to bind to hemoglobin to form carboxyhemoglobin (COHb) with a bond that is about 254 times stronger than that of oxygen, thereby reducing the blood's capacity of transporting oxygen throughout the body (WHO 2010). Acute carbon monoxide poisoning is usually due to lack of oxygen in the tissues of the body, with symptoms including headache, nausea, vomiting, dizziness, confusion, fainting, weakness, cardiac dysrhythmias, hypotension, cardiac arrest, seizures, and coma (ATSDR 2009). Chronic exposure to low levels of carbon monoxide can cause neurotoxic effects including headache, vertigo, irritation, and tinnitus (WHO 2010). Reports of fetal death have occurred when a pregnant mother experienced carbon monoxide poisoning (ATSDR 2009). The TLV-TWA for carbon monoxide is 25 ppm (ACGIH 2010). The World Health Organization recommends the 24-hour average concentration be less than 6.1 ppm and the 1-hour average be less than 30.6 ppm (WHO 2010). The United States EPA set its National Ambient Air Quality Standard for carbon monoxide at 9 ppm measured during any 8-hour exposure period and at 35 ppm for a 1-hour exposure (40 CFR 50.8).

A study of Korean high-rise apartment buildings demonstrated 30-minute mean concentrations on the lower floors of 0.8 ppm in the summer and 0.6 ppm in the winter, and on the higher floors of 0.7 for the summer and 0.3 for the winter (Jo 2006). A compilation of 6 studies from homes in industrialized nations revealed a mean CO concentration of 0.71 ppm (Logue 2010).

1.2.4. Particulate Matter Less Than 2.5 μm in Aerodynamic Diameter ($PM_{2.5}$)

$PM_{2.5}$, or fine particulate, are particles that are less than 2.5 μm in aerodynamic diameter. $PM_{2.5}$ is most often generated by combustion processes such as vehicle exhaust, coal burning for power generation, and other industrial processes (Pope 2006). In many poorer areas of the world, combustion of biomass for cooking and home heating is a large contributor to $PM_{2.5}$ indoor pollution (WHO 2005).

$PM_{2.5}$ is a pollutant with large health impacts. Since they are often from combustion of organic materials, they may contain metals, nitrates, sulfates, or acids that can be detrimental to health (Pope 2006). Also, due to the small size of the particles, they may penetrate deeply into the lungs, they can travel long distances, and they can infiltrate into indoor environments readily (Pope 2006). A decrease in $PM_{2.5}$ pollution of 10 $\mu g/m^3$ resulted in a 0.73 risk reduction for non-lung cancer related cardiovascular and respiratory disease (Laden 2006). Short-term exposures to $PM_{2.5}$ have been associated with increased hospital visits, myocardial infarctions, pulmonary inflammation, and exacerbation of existing pulmonary disease. Long-term exposures may cause chronic obstructive pulmonary disease, declines in lung function, pneumonia, and respiratory distress (Pope 2006). The World Health Organization set an air quality guideline of 10 $\mu g/m^3$ annually and 25 $\mu g/m^3$ for a 24 hour average (WHO 2005). The United States EPA lists $PM_{2.5}$ as a criteria pollutant in its National Ambient Air Quality Standards as 15 $\mu g/m^3$ for its annual average and 35 $\mu g/m^3$ for its 24-hour maximum (EPA 2011).

A study of homes in Belgium had a mean $PM_{2.5}$ concentration of 36 $\mu g/m^3$ (Stranger 2008). A study of non-smoking homes in Saskatchewan, Canada revealed geometric means of 5.32 $\mu g/m^3$ for the summer and 4.13 $\mu g/m^3$ in the winter (Heroux 2010). A study of Connecticut

homes found a mean of 18.7 μg/m^3 in air-conditioned homes and 21.1 μg/m^3 in non-air-conditioned homes (Leaderer 1999). In a collection of 13 studies in homes in industrialized countries, a mean concentration of 15.9 μg/m^3 was found (Logue 2010).

1.2.5. Formaldehyde

Formaldehyde is colorless gas with a strong, irritating odor. It is used in the production of resins, ethylene glycol, fertilizers, dyes, disinfectants, preservatives, embalming fluids, manufacture of particleboard, fiberboard, and plywood (ACGIH 1991). Indoor sources of formaldehyde found in homes include: plywood and particle board, insulating materials, textiles, paints, wallpapers, glues, adhesives, varnishes, detergents, disinfectants, electronic equipment, insecticides, and paper products (WHO 2010). As there are so many different possible sources of formaldehyde in the home, it is often difficult to determine which source contributes the most from house to house (WHO 2010).

As formaldehyde is very soluble in water, it is rapidly absorbed upon inhalation in the respiratory tract and metabolized (WHO 2010). Formaldehyde is very irritating to the upper respiratory tract, nose, and eyes in both acute and chronic exposures (ATSDR 1999). Some studies have suggested that eye and nose irritation in some humans occurs at 0.77 ppm (WHO 2010). Chronic bronchitis, shortness of breath, and nasal irritation were reported by embalmers with an average of 8.2 years of working in mean formaldehyde concentrations of 0.36 ppm (ATSDR 1999). These complaints are not unique to occupational exposure to formaldehyde, a study of people living in mobile and conventional homes found a positive concentration-response relationship between formaldehyde and health complaints. Many of these complaints occurred in homes with concentrations of formaldehyde less that 0.1 ppm, and when exposures reach 0.3

ppm, 90% of the occupants complained about respiratory tract irritation (ACGIH 1991). Some studies of children have suggested that formaldehyde can exacerbate allergies and asthma, though these effects have been difficult to quantify with the presence of several other confounding pollutants present in the study (WHO 2010).

Formaldehyde exposure has been noted to cause skin irritation, though this is less reported in exposures to formaldehyde gas than with aqueous solutions of formaldehyde contacting human skin (ATSDR 1999). Occupational exposure has been shown to cause neurological effects such as decreased performance on tests after exposure (ATSDR 1999). Formaldehyde is a group 1 carcinogen. Along with sufficient animal evidence of carcinogenicity, there is sufficient evidence that formaldehyde causes cancer in humans, with comprehensive studies finding an exposure-response relationship between formaldehyde exposure and deaths from nasopharyngeal cancer. Six of seven studies of occupationally exposed groups found excess mortality from leukemia, and there is limited evidence for other cancers such as sino-nasal, oral cavity, pancreas, larynx, lung, and brain (IARC 2006). The TLV-Ceiling for formaldehyde is 0.3 ppm (ACGIH 2010). The acute MRL is 0.04 ppm, the intermediate MRL is 0.03 ppm, and the chronic MRL is 0.008 ppm (ATSDR 2010).

In a study of homes in Hong Kong, a mean level of formaldehyde was found to be 91.4 ppb (Guo 2009). A study in Saskatchewan, Canada found a geometric mean of 26.2 ppb in the summer, and a geometric mean of 19.4 ppb in the winter (Heroux 2010). A Tokyo study found mean concentrations of formaldehyde of 70.0 ppb in 3-year old homes and 73.3 ppb in older homes (Park 2006). A compilation of 13 studies of homes in industrialized countries had a mean of 56.2 ppb (Logue 2010). A study by the US Department of Health and Human Services revealed an average baseline formaldehyde concentration in trailers that were used to house

those displaced by Hurricane Katrina of 1,040 ppb and 90 ppb when windows were opened (USDHHS 2007). Physicians treating people living in these trailers noted an increase in the prevalence of upper respiratory conditions.

TABLE I

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
2	Propene	IARC 1998 CA OEHHA 1999	Petroleum refining, Combustion of organic materials	TLV: TWA-500 ppm	Simple asphyxiant, Upper respiratory tract irritant, IARC Group 3 Carcinogen (not classifiable as a human carcinogen).
3	Dichloro-difluoro-methane	Azar 1972 WHO 1999 Lo 2006 Ritchie 2001 EPA 2010	Aerosol propellant, refrigerant, fire extinguishant, production banned in 1995 due to Montreal Protocol	TLV: TWA-1000 ppm	Cardiac sensitization; neurobehavioral effects in rats with high acute exposures
4	Chloro-methane (methyl chloride)	ACGIH 1991 IARC 1999 ATSDR 1998	Grass, wood, charcoal burning byproduct; formerly used as a refrigerant; catalyst for low pressure polymerization; silicone production	TLV: TWA-50 ppm TLV: STEL-100 ppm	Group 3 carcinogen (not classifiable as a human carcinogen); liver cirrhosis; confusion; staggering; slurred speech; headache; reproductive toxin; teratogenic effects

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
5	1,2-Dichlorotetrafluoroethane	ACGIH 1991 EPA 2010	Aerosol propellant, refrigerant, solvent, fire extinguisher, blowing agent, dielectric fluid; production banned in 1995 due to Montreal Protocol	TLV: TWA-1000 ppm	Weak narcotic with low toxicity, impairs pulmonary function,
6	Vinyl Chloride	ACGIH 1991 ATSDR 2006 IARC 2008	Manufacture of PVC plastics; plastic piping, floor covering, consumer goods, electrical appliances.	TLV: TWA 1 ppm	Group 1 Carcinogen (carcinogenic to humans); angiosarcoma of the liver; lung irritant at very high levels; development of Reynaud's phenomenon
7	1,3-Butadiene	ATSDR 2009 IARC 1999 ACGIH 1991 Austin 2001	Product of petrochemical industry; used in rubbers, resins and latexes; found in municipal fires; engine exhaust; cigarette smoke	TLV: TWA 2 ppm 2-hour LC_{50} (rats): 122,000 ppm MRL: 0.1 ppm	Group 2A Carcinogen (probably carcinogenic to humans); Cancer of the testes, digestive system and larynx; Hodgkin's disease; nausea; dry mouth and nose; headache

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
8	Bromo-methane (methyl bromide)	ACGIH 1991 ATSDR 1992 IARC 1999	Pest control fumigant; former refrigerant and fire extinguisher;	TLV: TWA-1ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); lung edema; skin irritant
9	Chloroethane (ethyl chloride)	IARC 1991 ATSDR 1998	Used in the production of leaded gasoline; dyes; medicines; refrigerant; solvent; formerly an anesthetic	TLV: TWA-100 ppm Deaths in guinea pigs at 40,000 ppm for 9 hours	Group 3 Carcinogen (not classifiable as a human carcinogen); liver damage,
10	Acrolein	ACGIH 1991 IARC 1995	Intermediate in the synthesis of acrylic acid; leather tanning, biocide	TLV: Ceiling 0.1 ppm Single and repeated exposures to 3–4 ppm caused death in rats and monkeys before the 10th day of exposure Acute MRL: 3 ppb Int MRL: 0.04 ppb	Group 3 Carcinogen ((not classifiable as a human carcinogen);); nose irritation; throat irritation; decreased respiratory rate
11	Acetone	ACGIH 1991	Solvent; chemical intermediate; paints; lacquers; varnishes	TLV: TWA 500 ppm TLV: STEL 750 ppm Acute MRL: 26 ppm Int. MRL: 13 ppm Chronic MRL: 13 ppm	Irritation of nose, trachea, lungs, and throat; eye irritation; central nervous system impairments

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
12	Halocarbon-11	ACGIH 1991 EPA 2010	Blowing agent; refrigerant; production banned in 1995 due to Montreal Protocol	TLV: Ceiling 1000 ppm	cardiac sensitization
13	1,1-Dichlor-ethene	ACGIH 1991 IARC 1999 ATSDR 1994	Production of plastic packaging materials and plastic films; carpet backing; flame retardant fiber coating	TLV: TWA 200 ppm	Group 3 Carcinogen ((not classifiable as a human carcinogen);); central nervous system, liver, kidney lung damage;
14	Methylene Chloride	IARC 1999	Solvent; paint stripper; photographic film	TLV: TWA 50 ppm	Group 2B Carcinogen (possibly carcinogenic to humans); lung and liver tumors in rats; increased carboxyhemoglobin levels in blood; central nervous system impairment
15	Carbon Disulfide	ACGIH 1991 ATSDR 1996	Manufacture of rayon, electronic vacuum tubes; and carbon tetrachloride	TLV: TWA 1 ppm	Peripheral nervous system impairments

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
16	1,1,2,-Trichloro-trifluoro-ethane	ACGIH 1991 EPA 2010	Refrigerant; solvent; degreasing; chemical intermediate; heat transfer medium; banned in 1995 due to Montreal Protocol	TLV: TWA 1000 ppm TLV: STEL 1250 ppm	Central nervous system impairments
17	Trans-1,2-Dichloro-ethylene	ATSDR 1996 ACGIH 1991	Solvent for perfumes, dyes, waxes, lacquers, thermoplastics; chemical intermediate for chlorinated compounds; food packaging adhesive; usually a mixture with cis-1,2-dichloroethylene	TLV: TWA 200 ppm	Irritation of the central nervous system; nausea; vertigo; fatigue; eye irritation;
18	1,1-Dichloro-ethane	ACGIH 1991 ATSDR 1990	Chemical intermediate; grain fumigant	TLV: TWA 100 ppm	Cardiac dysrhythmia;
19	Tert-Butyl Methyl Ether	IARC 1999 ATSDR 1996	Octane enhancer in gasoline	TLV: TWA 50 ppm 4-hour LC_{50} in rats: 33,370 ppm	Group 3 Carcinogen ((not classifiable as a human carcinogen);; upper respiratory tract irritation; nausea; vomiting; eye irritation

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
20	Vinyl Acetate	ACGIH 1991 IARC 1995 ATSDR 1992	Chemical intermediate; adhesive for paper, wood, metals, glass, porcelain; used in latex water paints; textile and leather finishing; emulsifying agent	TLV: TWA 10 ppm TLV: STEL 15 ppm Int. MRL: 0.01 ppm	Group 2B Carcinogen (possibly carcinogenic to humans); eye irritant; upper respiratory tract irritant; skin irritant
21	Methyl Ethyl Ketone	ACGIH 1991 ATSDR 1992	Used as a solvent; Surface coating; paints, glues, other finishes; vehicle exhaust;	TLV: TWA 200 ppm STEL: 300 ppm	Upper respiratory tract irritant, central nervous system and peripheral nervous system impairments
22	Cis-1,2-Dichloro-ethylene	ATSDR 1996 ACGIH 1991	Solvent for perfumes, dyes, waxes, lacquers, thermoplastics; chemical intermediate for chlorinated compounds; food packaging adhesive; usually a mixture with trans-1,2-dichloroethylene	TLV: TWA 200 ppm	Irritation of the central nervous system; nausea; vertigo; fatigue; eye irritation;
23	Hexane	ACGIH 1991 ATSDR 1999	Used in solvent mixtures for extraction of vegetable oil; present in gasoline, rubber cement;	TLV: TWA 50 ppm 4-hour LC_{50} in rats: 73,680 ppm	Peripheral neuropathy; eye irritation; central nervous system impairments

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
24	Chloroform	ACGIH 1991 IARC 1999 ATSDR 1997	Chemical intermediate; pesticide formulation; solvent; formerly used as an anesthetic	TLV: TWA 10 ppm 4-hour LC_{50} in rats: 9,770 ppm	Group 2B carcinogen (possibly carcinogenic to humans); liver damage; nausea; vomiting; central nervous system impairments
25	Ethyl Acetate	ACGIH 1991	Solvent; used in manufacture of silk, leather, perfumes, photographic films	TLV: TWA 400 ppm	Eye, nose, throat irritation;
26	Tetrahydro-furan	ACGIH 1991 ATSDR 2005	Solvent for polymers and resins; used in production of lacquers, glues, paints, ink	TLV: TWA 50 ppm TLV: STEL 100 ppm	Eye, nose, throat irritation; central nausea; headache; dizziness
27	1,2-Dichloro-ethane	ATSDR 2001 IARC 1999	Used in the production of vinyl chloride; solvent	8-hour LC_{50} in rats: 1000 ppm	Group 2B carcinogen (possibly carcinogenic to humans); cardiac arrhythmia; chronic bronchitis; nausea; vomiting; liver effects; kidney effects; weakness; dizziness; trembling; increase in premature births

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
29	1,1,1-Trichloro-ethane	ATSDR 2006 IARC 1999	Household solvent for glues, paints; industrial solvent for oil and grease; ceased production in 2002 because of ozone depletion	6-hour LC_{50} for rats: 10305 ppm	Group 3 Carcinogen (not classifiable as a carcinogen to humans); respiratory tract irritant; cardiac arrhythmia; reduced blood pressure; nausea; vomiting; diarrhea; central nervous system depression
30	Benzene	ACGIH 1991 ATSDR 2007 IARC 1982	Production of styrene, cyclohexane, and cumene; production of some types of dyes, rubbers, drugs, pesticides; naturally occurring in crude oil, gasoline, cigarette smoke	TLV: TWA 0.5 ppm TLV: STEL 2.5 ppm 4-hour LC_{50} for rats: 13,700 ppm Acute MRL: 9 ppb Int. MRL: 6 ppb Chronic MRL: 3 ppb	Group 1 Carcinogen (sufficient evidence that it is carcinogenic to man); leukemia; hematological effects; nasal irritation; sore throat; laryngitis; bronchitis; muscle pain; skin irritation; eye irritation; immunological effects; central nervous system effects; decreased fertility; increased miscarriages

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
31	Carbon Tetrachloride	ACGIH 1991 IARC 1999 ATSDR 2005	Used in the synthesis of chlorinated organic compounds, particularly chlorofluorocarbon which have been banned by the Montreal Protocol ; agricultural fumigant; solvent;	TLV: TWA 5 ppm TLV: STEL 10 ppm 8-hour LC_{50} for mice: 9,500 ppm	Group 2B carcinogen (possibly carcinogenic to humans); liver cancer; nausea; vomiting; gastrointestinal pain; liver damage; kidney damage; depression of central nervous system;
32	Cyclohexane	ACGIH 1991	Paint and varnish remover; solvent; chemical production; analytical chemistry	TLV: TWA 100 ppm	Central nervous system impairments, eye irritant;
33	1,2-Dichloro-propane	ATSDR 1989 IARC 1999	Industrial solvent; chemical intermediate;	4-hour LC_{50} in rats: 2000 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); respiratory tract irritant; liver damage; fatigue
34	Bromo-dichloro-methane	ATSDR 1989 IARC 1991 Torti 2001	Byproduct of the chlorination of water; Used in making other chemicals	Little research exists in regards to inhalation exposure, though mice were found to die when exposed to 30 ppm for 1 week	Group 2B carcinogen (possibly carcinogenic to humans, though through ingestion of drinking water); kidney damage; liver damage

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
35	Trichloro-ethylene	ACGIH 1991 IARC 1995 ATSDR 1997	Vapor degreasing of metals; adhesives; manufacture of poly vinyl chloride	TLV: TWA 10 ppm TLV: STEL 25 ppm 4-hour LC_{50} for rats: 12,500 ppm	Group 2A carcinogen (probably carcinogenic to humans); cancer of the liver; central nervous system depression; nausea; vomiting; liver damage; kidney damage; eye irritant; increased miscarriage rates;
36	1,4-Dioxane	ACGIH 1991 ATSDR 2007 IARC 1999	Solvent for chemical processing; paint and varnish remover; chemical intermediate;	TLV: TWA 20 ppm	Group 2B carcinogen (possibly carcinogenic to humans); liver damage; kidney damage; respiratory tract irritant; increased miscarriage rates
37	Heptane	ACGIH 1991	Standard for octane rating measurements; ingredient in gasoline; organic synthesis	TLV: TWA 400 ppm TLV: STEL 500 ppm	Upper respiratory tract irritant; central nervous system impairment

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
38	Cis-1,3-Dichloro-propene	ACGIH 1991 ATSDR 2008 IARC 1999	Soil fumigant; organic synthesis; often in mixture with trans-1,3-dichloropropene	TLV: TWA 1 ppm 4-hour LC_{50} for rats: 904 ppm	Group 2B carcinogen (possibly carcinogenic to humans); upper respiratory tract irritant; kidney damage; liver damage
39	Methyl Isobutyl Ketone	ACGIH 1991	Solvent in paints, lacquers, rubber cements;	TLV: TWA 20 ppm TLV: STEL 75 ppm	Liver cancer in mice; kidney effects; eye and skin irritant;
40	Trans-1,3-Dichloro-propene	ACGIH 1991 ATSDR 2008 IARC 1999	Soil fumigant; organic synthesis; often in mixture with cis-1,3-dichloropropene	TLV: TWA 1 ppm 4-hour LC_{50} for rats: 904 ppm	Group 2B carcinogen (possibly carcinogenic to humans); upper respiratory tract irritant; kidney damage; liver damage;
41	1,1,2-Trichloro-ethane	ACGIH 1991 ATSDR 1989 IARC 1999	Chemical intermediate for vinyl chloride production; solvent	TLV: TWA 10 ppm 6-hour LC_{50} in rats: 1654 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); liver damage; central nervous system impairments;

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
43	Toluene	ACGIH 1991 ATSDR 2000 IARC 1999	High octane blending stock for gasoline; solvent; chemical intermediate; household aerosols, paints, varnishes, rust inhibitors, thinners, solvent based cleaners, nail polish	TLV: TWA 20 ppm 7-hour LC_{50} in mice: 5320 ppm Acute MRL: 1 ppm Chronic MRL: 0.08 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); respiratory tract irritation; cardiac arrhythmia; irritation and impairment of the eye; central nervous system dysfunction; pregnancy loss;
44	Methyl N-Butyl Ketone	ACGIH 1991 ATSDR 1992	Waste product of wood pulping, coal gasification, and oil-shale processing; was in paint thinners, but is no longer produced in the United States	TLV: TWA 5 ppm TLV: STEL 10 ppm	Peripheral neuropathy; testicular damage
45	Dibromo-chloro-methane	ATSDR 2005 IARC 1999	Byproduct of chlorination of drinking water; chemical intermediate	1-hour doses of 56,000 ppm and 84,000 ppm have been reported to cause death in dogs	Group 3 Carcinogen (not classifiable as a human carcinogen though only studied with ingestion as route of exposure); liver damage; kidney damage; central nervous system depression

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
46	1,2-Dibromo-ethane	ACGIH 1991 ATSDR 1992 IARC 1999	Scavenger for lead in gasoline, production has fallen drastically with the elimination of leaded gasoline; chemical intermediate	6-hour LC_{50} for rats: 200 ppm	Group 2A carcinogen (probably carcinogenic to humans); lung tumors; bronchitis; liver damage; kidney damage; skin irritant
47	Tetrachloro-ethylene	IARC 1995 ATSDR 1997	Dry cleaning of fabrics; metal degreasing	TLV: TWA 25 ppm TLV: STEL 100 ppm 4-hour LC_{50} for rats: 5200 ppm	Group 2A carcinogen (probably carcinogenic to humans); esophageal cancer; cervical cancer; cardiac arrhythmias; upper respiratory tract irritation; liver damage; eye irritation; central nervous system impairments;
48	Chloro-benzene	ACGIH 1991 ATSDR 1990 IARC 1982	Solvent; chemical intermediate	TLV: TWA 10 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); liver damage; kidney damage;

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
49	Ethylbenzene	ACGIH 1991 ATSDR 2010 IARC 2000	Intermediate in the production of styrene; gasoline; paints; inks; pesticides; carpet glues; automotive products	TLV: TWA 20 ppm 4-hour LC_{50} in rats: 4000 ppm Acute MRL: 5 ppm Int. MRL: 2 ppm Chr. MRL: 0.06 ppm	Group 2B Carcinogen (possibly carcinogenic to humans); upper respiratory tract irritation; kidney damage; eye irritation; dizziness; cochlear impairment
50	M/P-Xylene	ACGIH 1991 ATSDR 2007 IARC 1999	Mixed xylene is in gasoline and aviation fuel, solvent in paints; m-xylene is an intermediate in isophthalic acid production; p-xylene is an intermediate in insecticide, pharmaceuticals, and terephthalic acid; p-xylene is used most prominently of all isomers in the production of polyester	TLV: TWA 100 ppm TLV STEL 150 ppm TLVs are for all isomers 4-hour LC_{50} for rats mixed xylene: 6350 ppm 6-hour LC_{50} for rats for m-xylene: 5267 ppm 6-hour LC_{50} for rats for p-xylene: 3907 ppm Mixed Xylenes MRLs: Acute: 2 ppm Intermediate: 0.6 ppm Chronic: 0.05 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); upper respiratory tract irritant; impaired pulmonary function; nausea; vomiting; eye irritation; central nervous system impairments; dizziness, muscle fatigue

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
51	Bromoform	ACGIH 1991 ATSDR 2005 IARC 1999	Byproduct of chlorination of drinking water; chemical intermediate; synthesis of pharmaceuticals; solvent	TLV: TWA 0.5 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); liver damage; upper respiratory tract irritant; eye irritant
52	Styrene	ACGIH 1991 ATSDR 2010 IARC 2002	Automobile exhaust; production of plastic and rubber; packaging, especially foam; pipes; flooring; furnishings; insulation; cups, plates, bowls	TLV: TWA 20 ppm TLV: STEL 40 ppm 4-hour LC_{50} for rats: 4940 ppm Acute MRL: 5 ppm Chr. MRL: 0.2 ppm	Group 2B carcinogen (possibly carcinogenic to humans); upper respiratory tract irritation; nausea; liver dysfunction; eye irritation; central and peripheral nervous system dysfunction
53	1,1,2,2-Tetrachloro-ethane	ACGIH 1991 ATSDR 2008 IARC 1999	Chemical intermediate; formerly used on large scale to produce chemicals but now is not widely used due to less toxic substitutes availability	TLV: TWA 1 ppm 4-hour LC_{50} in rats: 1253 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); mucosal irritation; stomach pain; nausea; vomiting; loss of appetite; liver damage; dizziness; headache; numbness; drowsiness

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
54	O-Xylene	ACGIH 1991 ATSDR 2007 IARC 1999	Mixed xylene is in gasoline and aviation fuel, solvent in paints; o-xylene is involved in the manufacture of phthalic anhydride;	TLV: TWA 100 ppm TLV STEL 150 ppm TLVs are for all isomers 6 hour LC_{50} for rats for o-xylene: 4595 ppm Mixed Xylenes MRLs: Acute: 2 ppm Intermediate: 0.6 ppm Chronic: 0.05 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); upper respiratory tract irritant; impaired pulmonary function; nausea; vomiting; eye irritation; central nervous system impairments; dizziness, muscle fatigue
55	1-Ethyl-4-Methyl Benzene	No information available			
56	1,3,5-Trimethyl Benzene	ACGIH 1991	Present in petroleum and coal tar; raw material in chemical synthesis; ultraviolet stabilizer;	TLV: TWA 25 ppm	Eye, nose, and throat irritant; central nervous system depression
57	1,2,4-Trimethyl Benzene	ACGIH 1991	Present in petroleum and coal tar; raw material in chemical synthesis; ultraviolet stabilizer;	TLV: TWA 25 ppm	Eye, nose, and throat irritant; central nervous system depression

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
58	Chloro-methyl Benzene (Alpha)	ACGIH 1991 IARC 1999	Chemical intermediate in the manufacture of tanning agents, pharmaceuticals, gasoline additives	TLV: TWA 1 ppm 2-hour LC_{50} for mice: 80 ppm	Group 2A carcinogen (probably carcinogenic to humans); lung cancer; respiratory tract irritant;
59	1,3- Dichloro-benzene	ACGIH 1991 ATSDR 2006 IARC 1999	Production of herbicides, pesticides, pharmaceuticals, dyes	N/A	Group 3 Carcinogen (not classifiable as its carcinogenic to humans); pituitary and thyroid gland damage
60	1,4- Dichloro-benzene	ACGIH 1991 ATSDR 2006 IARC 1999	Air freshener; moth ball ingredient; pesticides; dyes, mold repellant	TLC: TWA 10 ppm Acute MRL: 2 ppm Int. MRL: 0.2 ppm Chronic MRL: 0.01 ppm	Group 2B carcinogen (possibly carcinogenic to humans); respiratory tract irritant; liver damage; kidney damage
61	1,2- Dichloro-benzene	ACGIH 1991 ATSDR 2006 IARC 1999	Organic synthesizer of herbicides	TLV: TWA 25 ppm TLV: STEL 10 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); liver damage;
62	1,2,4-Trichloro-benzene	ACGIH 1991	Present in petroleum and coal tar; raw material in chemical synthesis; ultraviolet stabilizer;	TLV: TWA 25 ppm	Eye, nose, and throat irritant; central nervous system depression

[1]Numbers 1, 28, and 42 were used as calibration standards

TABLE I (CONTINUED)

INFORMATION ON THE 60 VOCs FOUND IN THE TO-15 ANALYSIS

No.[1]	Contaminant	References	Sources	Toxicity Levels	Health Effects
63	Hexachloro-1,3-Butadiene	ACGIH 1991 ATSDR 1994 IARC 1999	Formed during the process of making other chemicals; solvent for rubber;	TLV: TWA 0.02 ppm	Group 3 Carcinogen (not classifiable as a human carcinogen); kidney damage; lowering of respiratory rate

[1]Numbers 1, 28, and 42 were used as calibration standards

1.3. Conclusions of Literature Review

There is a relationship between housing and health and there is also evidence that certain interventions in housing improve health. Many housing-related exposures cause or exacerbate diseases in occupants in these homes. A review of the healthy homes literature highlights the need to control moisture (Jedrychowski et al.2011, Perzanowski et al. 1998, Taskinen et al. 2007, Andersson et al. 1997), pests (Brunekreek et al. 1989), and exposure to toxic chemicals (Edwards et al. 2001, Dawson and McAlary 2009). The literature related to indoor air quality, and more specifically, related to sick building syndrome (Seppänenn et al. 1999, Fisk et al. 2009) highlights inadequate ventilation and volatile organic compounds (VOCs) as likely causes of non-specific health complaints such as headache, fatigue, and nose and throat irritation. Formaldehyde is used in the manufacture of many building products. Use of these products can result in formaldehyde exposure due to materials off-gassing in the home (Park and Ikeda 2006, Maddalena et al. 2009).

The more detailed review of specific VOCs indicates these pollutants have the potential to cause adverse health impacts and their presence in materials that are used to build homes or in products that are brought into the home, indicate that they are indeed worthy of further study.

New construction and renovation of existing housing is beginning to embrace "green" elements. These elements are designed to decrease energy demand and water use, to improve sites, to use materials beneficial to the environment, and to improve the indoor environment. The energy reduction capabilities of a new green housing are documented (Breysse et al. 2010), however, there is only limited evidence that these homes improve human health (Breysse et al. 2011, Takaro et al. 2011).

This research was conducted as a part of the larger Moving Into Green Healthy Homes: The Yield in health benefits (MIGHHTY) study. The MIGHHTY study is looking at the possible health benefits of moving into green housing through interviews with residents of green housing, visual inspections, and examination of Medicaid data related to health-care system usage, at 3 low income housing developments. This sub-study, described here and in the next section, examined the levels of indoor air contaminants in a sample of non-smoking units in each of the three developments.

The developments included one development that was built more than 30 years ago, has not been significantly renovated in that time, and has no green construction features. The other two developments were built within the last 10 years and feature elements of green and healthy home construction including those shown in Table II.

This study investigated air contaminants including carbon dioxide, carbon monoxide, formaldehyde, particulate matter that is less than 2.5 microns in aerodynamic diameter, propene, acrolein, acetone, methyl ethyl ketone, benzene, toluene, 1,4-dichlorobenzene, vinyl acetate, heptane, ethyl benzene, m/p-xylene, o-xylene, styrene, and 1,2,4-trimethyl benzene. All of these pollutants are known to cause adverse health effects through exposure in people's homes, with the possible exception of carbon dioxide, which is generally used as an indicator of the influx of outdoor fresh air. All of these contaminants were measured above the limit of detection in at least 80% of the measurements.

The next chapter describes the research sub-study on indoor air contaminants. It is written in the form of a research manuscript for submission to a peer-reviewed journal. As such, some of the material presented here is repeated. The redundancy is intentional with Chapter 1

presenting a robust review of the literature related to residential indoor air quality and Chapter 2 presenting the research study examining indoor air contaminants measured in three low-income housing developments.

TABLE II

GREEN AND HEALTHY HOME FEATURES

New Development #1	New Development #2
A/C complies with ASHRAE 90A	ASHRAE 62 compliance
Air tight ducts	Adequate exhaust
Balance Air systems	Balance air handling and ductwork
Ventilation system balancing	Vent systems balanced and leak tested
Furnaces 85% efficient, 40% Relative Humidity spec	Programmable thermostat
High efficiency air handler filters	Kitchen exhaust vented to outside
Exterior Duct insulation (not internal)	Range hoods 160 cfm ducted to exterior
Vented range hood	Bath exhaust ducted to exterior
Toilet exhaust	Flashing
Building envelope	Foundation water proofing
Roof flashing	Waterproofing and insulated foundations
Window flashing	Asbestos-free damp-proofing
Damp-proof basements using non-asbestos films	Windows ASTM D4099-82 and ANSI AAMA 101V-86
Water infiltration for windows <10 lb/ft^2	Air infiltration barrier
Air infiltration for windows <0.6 cfm/ft^2	Air-infiltration barrier (Tyvek)
Wall infiltration criteria	Air infiltration for doors <0.06 cfm/ft^2 and water penetration <10 lb/ft^2
Formaldehyde-free insulation and moisture barriers	Air infiltration for storm doors < 0.05cfm/sq ft and condensation resistance
Vapor Retarder	Roof insulation R value >19
Backflow preventers	Vapor retarders
Hot water heater drain pans	Sound and thermal insulation
No carpet in kitchens and baths	Sound attenuation blankets
Acoustical ceiling	Energy Star appliances
Acoustical duct insulation	Radon testing completed all below 4 pci/L
Acoustical sealant for wallboard	Grab bars in baths
Vibration isolators for noise	
Water saving faucets	
CO monitor in garage	
Lead-free flux on soldered drinking water pipe joints	
Low VOC Paint	

1.4. Cited Literature

Agency for Toxic Substances and Disease Registry. (1989) Toxicological Profile for 1,1,2-Trichloroethane.

Agency for Toxic Substances and Disease Registry. (1989) Toxicological Profile for 1,2-Dichloropropane.

Agency for Toxic Substances and Disease Registry. (1989) Toxicological Profile for Bromodichloromethane.

Agency for Toxic Substances and Disease Registry. (1990) Toxicological Profile for 1,1-Dichloroethane.

Agency for Toxic Substances and Disease Registry. (1990) Toxicological Profile for Chlorobenzene.

Agency for Toxic Substances and Disease Registry. (1992) Toxicological Facts for 2-Butanone.

Agency for Toxic Substances and Disease Registry. (1992) Toxicological Profile for Vinyl Acetate.

Agency for Toxic Substances and Disease Registry. (1992) Toxicological Profile for 1,2-Dibromoethane.

Agency for Toxic Substances and Disease Registry. (1992) Toxicological Profile for 2-Hexanone.

Agency for Toxic Substances and Disease Registry. (1992) Toxicological Profile for Bromomethane

Agency for Toxic Substances and Disease Registry. (1994) Toxicological Profile FOR 1,1-Dichloroethylene.

Agency for Toxic Substances and Disease Registry. (1994) Toxicological Profile for Acetone.

Agency for Toxic Substances and Disease Registry. (1994) Toxicological Profile for Hexachlorobutadiene.

Agency for Toxic Substances and Disease Registry. (1996) Toxicological Profile for 1,2-Dichloroethene.

Agency for Toxic Substances and Disease Registry. (1996) Toxicological Profile for Methyl-*tert*-butyl Ether.

Agency for Toxic Substances and Disease Registry. (1997) Toxicological Profile for Chloroform.

Agency for Toxic Substances and Disease Registry. (1997) Toxicological Profile for Tetrachloroethylene.

Agency for Toxic Substances and Disease Registry. (1997) Toxicological Profile for Trichloroethylene (TCE).

Agency for Toxic Substances and Disease Registry. (1998) Toxicological Profile for Chloroethane.

Agency for Toxic Substances and Disease Registry. (1998) Toxicological Profile for Chloromethane.

Agency for Toxic Substances and Disease Registry. (1999) Toxicological Profile for Formaldehyde.

Agency for Toxic Substances and Disease Registry. (1999) Toxicological Profile for *n*-Hexane.

Agency for Toxic Substances and Disease Registry. (2000) Toxicological Profile for Methylene Chloride.

Agency for Toxic Substances and Disease Registry. (2000) Toxicological Profile for Toluene.

Agency for Toxic Substances and Disease Registry. (2001) Toxicological Profile for 1,2-Dichloroethane.

Agency for Toxic Substances and Disease Registry. (2005) Toxicological Profile for Bromoform and Chlorodibromomethane.

Agency for Toxic Substances and Disease Registry. (2005) Toxicological Profile for Carbon Tetrachloride.

Agency for Toxic Substances and Disease Registry. (2006) Toxicological Profile for 1,1,1-Trichloroethane.

Agency for Toxic Substances and Disease Registry. (2006) Toxicological Profile for Dichlorobenzene.

Agency for Toxic Substances and Disease Registry. (2006) Toxicological Profile for Vinyl Chloride.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for 1,4 Dioxane.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for Acrolein.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for Benzene.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for Xylene.

Agency for Toxic Substances and Disease Registry. (2008) Toxicological Profile for 1,1,2,2-Tetrachloroethane.

Agency for Toxic Substances and Disease Registry. (2008) Toxicological Profile for Dichloropropenes.

Agency for Toxic Substances and Disease Registry. (2009) Toxicological Profile For 1,3-Butadiene.

Agency for Toxic Substances and Disease Registry. (2009) Toxicological Profile for Carbon Monoxide.

Agency for Toxic Substances and Disease Registry. (2010) Addendum to the Toxicological Profile for 2-Butanone.

Agency for Toxic Substances and Disease Registry. (2010) Minimal Risk Levels (MRLs) for Hazardous Substances.

Agency for Toxic Substances and Disease Registry. (2010) Toxicological Profile for Ethylbenzene.

Agency for Toxic Substances and Disease Registry. (2010) Toxicological Profile for Styrene.

Agency for Toxic Substances and Disease Registry. (2011) About ATSDR.

Alptekin, F., Uskun, E., Kisioglu, A.N. and Ozturk, M. (2008) Unintentional non-fatal home-related injuries in Central Anatolia, Turkey: Frequencies, characteristics, and outcomes, *Injury*, 39, 535-546.

American Conference of Governmental Industrial Hygienists, Inc. (1991) Documentation of the Threshold Limit Values and Biological Exposure Indices: Sixth Edition.

American Conference of Governmental Industrial Hygienists, Inc. (2008) About.

American Conference of Governmental Industrial Hygienists, Inc. (2010) *2010 TLVs and BEIs: Based on the Documentation of the Threshold Limit Values for Chemical and Physical Agents & Biological Exposure Indices,* Cincinatti, OH, ACGIH Publications

American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. (2010) ANSI/ASHRAE Standard 62.1-2010, Ventilation for Acceptable Indoor Air Quality.

Ansari, F.A., Khan, A.H., Patel, D.K., Siddiqui, H., Sharma, S., Ashquin, M. and Ahmad, I. (2010) Indoor exposure to respirable particulate matter and particulate-phase PAHs in rural homes in North India, *Environ Monit Assess*, 170, 491-497.

Apte, M.G., Fisk, W.J. and Daisey, J.M. (2000) Associations Between Indoor CO_2 Concentrations and Sick Building Syndrome Symptoms in U.S. Office Buildings: An Analysis of the 1994-1996 BASE Study Data, *Indoor Air*, 10, 246.

Austin, C.C., Wang, D., Ecobichon, D.J. and Dussault, G. (2001) Characterization of Volatile Organic Compounds in Smoke at Municipal Structural Fires, *Journal of Toxicology & Environmental Health: Part A*, 63, 437-458.

Azar, A., Reinhardt, C.F., Maxfield, M.E., Smith, P.E. and Mullin, L.S. (1972) Experimental Human Exposures to Fluorocardon (sic) 12 (Dichlorodifluoromethane), *American Industrial Hygiene Association Journal*, 33, 207-216.

Breysse J, Jacobs DE, Weber W, Dixon S, Kawecki C, Aceti S and Lopez J. (2011) Health Outcomes and Green Renovation of Affordable Housing, *Public Health Reports*, 126, 64-75.

Brunekreef B, Dockery DW, Speizer FE, Ware JH, Spengler JD and Ferris BG. (1989) Home dampness and respiratory morbidity in children. *Am Rev Respir Dis*, 140, 1363.

Canadian Office of Environmental Health Hazard Assessment. (1999) Chronic Toxicity Summary: Propylene.

Center for Environmental Research Information. (1999) Compendium Method TO-15, *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air*.

Centers for Disease Control and Prevention. (2010) Facts about Mold and Dampness, 2011.

Code of Federal Regulations. Title 40: Protection of the Environment: Part 50- National primary and secondary ambient air quality standards.

Dawson, H.E. and McAlary, T. (2009) A Compilation of Statistics for VOCs from Post-1990 Indoor Air Concentration Studies in North American Residences Unaffected by Subsurface Vapor Intrusion, *Ground Water Monitoring & Remediation*, 29, 60-69.

De Bortoli, M., Kephalopoulos, S., Kirchner, S., Schauenberg, H. and Vissers, H. (1999) State-of-the-Art in the Measurement of Volatile Organic Compounds Emitted from Building Products: Results of European Interlaboratory Comparison, *Indoor Air*, 9, 103.

Delgado-Saborit, J.M., Aquilina, N.J., Meddings, C., Baker, S. and Harrison, R.M. (2011) Relationship of personal exposure to volatile organic compounds to home, work and fixed site outdoor concentrations, *Sci Total Environ*, 409, 478-488.

DiGuiseppi, C., Jacobs, D.E., Phelan, K.J., Mickalide, A.D. and Ormandy, D. (2010) - Housing Interventions and Control of Injury-Related Structural Deficiencies: A Review of the Evidence, - *Journal of Public Health Management and Practice*.

Edwards, R.D., Jurvelin, J., Saarela, K. and Jantunen, M. (2001) VOC concentrations measured in personal samples and residential indoor, outdoor and workplace microenvironments in EXPOLIS-Helsinki, Finland, *Atmos Environ*, 35, 4531-4543.

energystar.gov. (2011) Qualified New Homes, 2011.

Environmental Protection Agency. (2010) The Montreal Protocol on Substances that Deplete the Ozone Layer.

Estarlich, M., Ballester, F., Aguilera, I., Fernández-Somoano, A., Lertxundi, A. and et al. (2011) Residential Exposure to Outdoor Air Pollution during Pregnancy and Anthropometric Measures at Birth in a Multicenter Cohort in Spain, *Environ Health Perspect*.

Farrow, A., Taylor, H. and Golding, J. (1997) Time Spent in the Home by Different Family Members, *Environmental Technology*, 18, 605.

Fisk, W.J., Mirer, A.G. and Mendell, M.J. (2009) Quantitative relationship of sick building syndrome symptoms with ventilation rates, *Indoor Air*, 19, 159-165.

Golden, R. (2011) Identifying an indoor air exposure limit for formaldehyde considering both irritation and cancer hazards, *Crit Rev Toxicol*, 1-50.

Guo, H., Kwok, N.H., Cheng, H.R., Lee, S.C., Hung, W.T. and Li, Y.S. (2009) Formaldehyde and volatile organic compounds in Hong Kong homes: concentrations and impact factors, *Indoor Air*, 19, 206-217.

Héroux, M., Clark, N., Van Ryswyk, K., Mallick, R., Gilbert, N.L., Harrison, I., Rispler, K., Wang, D., Anastassopoulos, A., Guay, M., MacNeill, M. and Wheeler, A. (2010) Predictors of Indoor Air Concentrations in Smoking and Non-Smoking Residences, *Int J Environ Res Public Health*, 7.

Hun, D.E., Corsi, R.L., Morandi, M.T. and Siegel, J.A. (2010) Formaldehyde in residences: long-term indoor concentrations and influencing factors, *Indoor Air*, 20, 196-203.

International Agency For Research on Cancer. (1982) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 29: Some Industrial Chemicals and Dyestuffs.

International Agency For Research on Cancer. (1991) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 52: Chlorinated Drinking-water; Chlorination By-products; Some Other Halogenated Compounds; Cobalt and Cobalt Compounds.

International Agency For Research on Cancer. (1995) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 63: Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals.

International Agency For Research on Cancer. (1999) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 71: Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide.

International Agency For Research on Cancer. (1999) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 73: Some Chemicals that Cause Tumours of the Kidney or Urinary Bladder in Rodents and Some Other Substances.

International Agency For Research on Cancer. (2000) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 77: Some Industrial Chemicals.

International Agency For Research on Cancer. (2002) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 82: Some Traditional Herbal Medicines, Some Mycotoxins, Naphthalene and Styrene.

International Agency For Research on Cancer. (2006) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 88: Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxypropan-2-ol.

International Agency for Research on Cancer. (2008) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 97: 1,3-Butadiene, Ethylene Oxide and Vinyl Halides (Vinyl Fluoride, Vinyl Chloride and Vinyl Bromide).

International Agency For Research on Cancer. (2011) Preamble to the IARC Monographs (amended January 2006).

International Agency For Research on Cancer. (30 March 1998) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 19: Some Monomers, Plastics and Synthetic Elastomers, and Acrolein.

International Programme on Chemical Safety: Dichlordifluoromethane, (1999).

International Programme on Chemical Safety: P-Dichlorobenzene, (2004).

Jacobs, D.E., Brown, M.J., Baeder, A., Sucosky, M.S., Margolis, S., Hershovitz, J., Kolb, L. and Morley, R.L. (2010) - A Systematic Review of Housing Interventions and Health: Introduction, Methods, and Summary Findings, - *Journal of Public Health Management and Practice*.

Jacobs, D.E., Brown, M.J., Baeder, A., Sucosky, M.S., Margolis, S., Hershovitz, J., Kolb, L. and Morley, R.L. (2010) A Systematic Review of Housing Interventions and Health: Introduction, Methods, and Summary Findings, *Journal of Public Health Management and Practice*, 16.

Jedrychowski, W., Maugeri, U., Perera, F., Stigter, L., Jankowski, J., Butscher, M., Mroz, E., Flak, E., Skarupa, A. and Sowa, A. (2011) Cognitive function of 6-year old children exposed to mold-contaminated homes in early postnatal period. Prospective birth cohort study in Poland, *Physiol Behav*, 104, 989-995.

Jedrychowski, W., Maugeri, U., Perera, F., Stigter, L., Jankowski, J., Butscher, M., Mroz, E., Flak, E., Skarupa, A. and Sowa, A. (2011) Cognitive function of 6-year old children exposed to mold-contaminated homes in early postnatal period. Prospective birth cohort study in Poland, *Physiol Behav*, 104, 989-995.

Jo, W. and Lee, J. (2006) Indoor and outdoor levels of respirable particulates (PM10) and Carbon Monoxide (CO) in high-rise apartment buildings, *Atmos Environ*, 40, 6067-6076.

Krewski, D., Burnett, R.T., Goldberg, M.S., Hoover, K., Siemiatycki, J., Abrahamowicz, M. and White, W.H. (2004) Validation of the Harvard Six Cities Study of Particulate Air Pollution and Mortality, *N Engl J Med*, 350, 198-199.

Krieger, J. and Higgins, D.L. (2002) Housing and Health: Time Again for Public Health Action, *Am J Public Health*, 92, 758-768.

Krieger, J. and Higgins, D.L. (2002) Housing and Health: Time Again for Public Health Action, *Am J Public Health*, 92, 758-768.

Krieger, J., Jacobs, D.E., Ashley, P.J., Baeder, A., Chew, G.L., Dearborn, D., Hynes, H.P., Miller, J.D., Morley, R., Rabito, F. and Zeldin, D.C. (2010) Housing Interventions and Control of Asthma-Related Indoor Biologic Agents: A Review of the Evidence, *Journal of Public Health Management and Practice*.

Laden, F., Schwartz, J., Speizer, F.E. and Dockery, D.W. (2006) Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study, *American Journal of Respiratory and Critical Care Medicine*, 173, 667.

Leaderer, B.P., Naeher, L., Jankun, T., Balenger, K., Holford, T.R., Toth, C., Sullivan, J., Wolfson, J.M. and Koutrakis, P. (1999) Indoor, Outdoor, and Regional Summer and Winter Concentrations of PM_{10}, $PM_{2.5}$, SO_4^{2-}, H^+, NH_4^+, NO_3^-, NH_3, and Nitrous Acid in Homes with and *Environ Health Perspect*, 107, 223-231.

Leikauf, G.D. (2002) Hazardous Air Pollutants and Asthma, *Environ Health Perspect*, 110, pp. 505-526.

Leikauf, G.D. (2002) Hazardous Air Pollutants and Asthma, *Environ Health Perspect*, 110, pp. 505-526.

Letz, G.A. (1990) Sick Building Syndrome: Acute Illness among Office Workers—The Role of Building Ventilation, Airborne Contaminants and Work Stress, *Allergy and Asthma Proceedings*, 11, 109-116.

Lin, S., Jones, R., Munsie, J.P., Nayak, S.G., Fitzgerald, E.F. and Hwang, S.A. Childhood asthma and indoor allergen exposure and sensitization in Buffalo, New York, *Int J Hyg Environ Health*.

Lo, S., Chan, C., Chen, W. and Wang, J. (2006) Grand rounds: outbreak of hematologic abnormalities in a community of people exposed to leakage of fire extinguisher gas, *Environ Health Perspect*, 114, 1713-1717.

Logue, J.M., McKone, T.E., Sherman, M.H. and Singer, B.C. (2011) Hazard assessment of chemical air contaminants measured in residences, *Indoor Air*, 21, 92-109.

M A Andersson, M Nikulin, U Köljalg, M C Andersson, F Rainey, K Reijula, E L Hintikka and M Salkinoja-Salonen. (1997) Bacteria, molds, and toxins in water-damaged building materials. *Applied and Environmental Microbiology*, 63, 387.

Maddalena R, Russell M, Sullivan DP and Apte MG. Formaldehyde and Other Volatile Organic Chemical Emissions in Four FEMA Temporary Housing Units, *Environ Sci Technol*, - 5626.

Massolo, L., Rehwagen, M., Porta, A., Ronco, A., Herbarth, O. and Mueller, A. (2010) Indoor?outdoor distribution and risk assessment of volatile organic compounds in the atmosphere of industrial and urban areas, *Environ Toxicol*, 25, 339-349.

Norbäck, D., Björnsson, E., Janson, C., Widström, J. and Boman, G. (1995) Asthmatic symptoms and volatile organic compounds, formaldehyde, and carbon dioxide in dwellings. *Occupational and Environmental Medicine*, 52, 388-395.

Olansandan, T.A. and Hidetsuru, M. (1999) A passive sampler–GC/ECD method for analyzing 18 volatile organohalogen compounds in indoor and outdoor air and its application to a survey on indoor pollution in Shizuoka, Japan, *Talanta*, 50, 851.

Park, J.S. and Ikeda, K. (2006) Variations of formaldehyde and VOC levels during 3 years in new and older homes, *Indoor Air*, 16, 129-135.

Perzanowski, M.S., Sporik, R., Squillace, S.P., Gelber, L.E., Call, R., Carter, M. and Platts-Mills, T.A.E. (1998) Association of sensitization to Alternaria allergens with asthma among school-age children, *J Allergy Clin Immunol*, 101, 626-632.

Pope III, C.A. and Dockery, D.W. (2006) Health Effects of Fine Particulate Air Pollution: Lines that Connect, *J Air Waste Manage Assoc*, 56, 709-742.

Public Health Assessments & Health Consultations: Casmalia Resources Superfund Site: Appendix D-E, (2005).

Ritchie G.D., Kimmel E.C., Bowen L.E., Reboulet J.E. and Rossi J. (2001) Acute Neurobehavioral Effects in Rats from Exposure to HFC 134a or CFC 12, *Neurotoxicology*, 22, 233.

Runyan, C.W., Casteel, C., Perkis, D., Black, C., Marshall, S.W., Johnson, R.M., Coyne-Beasley, T., Waller, A.E. and Viswanathan, S. (2005) Unintentional injuries in the home in the United States: Part I: Mortality, *Am J Prev Med*, 28, 73-79.

Saillenfait, A.M., Gallissot, F., Sabate, J.P. and Morel, G. (2005) Developmental toxicity of two trimethylbenzene isomers, mesitylene and pseudocumene, in rats following inhalation exposure, *Food and Chemical Toxicology*, 43, 1055-1063.

Sandel, M., Baeder, A., Bradman, A., Hughes, J., Mitchell, C., Shaughnessy, R., Takaro, T.K. and Jacobs, D.E. (2010) Housing Interventions and Control of Health-Related Chemical Agents: A Review of the Evidence, *Journal of Public Health Management and Practice*.

Seaman, V.Y., Bennett, D.H. and Cahill, T.M. (2009) Indoor acrolein emission and decay rates resulting from domestic cooking events, *Atmos Environ*, 43, 6199-6204.

Seppänen, O.A., Fisk, W.J. and Mendell, M.J. (1999) Association of Ventilation Rates and CO2 Concentrations with Health andOther Responses in Commercial and Institutional Buildings, *Indoor Air*, 9, 226-252.

Shinohara, N., Ono, K. and Gamo, M. (2008) *p*-Dichlorobenzene emission rates from moth repellents and leakage rates from cloth storage cases, *Indoor Air*, 18, 63.

Stout, M.D., Herbert, R.A., Kissling, G.E., Suarez, F., Roycroft, J.H., Chhabra, R.S. and Bucher, J.R. (2008) Toxicity and carcinogenicity of methyl isobutyl ketone in F344N rats and B6C3F1 mice following 2-year inhalation exposure, *Toxicology*, 244, 209-219.

Stranger, M., Potgieter-Vermaak, S.S. and Van Grieken, R. (2009) Particulate matter and gaseous pollutants in residences in Antwerp, Belgium, *Sci Total Environ*, 407, 1182-1192.

Takigawa, T., Wang, B., Saijo, Y., Morimoto, K., Nakayama, K., Tanaka, M., Shibata, E., Yoshimura, T., Chikara, H., Ogino, K. and Kishi, R. (2010) Relationship between indoor chemical concentrations and subjective symptoms associated with sick building syndrome in newly built houses in Japan, *Int Arch Occup Environ Health*, 83, 225-235.

Taskinen, T., Hyvärinen, A., Meklin, T., Husman, T., Nevalainen, A. and Korppi, M. (1999) Asthma and respiratory infections in school children with special reference to moisture and mold problems in the school, *Acta Pædiatrica*, 88, 1373-1379.

Torti, V.R., Cobb, A.J., Everitt, J.I., Marshall, M.W., Boorman, G.A. and Butterworth, B.E. (2001) Nephrotoxicity and Hepatotoxicity Induced by Inhaled Bromodichloromethane in Wild-Type and p53-Heterozygous Mice, *Toxicological Sciences*, 64, 269-280.

Tuomainen, M., Tuomainen, A., Liesivuori, J. and Pasanen, A.-. (2003) The 3-year follow-up study in a block of flats – experiences in the use of the Finnish indoor climate classification, *Indoor Air*, 13, 136-147.

U.S. EPA. (1989) Health and Environmental Effects Document for N-Heptane.

U.S. EPA. (2010) Care for Your Air: A Guide to Indoor Air Quality, 2011.

U.S. EPA. (2011) National Ambient Air Quality Standards (NAAQS)

U.S. EPA. (2011) National Ambient Air Quality Standards (NAAQS)

US Department of Health and Human Services. (2007) Formaldehyde Sampling of FEMA Temporary-Housing Trailers.

Watson, R. (2010) Green Building Market and Impact Report 2010, *Green Building Market and Impact Report 2010*.

Williams, J., Kool, B., Robinson, E. and Ameratunga, S. Longer term health of young and middle-aged adults following unintentional falls at home resulting in hospitalisation, *Injury*.

World Health Organization. (2005) Air quality guidelines - global update 2005.

World Health Organization. (2010) WHO guidelines for indoor air quality: selected pollutants.

2. Manuscript

2.1. Introduction

The relationship between housing and public health has been the subject of study and policy for many years. People tend to spend large amounts of time in their homes, making the home an environment of particular interest with regard to health. Young people are at significant risk at home, since they are more susceptible to disease and spend large amounts of time in the house. For example, infants average 19.3 hours per day in the home (Farrow 1997).

A review of the healthy homes literature highlights the need to control moisture (Jedrychowski et al.2011, Perzanowski et al. 1998, Taskinen et al. 2007, Andersson et al. 1997), pests (Brunekreek et al. 1989), and exposure to toxic chemicals (Edwards et al. 2001, Dawson and McAlary 2009). The literature related to indoor air quality, and more specifically, related to sick building syndrome (Seppänenn et al. 1999, Fisk et al. 2009) highlights inadequate ventilation and volatile organic compounds (VOCs) as likely causes of non-specific health complaints such as headache, fatigue, and nose and throat irritation. Formaldehyde is used in the manufacture of many building products. Use of these products can result in formaldehyde exposure due to materials off-gassing in the home (Park and Ikeda 2006, Maddalena et al. 2009).

The construction of environmentally friendly, or "green," housing is becoming more commonplace in the United States, with over one million homes in the country qualifying as ENERGY STAR homes (ENERGY STAR 2011). These homes are beginning to include elements of design and construction that are meant to decrease energy demand and water use, to improve sites, and to improve the indoor environment. One method to minimize the energy

impact of homes is to reduce leakage of outdoor air into the home to reduce heating and cooling costs.

As outdoor pollution rates continue to decrease and as outdoor-to-indoor air exchange rates are lowered, indoor sources of air pollutants are becoming a more important concern in pollutant exposure (Logue 2010). Although the energy-use reduction capabilities of new green housing are well-documented, the effects of these homes on human health have not been well-established. Furthermore, an exhaustive ASHRAE review showed there is a need for additional research on ventilation rates and health, especially in housing (Sundell 2011).

This research was conducted as a part of the larger Moving Into Green Healthy Homes: The Yield in health benefits (MIGHHTY) study. The MIGHHTY study is looking at the possible health benefits of moving into green housing through interviews with residents, visual inspections, and examination of Medicaid data related to health-care system usage, at three low-income housing developments. The study presented here examined the level of indoor air contaminants in a sample of non-smoking units in each of the three developments.

The developments included one development that was built more than 30 years ago, has not been significantly renovated since that time, and has no green construction features. The other two developments were built within the last 10 years, and feature elements of green and healthy home construction (Table III).

This study investigated air contaminants including carbon dioxide (CO_2), carbon monoxide (CO), formaldehyde, particulate matter that is less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$), total volatile organic compounds (TVOC) expressed as hexane equivalent VOC, vinyl acetate, heptane, ethyl benzene, m/p-xylene, o-xylene, styrene, 1,2,4-trimethyl

benzene, propene, acrolein, acetone, methyl ethyl ketone, benzene, toluene, and 1,4-dichlorobenzene.

Exposure to these pollutants are known to cause adverse health effects, with the possible exception of carbon dioxide, which is generally used as an indicator of the influx of outdoor fresh air.

2.2. Methods

Participants were recruited into the larger MIGHHTY study through an initial survey process, in which a visual assessment of the home was taken, Medicaid data recorded, and a health interview completed using an adaptation of the CDC National Health Interview Survey. Survey responses revealed homes where cigarette smoke was not allowed, and these homes were then recruited to further participate in the air sampling portion of the study.

Once households agreed to participate in the air sampling, an investigator set up the air samplers in the home and returned approximately 24 hours later to collect the samplers. Samplers were typically located in the living room of the homes, though another location was occasionally chosen due to furniture arrangements and preferences of study participant.

CO and CO_2 were simultaneously measured using a data logging Q-Trak Indoor Air Quality Monitor made by TSI, Inc. then uploaded to a personal computer. Formaldehyde was measured using a UMEX-100 Passive Formaldehyde Sampler made by SKC, Inc. then sent to a laboratory for analysis. The laboratory is accredited by the American Industrial Hygiene Association.

TABLE III

GREEN AND HEALTHY HOME FEATURES OF THE NEW DEVELOPMENTS

FEATURES	New Development #1	New Development #2
Years Built	2004-2010	2005-2009
ASHRAE 62 compliance		x
Furnaces 85% efficient, 40% relative humidity	x	
High efficiency air handler filters	x	
Ventilation system balancing	x	x
Air tight ducts	x	x
Exterior duct insulation (not internal)	x	
Vented range hood	x	x
Bath exhaust ducted to exterior	x	x
Roof and window flashing	x	x
Air infiltration for windows <0.6 cfm/ft^2 and water penetration <10 lb/ft^2	x	x
Windows ASTM D4099-82 and ANSI AAMA 101V-86		x
Air infiltration for doors <0.06 cfm/ft^2 and water penetration <10 lb/ft^2		
Air infiltration for storm doors < 0.05cfm/sq ft and condensation resistance		
Air infiltration barrier (wall)	x	x
Formaldehyde-free insulation and moisture barriers	x	x
Foundation water proofing (asbestos-free)	x	x
Backflow preventers	x	
Roof insulation R value >19		x
Hot water heater drain pans	x	
No carpet in kitchens and baths	x	
Sound insulation	x	x
Vibration isolators for noise	x	
Sound attenuation blankets		x
A/C complies with ASHRAE 90A	x	
Water saving faucets	x	
Programmable thermostat		x
Energy Star appliances		x
CO monitor in garage	x	
Lead-free flux on soldered drinking water pipe joints	x	
Low VOC Paint	x	
Radon testing completed all below 4 pci/L		x
Grab bars in baths		x

$PM_{2.5}$ was collected on pre-weighed polyvinyl chloride (PVC) filters in a SKC PEM2.5 pre-collector, using a pump that was pre- and post-calibrated with a bubble tube. The filters were sent to the laboratory, where they were post-weighed for analysis. Samples were blank corrected and concentrations were determined using the recorded volume of air sampled.

Whole air samples were collected in a vacuum-sealed canister with a flow regulator calibrated to draw air for 24 hours. These canisters were sent to an AIHA-accredited laboratory where the air sample was analyzed for 60 VOCs using EPA Compendium Method TO-15 (Center for Environmental Research Information 1999). A literature review identified individual VOCs commonly found in homes. Of these, VOC measurement results were further examined if at least 80% of the measurements for that contaminant were above the limit of detection.

Distributions of the contaminants were determined using distribution plots; contaminants that were found to be log-normally distributed are reported using geometric means and geometric standard deviations while contaminants that were found to be normally distributed are reported using arithmetic means and arithmetic standard deviations. Two-sided t-tests, with a significance level of $p<0.05$ were applied to normalized mean values to determine if there was a significant difference in contaminant concentration between each of the new developments and the old development. Data were analyzed using SAS 9.2 software.

Measured concentrations were compared to indoor air guidelines such as the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) 62.1 guideline for CO_2 indoors, World Health Organization (WHO) indoor air guidelines, United States Environmental Protection Agency (EPA) National Ambient Air Quality Standards (NAAQS), and Minimum Risk Levels (MRL) from the United States Agency for Toxic Substances and

Disease Registries. These guidelines are all designated to protect human health during extended exposures to these concentrations. When none of these guidelines were available, Threshold Limit Values (TLV®) from the American Conference of Governmental Industrial Hygienists (ACGIH®) were used. TLVs® may not be an appropriate guideline for air quality in the home, because they are developed for 8 hour exposures to healthy workers; whereas occupants of homes could include infants, elderly, immunologically compromised people, or other more susceptible people and likely are exposed for more than 8 hours per day.

2.3. Results

Comparisons of contaminant concentrations among developments are shown in Table IV. Table V compares measured concentrations with available health guidelines. Table VI compares measured concentrations with values published in the literature.

As seen in Table IV, geometric mean concentrations of CO_2, TVOC as hexane, acrolein, methyl ethyl ketone, benzene, and toluene were significantly higher in new development 1 (ND1) than in the old development (OD). Mean concentrations of heptane and combined xylenes were also significantly higher in ND1 than in OD. In new development 2 (ND2), only mean styrene concentration was higher than in OD. Looking at Table V, we see that geometric mean concentrations of $PM_{2.5}$, formaldehyde, and acrolein exceed recommended exposure limits for outdoor air at all three developments. Examination of individual measurements for contaminants with mean or geometric mean concentrations below the recommended exposure limits determined that no individual measurements of these contaminants exceeded recommended exposure limits (not shown).

2.4. Discussion

The geometric mean (GM) concentration of CO_2 concentration in new development 1 (ND1) is significantly higher than the GM CO_2 concentration in the old development (OD). New development 2 (ND2) had a GM CO_2 concentration higher than OD but not significant at $p<0.05$. A higher level of CO_2 in the home is an indicator that less outdoor air is being introduced into the indoor air environment (Norbäck 1995). Noted in Table III, both new developments have features to reduce unplanned outdoor air infiltration in order to lower the energy demand associated with heating or cooling new outdoor air. OD lacks these green construction elements, and the lower CO_2 concentrations in these homes suggest that more outdoor air is entering the home, which is consistent with visual observations that showed many doors and windows were dilapidated and leaked considerably. Since outdoor air contains approximately 350 ppm CO_2 (Apte 2000), the reduction of outdoor air in the home allows for less dilution of the CO_2 that is being respired by the occupants of the home.

The American Society of Heating, Refrigerating, and Air-Conditioning Engineers has generated a guideline noting that indoor air concentrations of carbon dioxide should not exceed about 700 ppm above outdoor air concentrations or a concentration of 1000 ppm (ASHRAE 2010). It has also been found that in homes in which residents complain of nocturnal chest tightness, the mean CO_2 level was 1020 ppm whereas homes with no such complaint there was a mean concentration of 850 ppm (Norbäck 1995). Despite the differences in CO_2 concentration between the developments, all three developments are below the ASHRAE standard and even below the average level that Norbäck found to be associated with negative health outcomes. Although all geometric means were below the ASHRAE guideline, 4 units in ND1 and 3 units in ND2 exceeded it.

TABLE IV

COMPARISON OF MEASURED CONCENTRATIONS BETWEEN EACH NEW DEVELOPMENT AND THE OLD DEVELOPMENT.

	Old Development			Newer Development 1				Newer Development 2			
Contaminant[2]	n	GM	GSD	n	GM	GSD	p[1]	n	GM	GSD	p[1]
Carbon Dioxide (ppm)	8	635	1.25	20	839	1.34	**0.02**	15	777	1.33	0.10
Carbon Monoxide (ppm)	8	0.31	2.64	20	0.43	2.47	0.42	15	0.44	3.32	0.49
$PM_{2.5}$ ($\mu g/m^3$)	10	17.28	4.87	18[3]	23.2	4.18	0.62	15	26.4	1.82	0.35
Formaldehyde	10	21.70	1.54	19	27.6	1.47	0.14	15	25.7	1.54	0.28
TVOC as Hexane	9	46.90	1.63	19	93.30	2.18	**0.02**	15	63.90	1.76	0.19
Propene	10	6.10	2.90	19	8.40	2.90	0.44	15	6.00	3.20	0.98
Acrolein	10	0.60	2.60	19	1.60	1.70	**0.01**	15	1.17	2.60	0.11
Acetone	9[1]	27.70	8.80	19	35.4	1.80	0.64	15	29.9	1.90	0.90
Methyl ethyl ketone	10	0.71	2.50	19	1.28	1.88	**0.05**	15	1.07	1.60	0.16
Benzene	10	0.47	2.00	19	0.75	1.60	**0.05**	15	0.56	1.35	0.46
Toluene	10	0.78	2.50	19	1.51	1.61	**0.01**	15	1.19	1.57	0.14
1,4-dichlorobenzene	10	0.50	3.90	19	0.53	2.67	0.89	15	0.49	2.74	0.95
	Old Development			Newer Development 1				Newer Development 2			
Contaminant	n	mean	SD	n	mean	SD	p	n	mean	SD	p
Vinyl acetate	10	0.66	0.41	19	0.83	0.41	0.32	15	0.77	0.27	0.45
Heptane	10	0.27	0.15	19	0.43	0.17	**0.02**	15	0.38	0.23	0.21
Ethylbenzene	10	0.31	0.15	19	0.37	0.05	0.12	15	0.36	0.04	0.22
m/p-xylene	10	0.62	0.28	19	0.81	0.14	**0.02**	15	0.75	0.13	0.14
o-xylene	10	0.28	0.08	19	0.32	0.05	**0.05**	15	0.31	0.05	0.24
Combined xylene	10	0.89	0.35	19	1.13	0.18	**0.02**	15	1.05	0.18	0.15
Styrene	10	0.30	0.15	19	0.33	0.15	0.58	15	0.43	0.06	**0.01**
1,2,4-trimethyl benzene	10	0.37	0.10	19	0.45	0.12	0.07	15	0.43	0.07	0.07

[1](Statistically significant differences ($P<0.05$) are shown in bold font.

[2]All results are reported as ppb unless otherwise specified.)

[3] Outlier removed

TABLE V

COMPARISONS TO EXPOSURE LIMITS

			Old Dev.		New Dev. 1		New Dev. 2	
Contaminant[1]	Limit	Type	GM	over?	GM	over?	GM	over?
Carbon Dioxide (ppm)	1000	ASHRAE 62.1[2]	635	No	839	No	777	No
Carbon Monoxide (ppm)	6.1	WHO Guideline[3]	0.31	No	0.43	No	0.44	No
$PM_{2.5}$ ($\mu g/m^3$)	15	EPA NAAQS[4]	17.28	**Yes**	23.21	**Yes**	26.36	**Yes**
Formaldehyde	8	ATSDR Ch. MRL[5]	21.70	**Yes**	27.55	**Yes**	25.73	**Yes**
TVOC as hexane	142	U.S.G.B.C.[6]						
Propene	500,000	ACGIH TLV[7]	6.10	No	8.40	No	6.00	No
Acrolein	0.04	ATSDR Int. MRL	0.60	**Yes**	1.60	**Yes**	1.17	**Yes**
Acetone	13,000	ATSDR Ch. MRL	27.70	No	35.40	No	29.90	No
Methyl ethyl ketone	200,000	ACGIH TLV	0.71	No	1.28	No	1.07	No
Benzene	3	ATSDR Ch. MRL	0.47	No	0.75	No	0.56	No
Toluene	80	ATSDR Ch. MRL	0.78	No	1.51	No	1.19	No
1,4-dichlorobenzene	10	ATSDR Ch. MRL	0.50	No	0.53	No	0.49	No
			Old Dev.		New Dev. 1		New Dev. 2	
Contaminant	Limit	Type	mean	over?	Mean	over?	Mean	over?
Vinyl acetate	10	ATSDR Int. MRL	0.66	No	0.83	No	0.77	No
Heptane	400,000	ACGIH TLV	0.27	No	0.43	No	0.38	No
Ethylbenzene	60	ATSDR Ch. MRL	0.31	No	0.37	No	0.36	No
Combined xylene	50	ATSDR Ch. MRL	0.89	No	1.13	No	1.05	No
Styrene	200	ATSDR Ch. MRL	0.30	No	0.33	No	0.43	No
1,2,4-trimethyl benzene	25,000	ACGIH TLV	0.37	No	0.45	No	0.43	No

[1]All values are ppb unless otherwise noted

[2]American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. ANSI/ASHRAE Standard 62.1-2010, Ventilation for Acceptable Indoor Air Quality., 2010.

[3] World Health Organization. WHO Guidelines for Indoor Air Quality: Selected Pollutants. 2010.

[4]United States Environmental Protection Agency "National Ambient Air Quality Standards (NAAQS)." 2011. <http://www.epa.gov/air/criteria.html>.

[5] United Stated Agency for Toxic Substances and Disease Registry "Minimum Risk Levels (MRLs) List." 2011. <http://www.atsdr.cdc.gov/mrls/mrllist.asp>

[6]US Green Building Council. "LEED 2009 for New Construction and Major Renovations." 2009. <http://www.usgbc.org/DisplayPage.aspx?CMSPageID=220>

[7]American Conference of Governmental Industrial Hygienists, Inc. *2010 TLVs and BEIs: Based on the Documentation of the Threshold Limit Values for Chemical and Physical Agents & Biological Exposure Indices*. Cincinnati, OH: ACGIH Publications, 2010.

TABLE VI

COMPARISON OF MEASURED CONCENTRATIONS WITH POLLUTANTS OF CONCERN FROM LOGUE, 2011.

Contaminant[1]	OD mean	ND1 mean	ND2 mean	Logue # studies	Logue mean
$PM_{2.5}$ ($\mu g/m^3$)	30.81	39.39	30.65	13	15.9
Formaldehyde	23.51	29.57	27.13	13	56.20
Acrolein	0.84	1.82	1.61	4	1.00
Benzene	0.57	0.83	0.58	16	0.78
1,4-dichlorobenzene	1.10	0.85	0.87	12	9.15

[1]All values are ppb unless otherwise noted

Significantly higher geometric mean or mean concentrations of acrolein, methyl ethyl ketone, benzene, toluene, heptane, and xylenes were measured in ND1 than in OD. Accordingly, total VOCs are higher in ND1 than OD. Motor vehicle exhaust is a source for these contaminants (Table VII). Styrene, formaldehyde, and ethylbenzene are VOCs that are associated with motor vehicle exhaust but that did not have higher concentrations in ND1 compared to OD. Mean styrene concentration was significantly higher in ND2 than in OD. Although all three developments are in the large urban environment of Chicago and would be exposed to vehicular pollutants, it is possible that differing traffic levels on roads and highways near the developments could explain the differences seen in this study.

Since motor vehicle exhaust is an outdoor source of these pollutants, higher air infiltration rates should result in higher indoor concentrations. Without a local source we would expect to see the highest concentrations indoors at OD. The new developments provide for mechanical ventilation with high efficiency filtration also provided at ND1 (Table III), however, we measured higher $PM_{2.5}$ concentrations indoors at all three developments than are typically

measured outdoors in Chicago indicating indoor sources of $PM_{2.5}$. We observed significantly higher indoor concentrations of the gaseous components of motor vehicle exhaust while seeing no significant differences in $PM_{2.5}$. Simultaneous outdoor measurements would have allowed us to better explain the contribution of motor vehicle exhaust and other outdoor contaminants to indoor air pollution, but the resources associated with this project did not allow for those measurements.

It is also possible that the higher concentrations of VOCs come from sources inside the home as they all have potential indoor sources (Table VII). VOCs, including formaldehyde, are present in many building materials and consumer products.

Three measured contaminant levels, at all three developments, exceed applicable exposure guidelines, $PM_{2.5}$, formaldehyde, and acrolein. Acrolein was the only one of these where a significant difference was seen between ND1 and OD. No significant difference was seen between ND2 and OD.

The intermediate MRL for acrolein is 0.04 ppb and the acute MRL is 3 ppb (ATDSR 2010). Acrolein can be an intense eye or upper respiratory irritant, with the irritant threshold reported as 250 ppb (ACGIH 1991), and it has been noted to exacerbate asthma in exposed children (Leikauf 2002). All three developments have geometric mean acrolein concentrations that are above the intermediate MRL, while all three have geometric mean concentrations below the acute MRL. Every unit in every development had an acrolein concentration higher than the intermediate MRL. Although this indicates that residents at these three developments may be at some risk for adverse health effects with intermediate or chronic exposures; these developments

all fall between the acute and chronic MRLs. This suggests that although there is a significant difference in acrolein levels between OD and ND1, they are at similar toxicological risks.

$PM_{2.5}$ did not have significantly different results from OD to either ND1 or ND2. $PM_{2.5}$ is most often generated from combustion processes such as vehicular exhaust, coal burning for power generation, or other industrial processes. Due to the small size of the particles, $PM_{2.5}$ can penetrate deep into the lungs, can travel long distances, and readily infiltrate into indoor environments (Pope 2006).. Accumulation of the particulate and indoor combustion $PM_{2.5}$ sources appear to be comparable between the new and old developments.

The United States Environmental Protection Agency established a National Ambient Air Quality Standard for $PM_{2.5}$ of 15 $\mu g/m^3$ as an annual average and 35 $\mu g/m^3$ as a 24-hour maximum (USEPA 2011). $PM_{2.5}$ is associated with several forms of cardiovascular and respiratory system dysfunction including: increased hospital visits, myocardial infarctions, exacerbation of existing pulmonary disease, pulmonary inflammation, chronic obstructive pulmonary disease, respiratory distress, and decreased lung function (Pope 2006). Decreasing $PM_{2.5}$ pollution by 10 $\mu g/m^3$ resulted in a 0.73 reduction in risk for non-lung cancer relater cardiovascular and respiratory disease (Laden 2006). All of the geometric means of the developments fall below the 24 hour maximum NAAQS, although all fall above the annual average. Only 2 units in OD, 3 units in ND1, and 1 unit in ND2 fall below the recommended annual average. This indicates that all three developments have similar potential health threats from $PM_{2.5}$, with exposures consistently that high having the potential to cause cardiovascular and respiratory distress. One possible explanation for this is that while residents reported there was no smoking in their unit, there could have been tobacco smoke migration from other nearby units, or visitors to the unit could have smoked during the sampling period.

Formaldehyde concentrations were not significantly different between OD and from either ND1 or ND2. Indoor sources of formaldehyde include: insulating materials, plywood, paints, textiles, wallpapers, glues, adhesives, detergents, disinfectants, varnishes, electronic equipment, insecticides, and paper products (WHO 2010). ND1 used low formaldehyde insulation that appeared effective in keeping formaldehyde levels comparable with the OD. Though many of these consumer products were likely found inside several homes, none of the developments appeared to have any of these formaldehyde-containing products in excess of the others.

The United Stated Agency for Toxic Substances and Disease Registry (ATSDR) developed an acute minimum risk level (MRL) for formaldehyde of 40 ppb, an intermediate MRL of 30 ppb, and a chronic MRL of 8 ppb (ATSDR 2010). Minimum Risk Levels represent a concentration at which there is no appreciable risk of health effects from an exposure of between 1 and 14 days for the acute MRL, between 14 and 365 days long for the intermediate MRL, and longer than a year for the chronic MRL. As formaldehyde is very soluble in water, it is rapidly absorbed in the respiratory tract upon inhalation causing irritation to the upper respiratory tract in both acute and chronic exposures. Studies have noted eye and nose irritation in humans at 770 ppb formaldehyde (WHO 2010). Chronic bronchitis has been reported in workers exposed to 360 ppb of formaldehyde for an average of 8 years. A study by the US Department of Health and Human Services revealed an average baseline formaldehyde concentration in trailers that were used to house those displaced by Hurricane Katrina of 1,040 ppb and 90 ppb when windows were opened (USDHHS 2007). Physicians treating people living in these trailers noted an increase in the prevalence of upper respiratory conditions. Formaldehyde is a group 1 carcinogen; there is sufficient evidence of cancer in animals, along with sufficient human

evidence from comprehensive studies showing an exposure-response relationship between formaldehyde exposure and deaths from nasopharyngeal cancer (IARC 2006). Geometric means of the formaldehyde concentrations in all three of the developments were below the acute and intermediate MRLs, but above the chronic MRL. In fact, all units in all three developments exceed the chronic MRL. The three developments all have similar potential toxic effects that can occur due to formaldehyde exposure. Since it is such a strong respiratory tract irritant, even these low level exposures have some potential to cause adverse health effects in these homes, particularly in at-risk populations.

There were no significant differences in CO concentrations between OD and both ND1 and ND2. Typical sources of CO in the home are poorly maintained, incorrectly installed, or poorly ventilated cooking or heating appliances that burn fossil fuels (WHO 2010). Despite the lack of outdoor air infiltration suggested by the CO_2 results, CO concentrations remain similar throughout the developments. As seen in Table III, both new developments feature vented range hoods that could assist in removing CO from possible cooking sources from the home, thereby preventing the CO levels from reaching potentially dangerous levels.

Carbon monoxide levels in these homes were well below the level of 6.1 ppm for a 24 hour exposure recommended by the World Health Organization (WHO 2010). This limit was established to minimize possible negative health outcomes from chronic exposure to CO. The geometric means of the three different developments are less than 10% of this guideline with no individual unit exceeding the guideline, suggesting that possible adverse health effects from CO in these homes would be minimal.

VOCs propene, acetone, 1,4-dichlorobenzene, vinyl acetate, ethylbenzene, and 1,2,4-trimethyl benzene were not significantly different between OD and both ND1 and ND2. Table

VII lists the sources of these contaminants in the home, these results suggest that the use of building materials that emit these VOCs were not used in the construction of the new developments.

Mean or geometric mean concentrations for propene, acetone, methyl ethyl ketone, toluene, 1,4-dichlorobenzene, vinyl acetate, heptane, ethylbenzene, combined xylenes, styrene, and 1,2,4-trimethyl benzene were more than an order of magnitude less than the applicable recommended exposure limit. Geometric mean benzene concentrations were 4-6 times less than the chronic MRL for benzene.

In a review of 77 studies, Logue (2011) compiled the data on 267 pollutants that have been analyzed in homes in the United States and similar countries. Of these 267 contaminants, nine were identified as priority pollutants based on the typical concentrations in homes and the potential health risks associated with them. This study examined five of these 9 pollutants: $PM_{2.5}$, formaldehyde, acrolein, benzene, and 1,4-dichlorobenzene. Three of these five contaminants have been identified as above the MRL and potential health risks in this study: $PM_{2.5}$, formaldehyde, and acrolein. Table VI compares the results of this study with the studies from Logue's review.

Table VI shows that in all three developments, concentrations of $PM_{2.5}$ are higher than the average of the 13 studies reviewed. Mean acrolein concentrations of ND1 and ND2 are higher than the average of the 4 studies reviewed. Formaldehyde, benzene, and 1,4-dichlorobenzene had lower average concentrations in all three developments than the aggregate studies' means. Examining the concentrations of the pollutants that posed some risk to health in this study and the concentrations of those pollutants in several studies of the developed

countries' housing reveal that the potentially concerning concentrations found in this study are similar to housing found around the developed world.

	PM2.5	formaldehyde	Acrolein	Methyl ethyl ketone	Benzene	Toluene	Combined xylenes	Heptane	Styrene	Propene	Acetone	1,4-dichlorobenzene	Vinyl acetate	Ethyl benzene	1,2,4-trimethyl benzene	
Motor Vehicle Exhaust	x	x	x	x	x	x	x	x	x					x		
Indoor Combustion	x	x	x								x					
Insulation		x								x						
Plywood		x														
Plastics																x
Paint		x		x		x						x		x	x	
Flooring		x								x		x				
Adhesives				x											x	
Pipes										x						
Paper Products														x		
Disinfectants																
Finishes		x		x		x									x	
Solvents				x		x						x				
Cleaners						x						x				
Insecticides								x					x			

Figure 1. Sources of Contaminants.

2.5. Summary and Conclusions

Old homes tend to reach a steady level of VOC contamination as the building materials likely have completed off-gassing, while in new homes the VOC concentrations may be higher because building materials have yet to completely off-gas (Park 2006). This steadying of VOC contamination in older homes indicates the comparison of air in old homes to new homes can provide a good indicator of the effects of building materials and other household products in the home. Despite the fact that the new, green homes were built to reduce air infiltration in order to

conserve energy, the air quality of the homes was not significantly different from the general housing stock.

Although several VOCs in ND1 were higher than OD, it appears that these pollutants likely came from differences in motor vehicle exhaust from nearby traffic as opposed to building materials. ND2's excess of styrene compared to OD appears as though it may be due to building material, but the concentrations are too low to be a health concern. While the higher CO_2 level of ND1 compared to OD may indicate a lower amount of fresh air entering the home from the outdoors, the health impacts of the air in the homes in the three developments appear to be similar.

The levels of $PM_{2.5}$, formaldehyde, and acrolein in the three developments were all above an applicable health based guideline, and this should be cause for concern. As all three developments have similar over-exposures to these pollutants, the green construction of the new housing does not appear to be a factor in this concern. Indeed, these pollutants are of concern in units all over the United States and developed world as evidenced by the literature compilation of Logue. These pollutants should be of special consideration when selecting and developing new building materials for homes in order to protect the health of the occupants. These pollutants should also be studied further in regards to the outdoor air contribution to air in the home.

The health impacts of moving into green housing are not simply limited to the contaminants examined in this study. Individual sources of the pollutants could vary from home to home as a result of different furniture, cleaning and other consumer products, or home décor; study of the homes prior to occupants moving in could control for this confounder. Other

confounders could include changes in access to medical care, unemployment, neighborhood-level changes and others.

This study did not measure other indoor air contaminants known to have health impact, e.g., lead-based paint, mold and moisture, allergens, and radon. Since green housing sets design parameters for water infiltration, green homes may have lower levels of mold. Building envelope elements of green housing may reduce rodent and insect infestation resulting in lower allergen levels. Green housing design should eliminate a major source of lead in homes- lead-based paint. Evaluation and mitigation of radon are also design elements for green housing. Finally, the neighborhood effects on health, e.g., exposure to intentional violence risks, proximity to parks and playgrounds, and walkability, were not examined in this study and need to be considered when examining the role of housing on health.

As green building becomes a more common choice for new homes, it is important to consider the possible health impacts of the air in the home. The use and development of building materials that are low in potentially hazardous pollutants is important to ensuring homes are not only energy efficient, but healthy for the occupants as well.

2.6. Cited Literature

Agency for Toxic Substances and Disease Registry. (1999) Toxicological Profile for Formaldehyde.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for Acrolein.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for Benzene.

Agency for Toxic Substances and Disease Registry. (2007) Toxicological Profile for Xylene.

Agency for Toxic Substances and Disease Registry. (2010) Minimal Risk Levels (MRLs) for Hazardous Substances.

Agency for Toxic Substances and Disease Registry. (2010) Toxicological Profile for Styrene.

American Conference of Governmental Industrial Hygienists, Inc. (1991) Documentation of the Threshold Limit Values and Biological Exposure Indices: Sixth Edition.

American Conference of Governmental Industrial Hygienists, Inc. (2010) 2010 TLVs and BEIs: Based on the Documentation of the Threshold Limit Values for Chemical and Physical Agents & Biological Exposure Indices, Cincinnati, OH, ACGIH Publications.

American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. (2010) ANSI/ASHRAE Standard 62.1-2010, Ventilation for Acceptable Indoor Air Quality.

Apte, M.G., Fisk, W.J. and Daisey, J.M. (2000) Associations Between Indoor CO_2 Concentrations and Sick Building Syndrome Symptoms in U.S. Office Buildings: An Analysis of the 1994-1996 BASE Study Data, *Indoor Air*, 10, 246.

Brunekreef B, Dockery DW, Speizer FE, Ware JH, Spengler JD and Ferris BG. (1989) Home dampness and respiratory morbidity in children. *Am Rev Respir Dis*, 140, 1363.

Center for Environmental Research Information. (1999) Compendium Method TO-15, Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air.

Dawson, H.E. and McAlary, T. (2009) A Compilation of Statistics for VOCs from Post-1990 Indoor Air Concentration Studies in North American Residences Unaffected by Subsurface Vapor Intrusion, Ground Water Monitoring & Remediation, 29, 60-69.

Edwards, R.D., Jurvelin, J., Saarela, K. and Jantunen, M. (2001) VOC concentrations measured in personal samples and residential indoor, outdoor and workplace microenvironments in EXPOLIS-Helsinki, Finland, *Atmos Environ*, 35, 4531-4543.

energystar.gov. (2011) Qualified New Homes, 2011.

Farrow, A., Taylor, H. and Golding, J. (1997) Time Spent in the Home by Different Family Members, *Environmental Science and Technology*, 18, 605.

Fisk, W.J., Mirer, A.G. and Mendell, M.J. (2009) Quantitative relationship of sick building syndrome symptoms with ventilation rates, *Indoor Air*, 19, 159-165.

Hun, D.E., Corsi, R.L., Morandi, M.T. and Siegel, J.A. (2010) Formaldehyde in residences: long-term indoor concentrations and influencing factors, *Indoor Air*, 20, 196-203.

International Agency For Research on Cancer. (1982) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 29: Some Industrial Chemicals and Dyestuffs.

International Agency For Research on Cancer. (2006) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 88: Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxypropan-2-ol.

Jedrychowski, W., Maugeri, U., Perera, F., Stigter, L., Jankowski, J., Butscher, M., Mroz, E., Flak, E., Skarupa, A. and Sowa, A. (2011) Cognitive function of 6-year old children exposed to mold-contaminated homes in early postnatal period. Prospective birth cohort study in Poland, *Physiol Behav*, 104, 989-995.

Laden, F., Schwartz, J., Speizer, F.E. and Dockery, D.W. (2006) Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study, *American Journal of Respiratory and Critical Care Medicine*, 173, 667.

Leikauf, G.D. (2002) Hazardous Air Pollutants and Asthma, *Environ Health Perspect*, 110, pp. 505-526.

Logue, J.M., McKone, T.E., Sherman, M.H. and Singer, B.C. (2011) Hazard assessment of chemical air contaminants measured in residences, *Indoor Air*, 21, 92-109.

M A Andersson, M Nikulin, U Köljalg, M C Andersson, F Rainey, K Reijula, E L Hintikka and M Salkinoja-Salonen. (1997) Bacteria, molds, and toxins in water-damaged building materials. *Applied and Environmental Microbiology*, 63, 387.

Maddalena R, Russell M, Sullivan DP and Apte MG. Formaldehyde and Other Volatile Organic Chemical Emissions in Four FEMA Temporary Housing Units, *Environ Sci Technol*, - 5626.

Massolo, L., Rehwagen, M., Porta, A., Ronco, A., Herbarth, O. and Mueller, A. (2010) Indoor?outdoor distribution and risk assessment of volatile organic compounds in the atmosphere of industrial and urban areas, *Environ Toxicol*, 25, 339-349.

Norbäck, D., Björnsson, E., Janson, C., Widström, J. and Boman, G. (1995) Asthmatic symptoms and volatile organic compounds, formaldehyde, and carbon dioxide in dwellings. *Occupational and Environmental Medicine*, 52, 388-395.

Park, J.S. and Ikeda, K. (2006) Variations of formaldehyde and VOC levels during 3 years in new and older homes, *Indoor Air*, 16, 129-135.

Perzanowski, M.S., Sporik, R., Squillace, S.P., Gelber, L.E., Call, R., Carter, M. and Platts-Mills, T.A.E. (1998) Association of sensitization to Alternaria allergens with asthma among school-age children, *J Allergy Clin Immunol*, 101, 626-632.

Pope III, C.A. and Dockery, D.W. (2006) Health Effects of Fine Particulate Air Pollution: Lines that Connect, *J Air Waste Manage Assoc*, 56, 709-742.

Seaman, V.Y., Bennett, D.H. and Cahill, T.M. (2009) Indoor acrolein emission and decay rates resulting from domestic cooking events, *Atmos Environ*, 43, 6199-6204.

Seppänen, O.A., Fisk, W.J. and Mendell, M.J. (1999) Association of Ventilation Rates and CO2 Concentrations with Health and Other Responses in Commercial and Institutional Buildings, *Indoor Air*, 9, 226-252.

Taskinen, T., Hyvärinen, A., Meklin, T., Husman, T., Nevalainen, A. and Korppi, M. (1999) Asthma and respiratory infections in school children with special reference to moisture and mold problems in the school, *Acta Pædiatrica*, 88, 1373-1379.

U.S. EPA. (1989) Health and Environmental Effects Document for N-Heptane.

U.S. EPA. (2010) Care for Your Air: A Guide to Indoor Air Quality, 2011.

U.S. EPA. (2011) National Ambient Air Quality Standards (NAAQS).

U.S. EPA. (2011) National Ambient Air Quality Standards (NAAQS).

US Department of Health and Human Services. (2007) Formaldehyde Sampling of FEMA Temporary-Housing Trailers.

Watson, R. (2010) Green Building Market and Impact Report 2010, Green Building Market and Impact Report 2010.

World Health Organization. (2005) Air quality guidelines - global update 2005.

World Health Organization. (2010) WHO guidelines for indoor air quality: selected pollutants.

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