Health Outcomes Attributable to Housing Conditions in the United States

BY

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DISSERTATION

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This dissertation is dedicated to my children, Tabitha and Jasper, who teach me new things every day. You will always be my greatest accomplishment.

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LIST OF ABBREVIATIONS

ACGIH American Conference of Governmental Industrial Hygienists

AHS American Housing Survey

ASHRAE American Society of Heating, Refrigerating and Air

Conditioning Engineers

ATSDR Agency for Toxic Substances and Disease Registry

CDC Centers for Disease Control and Prevention

WONDER Wide-ranging ONline Data for Epidemiologic Research

CH₂O Formaldehyde

CO Carbon Monoxide

CO₂ Carbon Dioxide

DALY Disability Adjusted Life Year

ETS Environmental Tobacco Smoke

HIPAA Health Insurance Portability and Accountability Act

HUD Department of Housing and Urban Development

IARC International Agency for Research on Cancer

IDLH Immediately Dangerous to Life or Health

MCS Mental Component Score

mm Millimeter

NCHH National Center for Healthy Housing

NIOSH National Institute for Occupational Safety and Health

NOAEL No Observed Adverse Effect Level

OSHA Occupational Safety and Health Administration

LIST OF ABBREVIATIONS (continued)

PAF Population Attributable Fraction

PCS Physical Component Score

PEL Permissible Exposure Limit

PM_{2.5} Particulate Matter 2.5

ppb Parts per Billion

ppm Parts per Million

REL Recommended Exposure Limit

RR Relative Risk

SBS Sick Building Syndrome

SF-36 Short Form-36

SLE Significant Level for Entry

SLS Stay Significant Level

STEL Short-Term Expsoure Limit

STOVE IAQ Studying the Optimal Ventilation for Environmental Indoor Air

Quality

TLV Threshold Limit Value

US United States

USEPA United States Environmental Protection Agency

WHO World Health Organization

YLL Years of Life Lost

μg/m³ Microgram per Cubic Meter

SUMMARY

This research was a cross-sectional examination of the effects of substandard housing conditions in the United States. Air sampling and interviews were performed for 85 housing units in the Chicago metropolitan area. Air metrics included concentration levels for formaldehyde, nitrogen dioxide, particulate matter, carbon monoxide, carbon dioxide, ASHRAE 62.2 compliance. Health-related quality for occupants was assessed through the Short Form-36 scale. In addition, disease and monetary burden from pediatric asthma attributed to mold and moisture exposure in homes from the American Housing Survey.

Homes that complied with the ASHRAE 62.2 standard had significantly lower levels of formaldehyde. When restricted to homes within the city limits of Chicago, there were statistically significant lower levels of formaldehyde and carbon dioxide in homes that were compliant with the standard. In general, the trend was that most contaminants were lower in ASHRAE 62.2 compliant homes.

There was no statistically significant difference in self-reported occupant health-related quality of life outcomes between homes that were ASHRAE 62.2 compliant and those that were not. Modeling for physical health resulted in asthma, age, and carbon dioxide being significant variables. Modeling for mental health resulted in nicotine, carbon dioxide, carbon monoxide, and nitrogen dioxide being significant variables. However, it appeared that nicotine variable acted as a surrogate introduction of bias into the model because the directionality

SUMMARY (continued)

was opposite than expected. When the total air contaminant load was divided into tertiles there were no statistically significant differences observed. However, there was a consistent trend that increased air contamination load resulted in lower compliance with ASHRAE 62.2, lower physical health, and lower mental health.

Estimates for the disease and monetary burden were calculated utilizing most recent data from 2008 and calculated in 2009 dollars. Mold accounted for 13,870 Disability Adjusted Life Years (DALYs) and 5 deaths. Leaks from interior sources accounted for 29,839 DALYs and 11 deaths. Moisture from exterior sources accounted for 36,198 DALYs and 14 deaths. A combined factor of moisture from interior and exterior sources accounted for 60,136 DALYs and 23 deaths. The total cost of asthma for those under 15 that may be attributed to mold and moisture in homes was almost 2.4 billion dollars.

I. INTRODUCTION

A. Background

The disease burden attributable to environmental etiologies remains unclear and an important public health concern for investigation. However, varying definitions and estimating functions have limited the scope of enumerating the true disease burden that can be linked to the environment. The first obstacle to address this concern has been to consistently define what the term environment encompasses. The definition of environment can vary based on the inclusion and/or exclusion of natural, built, social, and genetic components that determine susceptibility [1, 2, 3]. The definition is also related to the locus of control: does the individual act on the environment though engaging in an activity such as smoking? Or rather, does the environment act upon the individual such as in the case of exposure to secondhand smoke? Perhaps the true action of environment is a complex mixture of both scenarios.

These issues are further complicated by disease definitions with multiple contributing factors [1]. Asthma represents an example of the complex interaction of environmental and non-environmental etiologies. As a respiratory disease, asthma can result from numerous factors including biologic causes and external forces acting upon an individual [4, 2]. It has been difficult to definitively determine if asthma is developed through environmental exposures or if they are exacerbating symptoms of a disease caused from other mechanisms or some

combination of the two [2, 3, 4]. Therefore, defining the portion solely from one's environment that contributes to disease development is also debated in the literature. The attributable portion of diseases and health-related quality of life aspects that can be correlated to environmental factors has uncertainty that this study attempts to address.

Singular air contaminants do not exist in isolation or remain at a steady state. Rather, the term "air" is a complex mixture with a varied array of components in different phases that are always changing. This mixture of air may contain liquid, gas, solid, and phases of materials as well as biologic or viral particles [5]. Several air contaminants exist at the same period of time in the same locations depending on emission sources. Some air contaminants are reactive and change into other forms or compounds quickly or more slowly over time. Others may agglomerate to produce other arrangements with different mechanistic effects. The alterations in composition can occur several times. These properties present unique challenges when trying to quantify exposure because reactions change the composition of air contaminants especially for measurement methods that are designed to measure a single substance.

Living environments and housing directly and indirectly impact population-based health. Homes provide shelter from extreme weather elements; they are vital for physical protection. Maslow's Hierarchy of Needs calls out shelter in the first tier of physiological needs [6]. This is a conceptual model in which each lower tier must be fully achieved before moving into the next tier [6]. Before someone

can reach the highest tier of self-actualization, shelter must be secured further demonstrating the foundational importance of homes. Homes should be as healthy as possible since they are a vital component in achieving other needs in life.

There have been several instances where housing initiatives have positively impacted health [7]. Lead poisoning prevention policies have drastically reduced childhood lead poisoning rates and lead based paint exposure [7]. Ventilation, occupancy limits, and sanitation helped to control typhoid and tuberculosis outbreaks [7]. People being quarantined in their homes helped to curb the spread of diseases [7]. The most recent example is from orders to remain at home that were enacted on various governmental levels during the outbreak of the SARS-CoV-2 that led to the COVID-19 pandemic [8]. It was determined that limiting movement outside of primary living dwellings protected public health by limiting the spread of the disease [8]. Since the home environment is crucial to both individual and population-based health it is imperative that recommendations, initiatives, and actions be taken to be sure it is healthy for all. This includes ensuring it is free from any potential harmful air contaminant exposures.

Exposure to air contaminants or air pollution is defined by both the contact event as well as the duration of the contact and frequency [5]. The below pathway has been proposed to explain the process between source of pollution to health effects in living organisms: $Source \rightarrow Emissions \rightarrow Concentrations \rightarrow Exposure \rightarrow Dose \rightarrow Health Effects$ [5, p. 62]. Historically, exposure assessment studies have

support in recent literature to indicate that indoor exposures represent an important pathway based on the concentration, time, and frequency of such exposures. Indoor air contaminants may be generated through primary sources or infiltrate from ambient environments as secondary sources [9, 10, 11, 12, 13, 14, 15, 16]. Recent published literature has shown that ambient exposure is not equivalent to indoor exposure [10, 13, 16, 15, 17]. Ambient sources vary according to the contribution of contaminants in indoor environments based on ventilation into and out of the home, penetration capabilities, decay rates, and reactivity [5]. There are also primary sources of air contamination that occur in homes such as off gassing, cooking, cleaning, occupancy, pets, and numerous other variables. These factors limit modeling potential to predict indoor exposure from ambient monitoring alone.

Seasonal trends contribute to variation in air contaminants in both ambient and indoor environments [18, 19]. For instance, homes may be more likely to have their windows closed when experiencing extreme temperature events [5].

Decreased ventilation in living units could lead to changes in the air contaminant levels depending on primary sources in the home and ambient intrusion sources.

Some evidence has suggested that the effort to make homes energy efficient may unintentionally concentrate air contaminants in homes through decreased ventilation [20]. There is also considerable variation based on the heating and cooling capacity of the homes. Some homes may have centralized air

conditioning, while other homes may rely on simply opening windows to increase air flow. Increased ventilation and open windows allow for more ambient pollution intrusion. Fans may be employed in units which could result in wide dispersion of air contaminants throughout the homes instead of microenvironments with variations in concentration gradients from generating activities [5]. Fans may also dilute contaminants in the air through mechanical removal as a result of exhaust. Pressure differentials also can play a role and can vary by season, because exhaust air can create negative pressures inside living areas with respect to outdoors [21]. Air contaminant concentrations are constantly changing due to human activity patterns, sources, climate, environment, and air flow. Therefore, it can be problematic to estimate concentrations that people are exposed to in the home over time.

Air pollution has been shown to negatively impact individual health in addition to population health. In December of 2020, a tragic story emerged out of the United Kingdom. A nine-year-old girl had her cause of death attributed to air pollution [22]. She was asthmatic and lived an area where the ambient NO₂ levels consistently exceeded 21 ppb [22]. Her mother was never informed about the link to the poor air quality in the area they lived and the potential health effects even though there were reports of the young girl being in the hospital approximately 30 times in just a few years before her death [22]. Statistics have often reported large values on mortality and disease incidence and prevalence, but such large values can obscure the fact that each number represents an individual life with value

beyond pen and paper. However, this is believed to be the first time that air pollution was actually listed on an individual death certificate. This is an important milestone because it sets a precedent for future examination into how the environment contributes to mortality. It also serves as a reminder of the cost beyond monetary values.

B. Air Contaminants in Homes

Nitrogen dioxide (NO₂) is an odorous compound that exists in the gaseous phase at room temperature due to a low partial pressure [23]. The largest fraction of nitrogen oxides generated are nitric oxide, but it readily oxidizes in the atmosphere to form NO₂ [23]. The primary source for ambient NO₂ is from roadway traffic and combustion motors [23]. The main source of NO₂ in homes is from gas appliances that are either unvented or improperly vented [24, 23]. Tobacco smoke, candles, welding, and ambient concentrations also contribute to indoor NO₂ levels in homes [24]. Homes that are located in areas with higher ambient concentrations from traffic tend to have higher concentrations of NO₂ indoors as well [23]. With the presence of additional primary sources of NO₂ in homes, concentrations are higher than ambient sources alone [23]. Seasonal variation has also been well documented in homes for NO₂. Levels trend higher in colder winter months than in summer months due to the increased use of heating appliances in homes and change in ventilation rates [23].

Formaldehyde presents as a gas at room temperature that is generally highly reactive. It is colorless but has a strong odor and is a known irritant [25]. In the air, formaldehyde will break down into formic acid and CO [26]. Bioaccumulation does not occur with formaldehyde [26]. There is a trace amount of endogenous formaldehyde formed from metabolism that is generally considered negligible on health [26, 25]. However, there have been health implications associated from exogenous exposure. Major sources of formaldehyde in homes come from off gassing from binders and glues in everyday objects and materials that contain formaldehyde [23, 27]. A study of emission rates found that pressed wood with formaldehyde containing resins, permanent press fabrics, floor finish, and nail polish had high formaldehyde emission rates [28]. Latex paints and wallpapers had measurable amounts of formaldehyde emissions and merit consideration of exposure due to their large surface applications inside residences [28]. Formaldehyde is also commonly found in automobile exhaust, antiseptics, cleaning chemicals, preservatives, tobacco products, cooking stoves, and fireplaces [26, 25]. The exposure pathways can include inhalation, dermal, and ingestion, but the most important pathway of formaldehyde exposure for humans in a home environment is inhalation [26].

Carbon monoxide (CO) also has relevant home-based exposures for occupants. At room temperatures it is present as a nonirritating, colorless, odorless gas [29]. Carbon monoxide can convert to carbon dioxide (CO₂) in the air but does so over the time frame of about two months. Some microorganisms can

convert CO into CO₂ [29]. Analogous to formaldehyde, endogenous CO is present through metabolic pathways, but exogenous exposures can have negative impacts. The major source of CO in homes is from incomplete combustion [29]. Contributors to CO levels in homes are gas appliances, unvented or improperly vented gas water heaters and furnaces, infiltration of exhaust from non-electric cars or motors, and tobacco smoke [30]. The United States Environmental Protection Agency (USEPA) currently estimates that CO levels inside of homes without gas stoves range from 0.5 to 5 ppm. Homes that have gas stoves can range from anywhere to 5 to over 30 ppm [24].

Carbon dioxide (CO₂) is a noncombustible, colorless, odorless gas that is naturally occurring but has been increasing in atmospheric levels due to anthropogenic activities [31, 32]. Burning of fossil fuels, industrialized processes, and deforestation have contributed to the increase in ambient CO₂ levels globally [32]. In addition to combustion sources in the home, occupants themselves are a source of CO₂ as it is a byproduct of respiration. Some have estimated that on average one adult human can exhale an average of approximately two pounds of CO₂ daily. One study estimated that the average human between the ages of 16 and 30 years old has a CO₂ generation rate of 4.5 mL/s during low activity levels [33]. However, it has been shown that CO₂ generation increases with activity level [34]. Females produce lower levels of CO₂ during periods of similar activity levels than men [34]. It has been suggested that CO₂ is an indicator of sick building syndrome (SBS) and inadequate outdoor air supply. Occupant well-being has

been linked to CO₂ [35, 36, 37]. This has resulted in ventilation guidelines being focused on air changes and occupancy to help alleviate these nondescript general malaise symptoms [38, 7].

Particulate matter (PM) can enter the home through intrusion, but levels are also greatly impacted through occupant activates. Traffic patterns have been linked to ambient PM levels and homes located near busy roadways also show increased PM levels through infiltration. A study by Afshari, Matson, and Ekberg (2005) investigated 13 common household particle generating activities [39]. They found that maximum particle concentrations were observed within a few minutes of beginning each potential source activity and that the decay after cessation of the activity took longer [39]. This suggested that generating activities can cause spikes in exposure of PM relatively quickly and that exposure will remain elevated for long periods of time even after the generating activity has subsided. Particle size, density, and aerodynamic diameter greatly impacts particle behavior in the air including how quickly they will settle or remain suspended. Air turbulence and activity patterns can also cause particle resuspension. Smoking and combustion within the home can also contribute to PM exposure in indoor environments [40, 41]. Some research now suggests that environmental tobacco smoke can contribute up to ten times more PM in homes than that emitted from engines [41].

C. <u>Housing Programs</u>

Several governmental and nongovernmental agencies have established healthy homes programs with comparable strategies. The Department of Housing and Urban Development (HUD) created an initiative to complement their Lead Hazard Control programs in 1999 to address children's home-based environmental hazards [42, 43]. The Centers for Disease Control and Prevention (CDC) has a supplemental initiative designed to address healthy homes issues through research, surveillance, and dissemination of recommendations [44]. The National Center for Healthy Housing (NCHH) was established in 1992 as a nonprofit organization dedicated to providing scientific research and advocating for policy and programs [45]. The NCHH offers a national network of professionals demonstrating the capacity to distinguish and address the link between hazards in the home and health outcomes [45, 46]. While the USEPA does not have a separate healthy homes program, they do oversee initiatives with housing components such as radon and indoor air quality [24]. In 2009, the office of the Surgeon General released the Call to Action to Promote Healthy Homes in which they laid out the scientific evidence linking the home environment and health implications as well as the importance of improving health through improving the home [7].

D. Stove IAQ

Enterprise Community Partners was founded in 1982 to address poverty in the US [47]. Today their mission is to create affordable, safe, and healthy homes

for people to live in [47]. They introduced the Green Communities Criteria as the first national standard for green and healthy homes in 2004 [48]. Since it began, Green Communities has created over 38 thousand healthy units for low-income people [48]. Studying the Optimal Ventilation for Environmental Indoor Air Quality (STOVE IAQ project) was a research study conducted through the partnership with Enterprise Community Partners, the NCHH, University of Illinois at Chicago, and Icahn School of Medicine at Mt. Sinai in New York City. Funding for the study was made possible through the JPB Foundation. The STOVE IAQ study aimed to demonstrate the variation in concentrations of indoor air respiratory contaminants and health outcomes associated with ventilation.

A key component of the Green Communities criteria for new construction and substantial renovation work includes complying with the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) 62.2-2010 and later voluntary standard regarding indoor mechanical ventilation [49]. This voluntary consensus standard has been updated every three years in recent times. It sets the requirement for minimum ventilation to ensure proper indoor air quality in dwellings [50]. However, it has been demonstrated that many renovation projects do not meet these recommended ventilation standards. The STOVE IAQ project investigated the quantitative associations of self-reported resident health related quality of life and air quality metrics over time for the two sites.

The inclusion criteria for the STOVE IAQ study included the following: Residential units underwent renovations in accordance with the Green Communities Criteria within the past five years prior to the date of enrollment, had working gas stoves, were not restricted according to age, and were determined to be in low-income multi-family housing developments. Approximately half of the units were designed to comply with ASHRAE 62.2 while the other half met other components of the Green Communities Criteria but did not comply with this ventilation standard. Primary participants were at least 18 years of age, lived in a low-income green unit for at least four months prior to enrollment, and planned to remain in the unit for minimum of eight months from the date of enrollment. Data collection occurred at baseline, four months, and eight months after the initial enrollment in both Chicago and New York City. The primary hypothesis from the STOVE IAQ study was that housing units that were compliant with the ventilation standard would have lower levels of NO2 at eight months compared to units that were not designed to comply with the ASHRAE 62.2 standard. Additional hypotheses were that that other air contaminants (PM2.5, CO, CO₂, and formaldehyde) would be lower in the ASHRAE 62.2 compliant units and occupant health-related quality of life would be better in the compliant group.

This dissertation research was nested within the larger STOVE IAQ study, but had its own unique hypotheses, methods, analyses, and outcomes, described in this following report. The dissertation also provided additional estimates of

certain health outcomes attributable to housing conditions in the US which was outside the scope of research for STOVE IAQ. This research was approved by the University of Illinois at Chicago's Institutional Review Board under protocol #2018-1105.

II. SPECIFIC AIMS AND HYPOTHESES

This current research aimed to explore the relationship between health and housing. Specifically, this study was designed to examine the relationship between indoor air quality and health outcomes through a quantitative health related quality of life assessment. It also aimed to estimate the disease burden of housing conditions using disability adjusted life years (DALYS) and mortality estimates and associated monetization evaluations for pediatric asthma as a case study. The long-term goal of this study was to promote and support healthy homes initiatives with sound evidence demonstrating the direct and indirect health impacts of housing conditions on residents. Research has linked both indoor and ambient air contaminants to many adverse health outcomes including respiratory, cardiovascular, cancer, mortality, and others [11, 12, 14, 51, 52, 53]. Although it has been proposed that indoor air quality issues can affect quality of life indicators, there have been limited studies exploring the potential link between residential indoor air quality and quality of life measures among occupants [54].

This study was conducted utilizing primary sampling methods. Indoor air quality metrics included passive sampling for formaldehyde, NO₂, CO and CO₂. Nicotine was also sampled to control for tobacco smoke. There was active sampling conducted for PM_{2.5}. The SF-36 was used to examine health-related quality of life among participants. Burden of disease from housing conditions in

the US was estimated through assessment of mold and moisture in homes, population exposed, increased risk of disease from exposure, quantification of DALY and excess mortality, and economic estimate equations. The following three aims and associated objectives and hypotheses achieved by this study are presented below.

A. <u>Aim 1</u>

Detail the indoor air quality metrics for low-income multifamily dwellings in the Chicago metropolitan area for formaldehyde, PM2.5, CO, CO2, NO2, and nicotine. Determine if there are any differences in indoor air contaminants based upon compliance with the ASHRAE 62.2 ventilation standard.

This research contributed to the literature that measured actual concentrations of air pollutants in residences. This study provided direct measurements rather than indirect estimations for home-based exposures from outdoor air or simulations. Therefore, it was more accurate in detailing indoor residential exposures. It also explored how compliance with the voluntary ASHRAE 62.2 ventilation standard impacted air contaminant concentrations in homes.

B. Aim 2

Explore associations between indoor air quality metrics in Chicago metropolitan area dwellings and resident self-reported health-related quality of life. These models included percent compliance with the ASHRAE 62 standard for

each living unit and controlled for demographic variables of age, sex, race, ethnicity, education, income, and asthma diagnosis.

This research investigated how indoor air quality measurements in domiciles influenced health-related quality of life. It modeled resident health-related quality of life with indoor air quality metrics as predictors including compliance with the most current ASHRAE 62.2 standard. The hypothesis of an inverse relationship between indoor air quality metrics and health-related quality of life was made. There has been little published research measuring occupant health-related quality of life as it pertains to ventilation and the ASHRAE standard especially in residential settings. Most earlier studies have focused on "comfort" measurements and air changes per hour rather than quantitative health outcomes. However, this study used a psychometrically verified scale (the SF-36) to scientifically explore the differences in health-related quality of life attributable to air quality metrics and the ASHRAE 62.2 ventilation standard.

C. <u>Aim 3</u>

Quantify the burden of disease from pediatric asthma attributable to mold and moisture exposure in US homes.

This study estimated the disease burden from asthma using dampness and mold exposures as a case study. Similar studies have been performed using other data sources, but none have looked at the US housing stock using multiple household exposures. This study also employed the American Housing Survey

(AHS), which is a nationally representative dataset of the housing stock. Disease burden estimates of DALYs and mortality were monetized to explore the economic impact from asthma due to mold and moisture in US homes. The results from this study provided proof-of-concept for additional research. This procedure can be used to examine other housing exposure factors and diseases to better grasp the magnitude of how housing influences health as well as future policy implications.

III. RESEARCH STRATEGY

A. Significance

Increasingly, people are spending more times indoors. The Environmental Protection Agency's (USEPA) National Human Activity Pattern Survey estimated that people spend almost 87 percent of their time indoors with approximately 67 percent of that time spent in a dwelling [55]. This represents a meaningful proportion of contact and frequency time which could greatly impact exposure. Traditionally, most exposure assessment models have been completed in the occupational setting or through outdoor modeling of exposure. However, there has now been a shift in the paradigm suggesting that dwelling and general interior exposures may greatly impact health outcomes. The International Agency for Research on Cancer (IARC) reviewed outdoor air pollution and determined that as an exposure category it is carcinogenic to humans (Group 1) [56]. A report by the EPA estimated that indoor air could contain two to five times the level of certain contaminants found in outdoor air [57]. Not only are people being exposed for longer durations inside their homes, but they may also be exposed to greater levels of contaminants as well.

The issue of housing concerns in the United States (US) contributing to preventable disease, illness, and/or injury has become an emerging research area. In 2013, the National Center for Healthy Housing (NCHH) estimated that

approximately 40 percent or 35 million homes located in metropolitan areas have one or more health hazards present [58]. The World Health Organization (WHO) projected that approximately 4.3 million deaths worldwide were due to household air pollution in 2012 [59]. These statistics represent large portions of morbidity and mortality that need to be addressed. In general, the US is lacking indoor exposure limits for housing unlike occupational settings. There are currently only enforceable ambient air standards in the US known as the National Ambient Air Quality Standards. In 2000, a WHO working group declared that everyone has a right to healthy indoor air quality [60]. This report set out to root the topic of indoor air within the framework of human rights as well as provide support and resources for the field [60].

B. <u>Innovation</u>

Previous studies have tried to use surrogate measures for indoor residential exposure to various air contaminants [9, 61]. These estimates have then been used to try to explore a potential association between indoor air quality and health outcomes. Other studies have measured indoor air quality in dwellings but have performed alternative analyses than presented in this study [9, 62, 63]. Additionally, other studies have measured slightly different metrics of indoor air quality metrics. Studies of residences' indoor air tend to have smaller cohort sizes (compared to outdoor air studies). Therefore, additional sampling is needed.

Current literature presents limited investigation into how indoor air quality in residences can impact quality of life. This thesis is unique in that it contained a comparatively moderate sample size and collected quantitative measurements of air quality metrics inside homes with concurrent health-related quality of life outcomes from residents. The study took direct measurements of selected indoor air contaminants and represent the actual exposures experienced by the participants within the home. The study design also aimed to control for potential primary sources of indoor air contaminants between units by requiring all participants to have working gas stoves. Literature has shown that gas stoves are a meaningful contributor of indoor air pollution, so this was held constant across all participants to control for bias.

There has been little exploration into how ASHRAE 62.2 compliance can impact resident health-related quality of life. Previous research focused primarily on occupational Sick Building Syndrome for employees. Limits and guidelines for air changes per hour were introduced to alleviate complaints of general malaise and body effluent mitigation without researching specific exposures or health end points. Studies exploring how ventilation impacts occupants inside homes are generally lacking in the published literature. This current study determined how ASHRAE 62.2 compliance influenced occupant health within residential dwellings.

Limited studies have explored the environmental burden of disease attributable to substandard housing conditions in the US through DALYs, mortality, and the associated monetary impact. However, estimates have not

utilized the representative American Housing Survey (AHS) results and did not explore mold and moisture sources separately. The AHS is a representative national sampling of the US housing stock and the use of it in this study was a strength. Previous studies have used combined estimates for mold and moisture in the home, but this study examined them separately, another strength. These results support the need for programs that address housing-related health conditions in the US.

IV. METHODS

A. Indoor Air Quality Measures

Indoor air quality in dwellings was assessed through both active and passive sampling. Fine particulate matter 2.5 (PM_{2.5}) micrometers (µm) in size and smaller was collected through active sampling with a pump set to 3 liters per minute and a SKC single-stage Personal Modular Impactor. An accredited laboratory conducted gravimetric analysis of the tared filter. Passive sampling was conducted for formaldehyde, nitrogen dioxide (NO₂), carbon monoxide (CO), carbon dioxide (CO₂), and nicotine. A Telaire 7001 monitor attached to a HOBO datalogger measured CO₂ in the dwellings. The Telaire was capable of reporting measurements from 0 to 4000 parts per million (ppm) of CO₂ in the air. Formaldehyde was collected with UMEx 100 passive badges and NO₂ was sampled with UMEx 200 passive badges. These sampling techniques resulted in quantitative data.

This study utilized sampling within the homes from a fixed site. This methodology was more accurate and detailed than sampling that relied on ambient point source estimations such as distances from highways or from additional indirect modeling estimates [5]. This method still allowed for discrepancies in estimating personal exposure within the participants' homes because it was not attached to individual breathing zones and only sampled from one location in the home. However, literature has shown that area sampling from

living spaces is highly correlated to breathing zone samples and this method is less cumbersome to the participants than attaching monitors within breathing zones [64, 65, 66]. The fixed site was designated to sample within the main living area of the home to capture where participants were centrally located in the home. Care was taken to avoid dead air spaces from vents and air flow patterns, windows, and proximity to the kitchen or other areas that would have presented a prevailing point source of air contamination.

B. Guidelines and standards

Most residential indoor air quality metrics are not enforceable standards in the US. Some exclusions to this are those contaminants that are can be immediately dangerous to life such as high levels of carbon monoxide. However, there are no regulations for levels below those determined to be life threatening. This is problematic as scientific literature reports health impacts at low levels for extended periods of exposure duration. As a result, several advisory agencies have provided guidance on voluntary exposure limits. There may also be international agencies that have standards that are stricter than in the US. Guidance on contaminant levels vary based on acute and chronic health effects that have been shown from different contaminants.

There are several agencies responsible for setting benchmarks for occupational exposures in the US. The Occupational Safety and Health Administration (OSHA) is the only governmental agency that sets enforceable

standards in occupational settings. Standards are designed to protect most healthy working adult populations, but they often neglect any susceptible groups within the workforce, for example, asthmatics. They have developed permissible exposure limits (PEL) that are a time weighted average for a typical eight-hour workday [67]. Some OSHA standards may also include a short-term exposure limit (STEL) which is the maximum exposure over 15 minutes or a ceiling value which may not be exceeded [67]. The National Institute of Occupational Safety and Health (NIOSH) provides recommended exposure limits (REL) for either an eight or 10-hour work shift, of a 40-hour work week, and a 45-year lifetime of workplace exposure [67]. There may also be NIOSH STELs which are 15-minute time weighted averages that should not be exceeded as well as ceiling limits which should never be exceeded [67]. There are also determinations of limits that are defined as immediately dangerous to life or health (IDLH) set by NIOSH [67]. The American Conference of Governmental Industrial Hygienists (ACGIH) is a nongovernmental organization that establishes threshold limit values (TLVs) as recommended exposure limits in occupational settings [67]. These TLVs are designed to represent a time weighted average over an eight-hour day within a 40-hour workweek [67]. These recommendations may also include a STEL or ceiling similar to the OSHA regulations [67].

The EPA is the primary regulatory agency in the US responsible for setting standards for ambient air through the Clean Air Act under title 40 of the code of regulations part 50 [68]. The following six criteria air pollutants have been defined

in the National Ambient Air Quality Standards (NAAQS): carbon monoxide, lead, nitrogen dioxide, ozone, particulate matter, and sulfur dioxide [68]. These standards were developed in order to protect public health, especially vulnerable populations such as children, geriatrics, and asthmatics [68]. Three out of the six criteria pollutants were sampled inside Chicago metropolitan area homes for this study- CO, NO₂, and PM_{2.5}. The USEPA has set CO air contaminant limits of 9 ppm for 8 hours and 35 ppm for one hour in an effort to protect vulnerable populations [68, 69]. The standards set for NO₂ are 100 ppb for one hour and an annual mean exposure of 53 ppb [68]. The EPA has set limits for PM_{2.5} as a criteria air pollutant based on the source. There is a limit of 12 µg/m³ and 15 µg/m³ for primary and secondary pollution sources respectively [68]. There is a 35 µg/m³ limit set for primary and secondary sources combined within 24 hours [68]. However, it should be emphasized that these are once again standards set for ambient air and not for air inside dwellings.

There has been significant research conducted to demonstrate the serious health impacts of PM_{2.5}. The WHO set an air quality guideline annual mean level of 10 µg/m³ for PM_{2.5} [5]. They have also established a 24-hour mean level of 25 µg/m³ [5]. These levels are also for ambient air and not applicable for indoor air. Some research suggested that there is no actual threshold value for PM_{2.5} values in which there is no observed adverse effect level (NOAEL) [5]. If there is no true NOAEL, then it is difficult to define what a true safe exposure would constitute even at comparatively low exposures. Regulations of particulate matter are

otherwise regulated. Therefore, OSHA set a PEL of 15 mg/m³ for total particulates and 5 mg/m³ for respirable particles [70]. It should be noted that respirable particles are not entirely equivalent to the PM_{2.5} particle size definition.

The WHO released select guidelines to address indoor air contaminants. They have determined there should be a 30-minute exposure limit of 0.1 mg/m³ (80 ppb) for formaldehyde. The Agency for Toxic Substances and Disease Registry (ATSDR) suggested a minimal risk level of 8 ppb for chronic exposure to formaldehyde to protect the respiratory system [71]. Due to the probable carcinogen classification by the USEPA, they designated an inhalation unit risk of 1.3E10⁻⁵ µg/m³ (0.0104 ppb) [72]. The inhalation unit risk is the estimate of excess cancer within a population that is attributable from an exposure of 1 µg/m³ in the air over a lifetime [73]. For occupational exposure, NIOSH set a REL of 16 ppb with a ceiling of 100 ppb and an immediately dangerous to life and health (IDLH) value of 20,000 ppb. The ACGIH set a TLV of 100 ppb and a STEL 300 ppb [70]. The California Office of Environmental Hazard Assessment established their own recommendations for formaldehyde exposure. They set an acute REL of 44 ppb, eight-hour REL of 7 ppb, and a chronic REL of 7 ppb [74].

Carbon monoxide levels have been examined for ambient and indoor levels because it can be lethal at high levels of exposure with relatively few symptoms.

The WHO has determined that CO levels should not be above 6.11 ppm for a 24 period, 8.73 ppm for eight hours, 30.55 ppm for one hour, and 87.29 ppm for 15

established occupational limits for CO exposures. There is a REL of 35 ppm set from NIOSH with a ceiling of 200 ppm and an IDLH value of 1,200 ppm [70, 67]. A PEL of 50 ppm is set by OSHA [70, 67]. The ACGIH set a TLV of 25 ppm [70, 67]. This TLV was designed to protect against carboxyhemoglobinemia as the critical end point [67]. Buildup of carboxyhemoglobin in the body can be lethal [23].

The WHO recommended NO₂ limits of 200 μ g/m³ (106 ppb) for 1 hour and an annual average concentration of 40 μ g/m³ (21 ppb) for indoor environments [23]. They reported that homes having an indoor gas stove resulted in NO₂ concentrations that were 28 μ g/m³ higher on average than homes that had an electric stove [23]. Another study of Los Angeles area homes found an average increase of 10 ppb for residences with homes that had a gas pilot stove and 4 ppb that had gas ranges with electronic ignition capabilities [75]. For occupational settings, OSHA established a ceiling of 5 ppm (9 mg/m³), NIOSH set a STEL 1 ppm, and ACGIH set a TLV of 0.2 ppm [70].

A PEL, REL, and TLV of 5,000 ppm (9,000 mg/m³) was set for CO₂ [70]. There was also a 30,000 ppm STEL established from NIOSH and ACGIH [70]. An IDLH value of 40,000 ppm has been documented in the literature [76]. A typical practice suggested limits on CO₂ in indoor environments is to simply add to the background levels found in ambient air. This was the strategy employed by ASHRAE which had suggested using 700 ppm CO₂ above background levels for a concentration of 1000 to 1200 ppm in occupied areas of people with low activity

levels but have since redacted it [77]. It should be noted that using concentrations above background levels were not based on health standards and were mostly used to control for odors.

Nicotine exposures have been regulated for occupational exposures. There was a PEL, REL, and TLV set at 0.5 mg/m³ [70]. A study of US homes found that nicotine concentrations ranged from less than 1 μ g/m³ to more than 10 μ g/m³ [78]. Nicotine and environmental tobacco smoke (ETS) related air contaminants are directly correlated to the number of smokers in an area. It has been estimated that over half of the residences in countries like the US have occupants who smoke [79]. The WHO in Europe indicated that there was no known level of nicotine or ETS that was shown to be safe. No NOAEL existed for ETS or nicotine. They reported a 1E10⁻³ unit risk for cancer based on residing in a dwelling with just one smoker [79]. Therefore, they recommend no acceptable level of nicotine, especially in homes. The WHO also reported that ETS contained an average formaldehyde concentration range of 41 ppb to 285 ppb, and that smoking 20 cigarettes a day had a cumulative average formaldehyde concentration of 48,858 ppb to 105,858 ppb [79].

An important distinction must be made, occupational settings are generally not appropriate for comparison in residential settings for several reasons. First, the exposure time is different. Work exposures are typically defined as 8 hours, 5 days a week. This pattern allows for recovery time away from the exposure source. In the home exposure durations are much longer and there is no extended

period of time away from the exposure sources that would allow for recovery.

Second, the work force may generally be healthier than all occupants of a home.

Occupational exposure limits do not account for sensitive populations, such as persons with comorbidities, children, or the elderly due to the healthy worker effect. Therefore, occupational limits are unlikely to be protective enough to apply to residential exposure situations.

Caution should also be employed when extrapolating USEPA ambient limits for indoor air contaminant levels. Congress has not authorized the USEPA to establish indoor air limits under the Clean Air Act or other statutes. Therefore, limits established by the USEPA are for ambient concentrations. These limits also reflect much longer exposure times typically such as quarterly or annual averages which were much longer than our nominal sampling period inside residences, which was four days.

For the purposes of this research study and the larger STOVE IAQ study, I utilized comparison exposure values found in the scientific literature to help participants understand their own air contaminants levels. There were no comparison values for nicotine or PM_{2.5} provided. A range of 7 to 80 ppb was utilized for formaldehyde. For CO a comparison value of 6 ppm with a 15-minute maximum concentration of 87 ppm was set. Carbon dioxide had a comparison value of 1000 ppm. Nitrogen dioxide was set to 21 ppb. Table I below shows the exposure comparison values that were used for each contaminant measured. None of these comparison values for the measured air contaminants were legally

enforceable. However, these comparison values were pulled from suggested guidelines, best practices, and health outcomes supported by current scientific literature.

TABLE I:MEASURED AIR CONTAMINANTS AND COMPARISON VALUES

Contaminant	Comparison Value	
Nicotine	None	
Formaldehyde	7 to 80 ppb	
Average CO	6 ppm	
15-min CO Maximum	87 ppm	
CO ₂	1000 ppm	
PM _{2.5}	None	
NO ₂	21 ppb	

C. ASHRAE 62.2 Standard

The first ventilation standard was set at 30 cubic feet per minute per person for all buildings in 1894 [80]. This standard morphed throughout the years as the field continued to advance. The committee tasked with creating the ASHRAE 62.2 standard was formed in 1996 [81], although there were earlier ASHRAE standards for ventilation. This standard titled "Ventilation and Acceptable Indoor Air Quality

in Low-Rise Residential Buildings" sets the minimum recommended standard for ventilation in homes. Air change rate became a function of the number of occupants and the size of the home and required some form of mechanical ventilation coupled with kitchen exhaust [80]. This current study employed the ASHRAE 62.2-2010 or later version of the standard. This standard has had some noted criticisms. For example, it does not suggest variations in the standard to address activities that may generate large concentrations of indoor air contaminants, combustion appliances that are not vented, or special protections for vulnerable populations [81]. Occupancy was controlled for in this standard, but the number of bedrooms is used in the calculations to account for this, which may not always be a good predictor of the actual number of occupants.

D. Health-Related Quality of Life

The definition of health-related quality of life has not been well-defined consistently throughout the literature [82]. While health refers to disease status, it also offers some contribution the quality of life as a construct. Quality of life accounts for several other aspects outside of health as well [82]. The CDC defines health-related quality of life as the perceived physical and mental health status [83]. Other definitions also include the emotional and social functioning [84, 82]. It is the multi-faceted philosophy that explains the impact perceived health has on quality of life. This construct has become an important factor in public health research to assess other measures of health status indicators as well as impacts of

interventions [85]. Health-related quality of life offers important information that had been previously neglected when just assessing disease status or life expectancy [85, 84]. It captures multiple aspects of physical and mental health from a wide variety of outcomes rather than being restricted to one outcome variable with a narrow definition [85]. Health-related quality of life is used to inform policy decisions, individual treatment plans, and population health outcomes [82, 85, 86].

Health-related quality of life was assessed through the widely implemented scale known as the short-form 36 (SF-36) licensed through the RAND Corporation. This scale was developed through the Medical Outcomes Study [87, 88, 89]. The SF-36 assessed the following eight health constructs: limitations in physical activities, limitations in social activities, limitations in usual role activities, bodily pain, general mental health, vitality, and general health perceptions [88]. The scale also resulted in a summary score for both physical and mental health [88, 89]. The instrument was designed to for individuals 14 years old and older through self-administration or through a trained interviewer either in person or over the telephone [88].

Previously published studies have shown this scale to be psychometrically sound [90, 91]. There has been strong Cronbach's alphas and reliability coefficients reported [90, 92]. Scoring for the SF-36 was conducted through the *User's manual for the SF-36v2 health survey* (third edition) and the Optum scoring portal for QualityMetric [93]. Published studies have also shown support of the

use of SF-36 for respiratory illnesses and indoor air quality assessments [94, 95, 96]. Responses were assigned numerical values from zero to 100 and averaged for component scores; higher scores indicated a higher health status [97, 93].

E. Recruitment

This study was a cross-sectional design in which exposure data and participant responses were collected at a single point in time. Recruitment occurred on a rolling basis between November 2018 and August 2019 in the Chicago metropolitan area. All units were low-income multifamily dwellings. All units were renovated within the previous five years to comply with the Green Communities Criteria, but approximately half were intended to comply with the ASHRAE 62.2 standard while the other half were not. Both study units and control units were enrolled simultaneously until 43 study units and 42 control units were reached, respectively. Enrollment activities included flyers with contact information, engagement from property managers, community meetings, and door-door knocking. This was not a randomized sampling pool so there is a concern of selection bias. However, all residents of the properties were approached about the study. This study was not also intended to be a representative sample to larger populations, but rather designed to characterize exposures and begin to explore associations that may exist.

F. Burden of Disease from Mold and Moisture in Homes

The burden of disease from pediatric asthma from home exposures to mold and moisture was assessed through the prescribed WHO methodology and the population attributable fraction (PAF). Literature review of epidemiologic studies provided the risk estimates through relative risks and odds ratios. Monetary estimates were determined though published economic studies. These estimates were important to support policy and resource allocation decisions when considering home-based interventions.

V. CONTAMINANT CHARACTERIZATION

A. Objective and Hypothesis

Detail the indoor air quality metrics for low-income multifamily dwellings in the Chicago metropolitan area for formaldehyde, PM_{2.5}, CO, CO₂, NO₂, and nicotine. Indoor air contaminants are lower in units that are compliant with ASHRAE 62.2 standard compared to units that are not compliant.

B. Introduction

This research measured levels of specific indoor air quality metrics in Chicago metropolitan area dwellings directly. These were direct measurements and did not rely on modeling of outdoor air to estimate exposures indoors. The study focused on the presence, range, and magnitude of various indoor air quality metrics in low-income multifamily homes in the Chicago metropolitan area.

Various indoor air quality metrics impact health-related quality of life. It is widely accepted that poor indoor air quality can have negative repercussions on health, but there is great variation in the literature as to the extent and magnitude.

However, studies with direct measurement of indoor air conditions are limited by small sample sizes. The objectives of this aim were to report the presence of indoor air quality metrics within low-income Chicago metropolitan area multifamily housing units and describe how these concentrations are influenced through ASHRAE compliance. I reported the measurements of PM_{2.5}, CO₂, CO,

NO₂, formaldehyde, and nicotine measured inside of the residences. The <u>approach</u> included field sampling of air inside low-income multifamily Chicago metropolitan area dwellings. These samples were then analyzed either through data logger downloads or through the accredited laboratories. Ventilation experts were contracted to determine ASHRAE compliance in enrolled housing units.

C. <u>Justification and Feasibility</u>

Indoor air contaminants have been shown to have many adverse health effects for residents. These health effects can range from both chronic and acute depending on the exposure levels and duration and mechanism of toxicity. The majority of previous scientific literature published regarding the health effects from air contaminants were from occupational settings, relied on ambient exposures, or generated estimates from modeling. One modeling study in California found that 62 percent of occupants were overexposed to NO₂, 9 percent were overexposed to CO, and 53 percent were overexposed to formaldehyde in homes with gas stoves [98]. Gas stoves resulted in a median maximum one hour exposure increase of 100 ppb for NO₂, 3,000 ppb for CO, and 20 ppb for formaldehyde [98]. These models assumed that there was no local exhaust ventilation above the stoves in the home. Additional research also reported that cooking times and gas appliances influenced indoor air quality and the effects may be compounded in buildings with multiple units [99].

Ambient air and indoor air could present very different exposure composition to air contaminants. Indoor air is composed of secondary intrusion pollution from ambient sources, but also from primary sources indoors.

Occupants themselves can produce air contaminants such as particulates and CO₂. Human activities within residences also contribute to pollution inside such as cooking, smoking, cleaning, candles, running appliances, etc. Housing components and furnishings can also contribute to indoor air contaminants through off gassing. Stoves that use natural gas as an energy source are a large contributor to indoor air contaminants. It has been estimated that between 33 percent of homes in the US currently use natural gas stoves [100, 101]. These stoves have been found to generate NO₂, CO, CO₂, and formaldehyde [98].

Additionally, approximately 42.8 percent and 46 percent of US homes use natural gas for home heating and water heating respectively [101].

Studies have shown a wide range in indoor NO₂. In North Carolina, homes reported NO₂ concentrations of 10.8 ppb before weatherization work and 5.5 ppb after with median concentrations of 2.2 ppb for both time periods [102]. A study from three areas in Europe found median NO₂ levels ranging from 5.79 ppb to 23.87 ppb [103]. In a large study of English homes there was a geometric mean of 11.60 ppb in the kitchen, 6.33 ppb in the bedroom, and an ambient average of 11.11 ppb [104]. An international comparative study found that personal exposures were more strongly correlated to indoor NO₂ levels compared to ambient levels [105]. In Quebec City, Canada, a study of 96 homes reported a

geometric mean concentration of 16 ppb for NO₂ [106]. A Chicago study also reported low correlations between personal NO₂ levels and ambient levels [107].

People are primarily exposed to formaldehyde through the air and inhalation pathways. Formaldehyde in the air decomposes into formic acid and CO [26]. Urban areas have been found to have higher concentrations of formaldehyde in the air than rural areas, mostly due to combustion sources from traffic and industrial components [26]. Studies have also shown that formaldehyde levels are higher in indoor environments compared to ambient levels because it is commonly found in multiple household products [26]. In England, a geometric mean of 17.76 ppb was reported [104]. A study of 36 homes in New York found an average formaldehyde concentration of 9.7 ppb in the winter and 16.7 ppb in the summer [108]. In Victoria, Australia 80 homes were measured over four time periods to find an average concentration of 12.6 ppb with a maximum of 111 ppb [109]. In North Carolina, a study of 69 homes that underwent weatherization work found an average concentration of 22 ppb both pre and post renovation work with median concentrations of 17 ppb before the weatherization work and 15 ppb after [102]. A group of 50 homes with different stages of pre- and post-green renovation projects and a control subset with no renovation work were sampled in Cincinnati, Ohio to find a median concentration of 20 ppb [110]. Research of 96 Quebec City, Canada homes reported a geometric mean of 7 ppb of formaldehyde [106]. Ten homes in Spain reported a range of 8 ppb to 30 ppb of formaldehyde in living rooms [111]. The WHO has previously

estimated the average concentration in conventional homes to be between 24 ppb and 49 ppb [79]. A way to control formaldehyde inside homes is to have efficient ventilation and air changes [112, 113]. However, it has been suggested that this relationship may not be directly linear [113].

The study of North Carolina homes showed an average CO concentration of 0.38 ppm before weatherization work and 0.21 ppm after weatherization work with median concentrations of 0.04 ppm at both times [102]. A study of over 830 homes in England reported a geometric mean of 0.34 ppm in bedrooms and 0.41 ppm in kitchens [104]. A study of 10 US homes reported an average of 0.85 ppm [114]. Carbon monoxide levels have also been shown to have seasonal variation in homes [104]. Levels of higher CO have also been associated with the presence of a gas cooking stove [104].

Most indoor exposure assessments of CO₂ have occurred in workplace office settings. However, there have been some published results from homebased CO₂ exposure. North Carolina homes reported average CO₂ concentrations of 799 ppm in homes before weatherization work and 690 ppm after weatherization work with median concentration of 696 ppm and 670 ppm respectively [102]. A small study of ten homes across four states reported an average CO₂ concentration of 663.2 ppm [114].

A New York City study of 38 homes found a $PM_{2.5}$ concentration of 20.9 $\mu g/m^3$ in winter and 19 $\mu g/m^3$ in summer [108]. In North Carolina homes had an average concentration of 27 $\mu g/m^3$ before weatherization work with a median of 10

μg/m³ and an average of 21 μg/m³ with a median of 7.5 μg/m³ after the weatherization work [102]. A median PM_{2.5} concentration of 41 μg/m³ was reported in approximately 50 Cincinnati, Ohio homes over 3 years during a green renovation initiative [110]. A review of indoor air quality in social housing found that occupants may be disproportionally exposed to higher levels of PM_{2.5} [115].

Many indoor air quality studies that focus on conducting direct measurements inside residential dwellings had small sample sizes. As shown in the studies cited above, the majority of the study populations are under 100 homes for direct measurements inside homes. Some in the literature even report on cohorts of 10 homes or fewer. A study examining indoor air quality metrics in homes that were rehabbed either through conventional means or to meet a green criterion in Boston only included 37 participants [116]. A study in Alexandria, Egypt only had a sample of 15 homes to quantify PM_{2.5}, CO₂, and ventilation rates [117]. The study in New York City engaged 46 participants to conduct personal exposure to air contaminants as well as inside the home and outside the home [108].

D. Research Design

Study data were collected from 85 nonrandomized low-income multifamily
Chicago metropolitan area residences at a single point in time between November
2018 and August 2019. Sampling was conducted during four consecutive days
that included two weekdays and two weekend days to account for resident activity

pattern variation. Approximately half (42) of the units were rehabbed within the last five years with elements of the Green Communities Criteria, but not in accordance with the voluntary ASHRAE 62.2 standard. The other half (43) of the units were rehabbed with elements of the Green Communities Criteria and were intended to comply with ASHRAE 62.2.

E. Methods

Data were collected using active sampling, passive sampling, real time data loggers and structured health interviews. Environmental monitoring measured PM_{2.5}, CO₂, CO, NO₂, formaldehyde, and nicotine within the dwellings. Samplers were located about 3-6 feet above the floor in the main living quarters of the dwellings. Residents were asked to refrain from opening windows to standardize ventilation metrics, as well as not to smoke inside, because the study was focused on gas stoves. Bathroom doors were left open when not in use to ensure ventilation measures were operating as intended within the units. Meaning that bathroom exhaust fans were able to draw air from the rest of the dwelling unit.

Active sampling for PM_{2.5} was conducted using a Personal Modular Impactor (PMI) attached to a SKC Aircheck XR 5000 pump set to operate at a nominal flow rate of 3.0 liters/minute. Flow rates were calibrated using a rotameter attached to a calibration cap before and after each field sampling event. The pumps were also calibrated weekly with a DryCal as a primary calibration method to ensure each was operating at the proper flow rate. Every PMI was

assembled with a 37-millimeter pre-weighed filter at the accredited Wisconsin Occupational Health Laboratory (WOHL). Gravimetric analysis at the WOHL reported weight of PM_{2.5} inside the housing units. These gravimetric weights were then converted into concentrations by the known volume of air pumped through each filter.

Passive badges were utilized to determine levels of NO₂, formaldehyde, and nicotine inside the housing units. Formaldehyde was quantified with the UMEx 100 passive sampler badges with an accuracy of five parts per billion to five parts per million (ppm) plus or minus 25 percent. This was achieved through analysis at the WOHL in accordance with the International Standard for Determination of Formaldehyde-Diffusive Sampling Method (ISO/FDIS) [118, 119]. The UMEx 100 samplers had a limit of detection between 200 ppb for 15 minutes and 0.2 ppb for 7 days [119]. The diffusion sampling rate between one to seven days was 20.4 milliliters per minute (ml/min) at air velocities less than five centimeters per second (cm/s) [119]. The samplers were individually contained in a sealed aluminum pouch prior to use and during shipment to minimize contamination. All formaldehyde samplers were stored in a freezer at less than four degrees Celsius and were transported via ice packs to ensure sensor stability in accordance with the manufacture's specifications.

Similarly, NO₂ was sampled through passive sampling badges. The UMEx 200 passive samplers measured NO₂ concentrations through ion chromatography with conductive detection comparable to OSHA Method ID-182 [120].

Quantification was conducted at the WOHL to determine the concentration of NO₂ inside the homes enrolled in the study. These samplers were determined to have a range of 0.051 to 8.5 ppm plus or minus 27 percent through this methodology [120]. The UMEx 200 badges had a sampling diffusion rate of 17.3 ml/min with a standard deviation of 11.5 percent [121]. Ambient storage temperature was utilized in accordance with the manufacture's specifications. Each NO₂ was packaged in a sealed individual aluminum pouch prior to and after sampling.

Nicotine was collected through the passive diffusion method established by Hammond and Lederer (1987) [122]. The sampler is composed of an altered 37-millimeter (mm) cassette, a glass fiber filter coated with Teflon and treated with a sodium bisulfate solution, a supportive filter pad, a diffusion screen, and a removable cap [122]. Samples were analyzed by gas chromatography at the Johns Hopkins School of Public Health's Secondhand Smoke Exposure Laboratory. The nicotine badges sampled at an approximate diffusion rate of 25 ml/min [122].

Carbon dioxide was analyzed using a Telaire 7001 device attached to a U30 HOBO datalogger with a CABLE-2070. The Telaire devices used patented absorption infrared technology to quantify CO₂ as an air contaminant [123]. The full sampling set-up was able to capture readings from 0 to 4000 ppm for CO₂, an accuracy of plus/minus 50 ppm or five percent of the reading, and a repeatability of plus or minus 20 ppm [123]. The sampler collected one reading every 30

seconds in order to maximize the number of readings inside the participants' homes for each 4-day sampling period.

The Lascar EasyLog (EL-USB-CO) was used to sample for CO within the study homes. These data loggers were able to hold a maximum of 32,510 readings [124]. The EL-USB-CO boasted a measurement range of three to 1,000 ppm of CO with an accuracy of plus or minus seven ppm [124]. These samplers were also operated at 30 second intervals in the field to ensure the most data readings for the four-day sampling periods.

Compliance with the ASHRAE 62.2 ventilation standard was determined through the use of a hired contractor, Elevate Energy, with expertise in these operations. Dwelling ventilation performance testing was conducted to measure volumetric air flow rate. Flow hoods measured exhaust air flow rates for bathroom exhaust fans if they were present. Duct leakage, unit leakage, and pressure differentials were calculated for each unit. Housing units were given a blower door test to estimate unit infiltration rate in cfm at 50 Pascals pressure. Leakage was measured through duct testing fans with an air flow accuracy of plus or minus five percent. Pressure gauges measured differences with a sensitivity of 0.1 Pascals and an accuracy of plus or minus one percent of the result or 0.5, whichever was greatest. Interstitial pressure from hallway or exterior walls was assessed with pressure gauges as well. The flow hoods measured the airflow with an accuracy of plus or minus five percent. The square footage of each unit was calculated from floorplans supplied from building managers. Other variables

recorded were number of bedrooms, ceiling height, perimeter length, number of stories, number of exterior walls, and number of walls shared with other units in the building.

Data collected from each unit were used to calculate ventilation statistics. These included unit air exchange rate, the cubic feet per minute at 50 Pascal divided by the building envelope in square feet (CFM₅₀/sfbe), and the percent compliance with the ASHRAE 62.2 standard [50]. Equation 1 from ASHRAE gives the total ventilation required to meet the 62.2 standard, where Ω_{tot} is the required ventilation rate in cubic feet per minute, A_{floor} is the unit floor area in square feet, and N_{br} is the number of bedrooms [50]. This value was then compared to the results reported by the ventilation dwelling tests to give a percent of compliance. The percent compliance calculation is shown in Equation 2.

Equation 1

$$Q_{tot} = 0.03A_{floor} + 7.5 (N_{br} + 1)$$

Equation 2

These analyses had the following two unique objectives: to examine the data as a whole to describe actual measured concentrations of indoor air contaminants in low-income multifamily Chicago metropolitan area housing units

and explore how these concentrations varied based on compliance with the ASHRAE 62.2 ventilation standard. A sub analysis was performed to examine classification definitions for compliance with the ASHRAE 62.2 standard.

F. Data Analysis

The accredited WOHL reported air mass concentrations for PM_{2.5} in micrograms of particulate matter per cubic meter of air. The laboratory reported concentrations of CH₂O and NO₂ as ppb. Both CO and CO₂ were reported in ppm through the Telarie/HOBO sampling apparatus and Lascar devices, respectively. Nicotine and PM_{2.5} were reported as µg/m³.

All environmental concentrations were imported into SAS 9.4 for data analyses. Descriptive statistics were developed to report the presence and magnitude of air quality contaminants in residential units in the Chicago metropolitan area. Tests were conducted to explore data normality. Parametric and non-parametric tests were utilized to see if compliance with the ASHRAE 62.2 standard influenced concentrations of the air contaminants in enrolled units.

Transformation of raw data was explored utilizing the log normal and log base 10 transformations to achieve more normal distributions.

Environmental concentrations were examined to characterize exposure of participants. Exposures were considered for all units enrolled in the study as well as separately based on compliance groups. Compliance with the ASHRAE 62.2 standard was considered based on design intent, any positive percent

compliance, 100 percent compliance, and the exploration for a new effective percent compliance threshold.

G. Results

Some air quality contaminants did not have levels reported for all the units. There were either field errors, sampler failure, quantification errors at the laboratories, or levels were below the limit of detection for analysis method. The few missing values were imputed using averages from all the home visits. One missing PM_{2.5} result could not be ascertained as there was no additional follow-up in the unit; there were no Phase 2 or 3 data to estimate indoor PM_{2.5} levels inside this unit that may have been comparable during Phase 1. One NO₂ sample was missing a value for this Phase 1 study so the average was taken from the subsequent other phases to fill in the missing data. There were 60 nicotine samples that were under the limit of detection. This meant that measurable nicotine levels were only present in 25 of the homes surveyed in the Chicago metropolitan area. Results that were reported lower than the limit of detection were replaced with values of half of the limit of detection. These methods aligned with the reports from the Second-Hand Smoke Exposure Laboratory that completed the quantitative analysis at Johns Hopkins University School of Public Health.

Table II below depicts the general descriptive analysis and range of air contaminant concentrations in all 85 units in the Chicago metropolitan area, which

for this study included four zip codes: 60622, 60647,60653, and 60472. The results for the individual zip codes are presented in Tables II-VI respectively. There were 39 homes sampled in zip code 60622, 23 homes in 60647, six homes in 60653, and 17 homes in 60472. These results show the general distribution of indoor air contaminants within the homes sampled in the study.

As presented in the tables above, there was minimal variation in the contaminant concentrations among the zip codes. All homes were in the Chicago metropolitan area, but three zip codes were within the Chicago city limits. All the homes were in metropolitan areas and were located less than two miles from expressways. However, it is possible that those within the city boundaries were in areas with higher ambient pollution concentrations. While previous literature has shown that ambient air is not equivalent to indoor air exposures in the home, it is.

TABLE II: DESCRIPTIVE STATISTICS FOR ALL UNITS

Contaminant	AM	SD	GM	GSD	Min	Max
CH2O (ppb)	19.59	8.95	17.78	1.55	7.20	43.00
NO2 (ppb)	25.96	15.49	23.00	1.62	5.20	100.00
PM2.5 (ug/m3)	20.69	19.35	15.91	1.98	3.20	120.00
COmean (ppm)	0.40	0.58	0.13	6.97	8.83E-5	3.19
COmax (ppm)	4.92	3.74	3.13	3.76	0.02	19.43
CO2 (ppm)	791.72	306.14	735.43	1.50	159.14	1955.79
Nicotine (ug/m3)	0.53	1.54	0.08	5.08	0.03	10.27

TABLE III: DESCRIPTIVE STATISTICS FOR ZIP CODE 60622

Contaminant	AM	SD	GM	GSD	Min	Max
CH2O (ppb)	21.4	8.82	19.80	1.49	9.10	43.00
NO2 (ppb)	26.68	14.19	24.32	1.52	5.60	100.00
PM2.5 (ug/m3)	18.52	10.21	16.31	1.65	6.90	48.00
COmean (ppm)	0.35	0.42	0.15	4.93	6.8E-4	1.68
COmax (ppm)	5.02	3.96	3.57	2.75	0.05	19.43
CO2 (ppm)	829.43	254.36	787.23	1.43	159.14	1539.17
Nicotine (ug/m3)	0.46	1.82	0.05	3.95	0.03	10.27

TABLE IV: DESCRIPTIVE STATISTICS FOR ZIP CODE 60647

Contaminant	AM	SD	GM	GSD	Min	Max
CH2O (ppb)	19.3	9.56	17.41	1.57	7.2	42.00
NO2 (ppb)	28.22	19.60	24.17	1.70	11.00	100.00
$PM_{2.5} (\mu g/m^3)$	18.34	23.87	12.47	2.25	3.20	120.00
COmean (ppm)	0.67	0.88	0.22	8.47	0.00	3.19
COmax (ppm)	5.99	3.77	4.13	3.82	0.02	15.47
CO2 (ppm)	848.51	404.14	768.80	1.58	224.51	1955.79
Nicotine (µg/m³)	0.46	1.18	0.08	5.11	0.03	5.38

TABLE V: DESCRIPTIVE STATISTICS FOR ZIP CODE 60653

Contaminant	AM	SD	GM	GSD	Min	Max
CH2O (ppb)	14.18	6.47	13.16	1.51	8.10	26.00
NO2 (ppb)	17.67	4.80	17.09	1.33	11.00	24.00
$PM_{2.5} (\mu g/m^3)$	10.90	4.13	10.26	1.46	6.40	17.00
COmean (ppm)	0.10	0.11	0.04	10.94	0.00	0.31
COmax (ppm)	2.01	1.51	1.39	3.01	0.20	4.02
CO2 (ppm)	626.86	231.24	569.10	1.74	189.32	843.42
Nicotine (µg/m³)	0.03	0.00	0.03	1.00	0.03	0.03

TABLE VI: DESCRIPTIVE STATISTICS FOR ZIP CODE 60472

Contaminant	AM	SD	GM	GSD	Min	Max
CH2O (ppb)	17.75	8.64	15.89	1.63	7.2	36.00
NO2 (ppb)	24.19	14.47	20.99	1.75	5.20	72.00
PM _{2.5} (µg/m³)	33.06	27.27	25.04	2.12	10.00	100.00
COmean (ppm)	0.24	0.34	0.07	7.70	8.0E-4	1.02
COmax (ppm)	4.24	3.26	2.10	6.42	0.02	10.20
CO2 (ppm)	686.58	258.92	648.56	1.40	390.05	1369.84
Nicotine (µg/m³)	0.95	1.53	0.28	6.15	0.03	6.32

possible that ambient air contaminants influence indoor air quality through infiltration. Statistical analyses were run to see if any significant differences existed among the zip codes for all the air contaminants sampled. There was one significant difference (p<0.05) reported for PM_{2.5} concentrations between 60622 and 60472. Homes in zip codes 60472 had statistically significant higher concentrations of PM_{2.5} than those located in the 60622 zip code.

The homes in the study were also categorized into two groups: those located within City of Chicago limits and those outside of city limits to control for geographic location. In this analysis, the sample sizes were 68 homes within city of Chicago limits and 17 outside. Statistically significant results remained for PM_{2.5}, but also included statistically significant higher concentrations of nicotine in homes outside of the city of Chicago. There were statistically significant lower levels of CO₂ in homes outside the city of Chicago.

As the tables above indicate, most of the variables were log normally distributed. Formaldehyde, NO₂, PM_{2.5}, CO mean, CO max, CO₂, and nicotine kurtosis and skewness values indicate that the raw data has a positive skew distributed to the right. The raw data were also log transformed which is common practice for environmental results in order to try to achieve a normal distribution for the results to utilize alternative statistical analysis methods. The histograms of both the raw and transformed contaminant concentrations are found in Appendix A

Most normality trends were improved after transforming the data as displayed in Appendix A. Some concentrations were even transformed to have statistically significant results for the normality tests after the transformation. While none of the normality tests were greater than 0.05 for NO₂, the Kolmogorov-Smirnov test was close at 0.04. None of the transformed formaldehyde tests for normality were greater than 0.05, but the Shapiro-Wilk test was equal to 0.05 For PM_{2.5}, the tests for normality of Shapiro-Wilk, Kolmogorov-Smirnov, Cramer-von Mises, and Anderson-Darling all had p values greater than 0.05 after transforming the data. The results for both COmean and COmax show that the skew trended left after transformation. Nicotine was difficult to analyze because most of the results were below the limit of detection. Since imputation of the missing nicotine concentrations were all set to half of the limit of detection the distribution will be pulled to this value. Percent compliance with the ASHRAE ventilation standard was also positively skewed; however, since approximately half of the units had

zero compliance based on the study design and quantitative verification, log transformation was not possible.

Using the comparison values enabled a determination of whether occupants were potentially overexposed within their living environments, except for indoor exposure to PM_{2.5} or nicotine in homes (which had no comparison value). All homes had measurable levels of PM_{2.5} with the exception of one home in which the sampling apparatus failed. Twenty five out of the 85 homes in the metropolitan Chicago homes were above the nicotine limit of detection, while 60 were below. This resulted in a nicotine detection rate of 29 percent in these homes sampled in this defined geographic region. None of the homes were above the recommended comparison values for formaldehyde upper level of 80 ppb; however, all the homes exceeded the lower recommendation of 7 ppb. Depending on the recommended guidelines being utilized it is possible that 100 percent of the homes sampled were overexposed to formaldehyde. When the CO levels were examined, none of the homes were over the mean or the 15-minute maximum value. However, 16 of the homes did exceed the comparison value for the average CO₂ levels or almost 19 percent of the homes in this study.

The primary contaminant of interest in this study was NO₂ as all homes enrolled at baseline had working gas stoves. Gas stoves and appliances are known emission sources of NO₂ especially in indoor environments. Out of the 85 total homes sampled, 47 exceeded recommended level of NO₂ in the home. This meant that approximately 55 percent of the units sampled were overexposed to

NO₂ within the home according to the comparison value. While it is important to explore these levels and look for potential overexposure, it should be reiterated that these levels are not enforceable. Therefore, only inferences of implications for these exposures can be made.

Of the 85 homes enrolled, 77 completed the ventilation testing (over 90 percent of the enrolled homes). Of the homes intended to comply with the ASHRAE standard, 40 out of the 43 units completed the ventilation testing. However, only 35 of these units (or 93 percent) showed any level compliance through continuous mechanical ventilation in bathroom exhaust. The percent compliance ranged from 22 percent compliant to 383 percent compliant. The mean compliance among this group was 87 percent with the ventilation standard. Only 70 percent of units designed to comply with ASHRAE 62.2 met the 70 percent cut-point definition. In the comparison group, 37 homes out of 42 completed the ventilation testing. In this case all but one value showed zero compliance with the ASHRAE 62.2 standard. This was expected as there was no mechanical ventilation in these homes, and they were not designed to comply with the standard. There was one home in this comparison group that tested at 68 percent compliant even though renovation work was not intended to comply. Table VII shows the summary ventilation statistics from the units enrolled in the study.

Of the 77 units that completed ASHRAE 62.2 compliance testing, 36 had some positive percent compliance ash shown in Table VII. The quantitative results from blower door testing were used to define the group classification. The

working definition of an ASHRAE compliant unit for this research was any unit with any positive percent compliance with the ventilation standard regardless of the value or the design intent during green renovation. For coding purposes, 1 indicated the ASHRAE 62.2 compliant group while 2 indicated no compliance with the ventilation standard. The sample size was 77 total units that were tested for compliance using the blower door method. Of these 77 units 36 were compliant with the ASHRAE standard and 41 were not compliant with the standard at all. The distributions of the air contaminants by degree of ASHRAE compliance are shown below in Figures 1-6.

While the boxplot figures show the general distribution of indoor air contaminants in Chicago metropolitan area housing units by ASHRAE compliance, they do not show any statistically significant differences. To explore these potential differences between the two groups, the raw data were examined through the non-parametric Wilcoxon Scores test because the data were not normally distributed. The two compliance groups were independent of each other.

Table VIII shows the average concentrations for both compliance groups as well as the respective p values. Two-tailed p values were used because previous scientific literature has not conclusively demonstrated how ventilation compliance impacts indoor air contaminants in homes. Formaldehyde was the only contaminant that had a statistically significant difference between the two groups. Formaldehyde and CO₂ levels were generally lower in the ASHRAE

TABLE VII: UNITS THAT WERE TESTED FOR ASHRAE 62.2 COMPLIANCE

Units	Units Tested	Minimum Compliance (%)	Maximum Compliance (%)	Units that were compliant
Total	77	0	383	36
ASHRAE Designed Non-ASHRAE	40	0	383	35
Designed	37	0	68	1

compliant group, but NO₂, PM_{2.5}, COmean, and COmax were higher in these units when comparing the medians. Formaldehyde, PM_{2.5}, and CO₂ were generally lower in ASHRAE compliant units and NO₂, COmean, and COmax were higher in ASHRAE homes when comparing mean concentrations. Average nicotine levels and the median concentrations were the same between ASHRAE compliant and non-complaint units; there was no statistically significant difference observed.

The data were also log transformed in order to achieve a more normal distribution pattern. Parametric statistical tests tend to be stronger in their calculation power and are considered preferred if the data fit normality. Therefore, a simple t-test was also employed to examine if any statistically significant differences existed for air contaminants between the two compliance groups using the log transformed data. Each contaminant was tested independently for variance between the groups. Table IX shows the t-test results when comparing the transformed data between both groups.

These results listed below in Table IX which contaminants were statistically significant between the two ASHRAE compliance groups. Once again, formaldehyde was the only air contaminant that had statistically significant differences between the two ASHRAE compliance groups. In this case, formaldehyde levels in ASHRAE compliant units were significantly lower than in non-compliant ASHRE units. None of the other air contaminants showed statistically significant differences. However, it is important to note that nicotine levels displayed no statistical difference between the two groups and that the group variances were determined to be equal. This is important when considering nicotine as a potential confounder for indoor air contaminants between these two groups.

Due to the potential of environmental tobacco smoke to act as a confounder, nicotine differences between the groups were investigated further. A Chi-Square Test was used to determine if there was an association between ASHRAE compliance and nicotine. Raw numerical nicotine data were transformed into binary categories either above the limit of detection or below. The null hypothesis was that ASHRAE 62.2 compliance and presence of nicotine were independent. The alternative hypothesis was that ASHRAE 62.2 compliance and presence of nicotine were not independent. The Chi-Square resulted in a p value of 0.17 in which the null hypothesis was accepted. There was no statistically significant association detected between compliance and presence of nicotine.



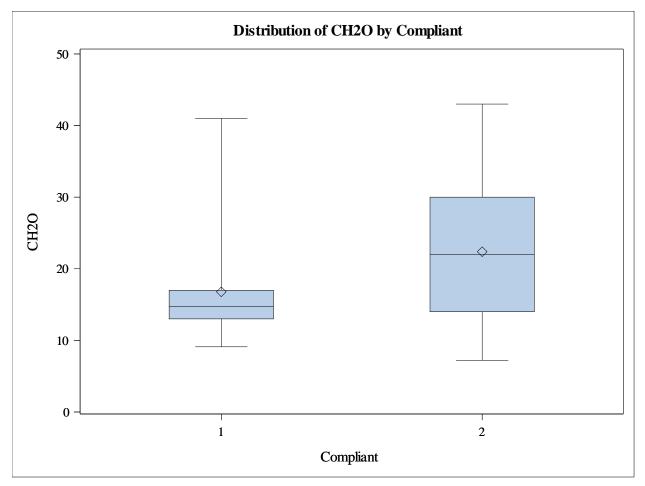
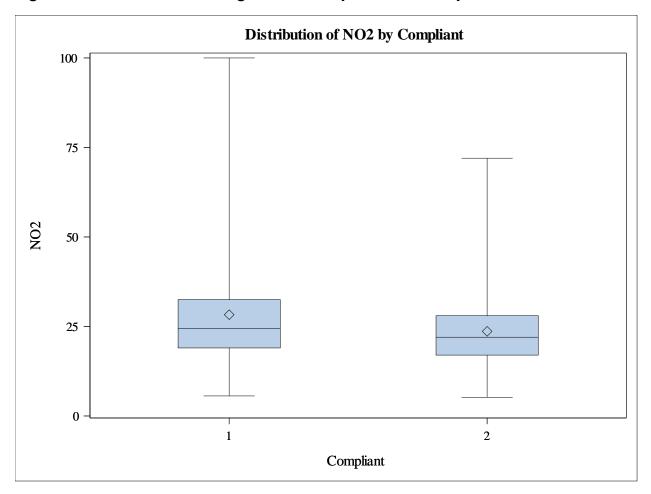


Figure 2: Distribution of nitrogen dioxide by ASHRAE compliance





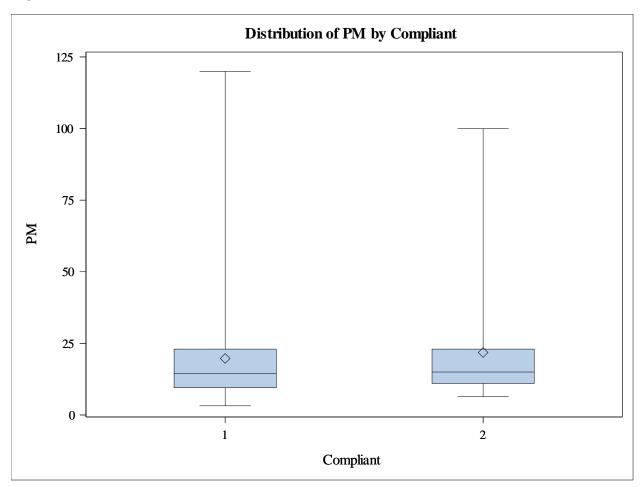
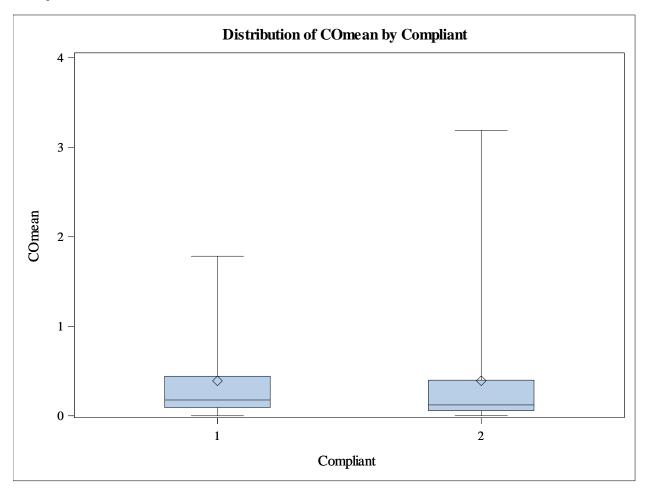


Figure 4: Distribution of the mean carbon monoxide levels by ASHRAE compliance



: Distribution of 15-minute maximum carbon monoxide levels by ASHRAE compliance

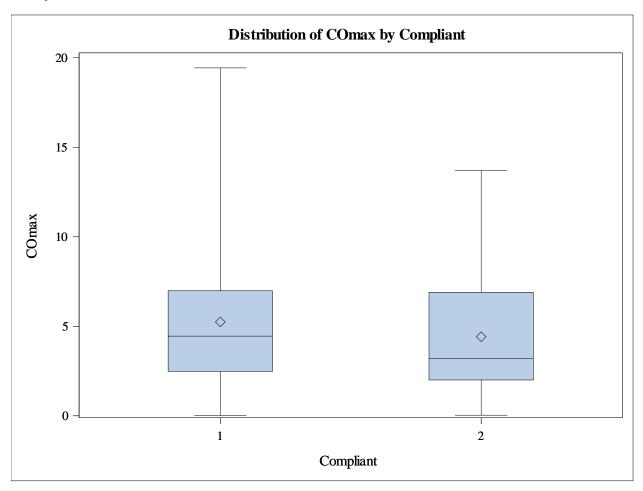


Figure 5: Distribution of carbon dioxide by ASHRAE compliance

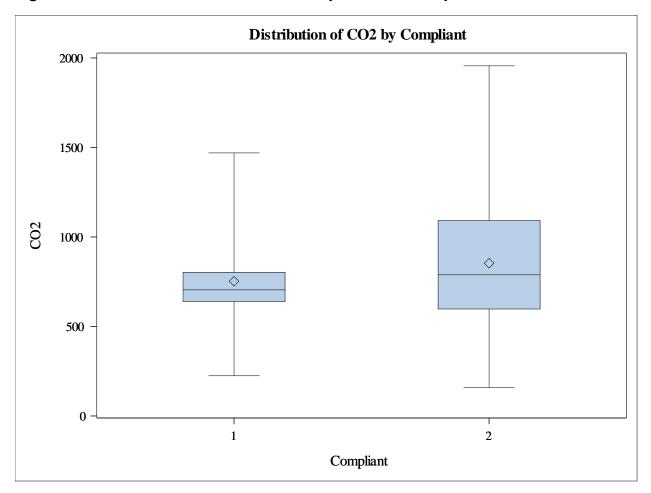


TABLE VIII: AVERAGE CONTAMINANT CONCENTRATION BY ASHRAE COMPLIANCE

Contaminant	Compliant	Non-	P values	Significant
	(n=36)	Compliant		
		(n=41)		
Formaldehyde	16.74	22.10	0.02	Yes
(ppb)	10.74	22.10	0.02	165
NO ₂ (ppb)	27.70	23.97	0.67	No
PM _{2.5} (µg/m³)	20.56	21.14	0.80	No
COmean (ppm)	0.40	0.38	0.23	No
COmax (ppm)	5.31	4.40	0.17	No
CO ₂ (ppm)	755.03	845.99	0.42	No
Nicotine	0.57	0.57	0.17	No

TABLE IX: LOG TRANSFORMED CONCENTRATIONS BETWEEN THE TWO COMPLIANCE GROUPS

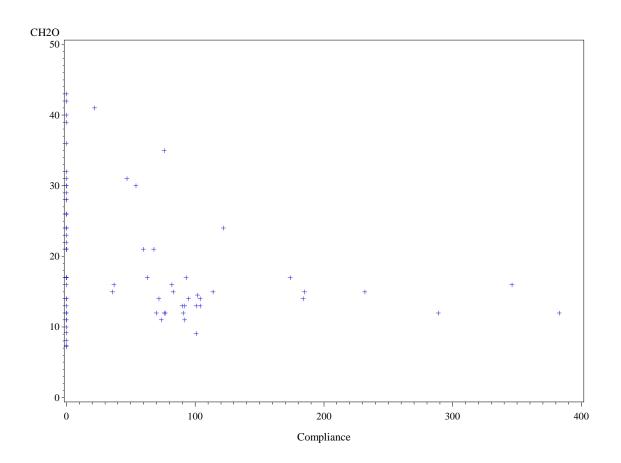
Contaminant	Folded F	Group	Method	p value	Significant
	test	Variances			
Formaldehyde	0.03	Unequal	Satterthwaite	0.02	Yes
NO ₂	0.30	Equal	Pooled	0.45	No
PM _{2.5}	0.70	Equal	Pooled	0.76	No
COmean	0.31	Equal	Pooled	0.25	No
COmax	0.40	Equal	Pooled	0.21	No
CO ₂	3.5E ⁻³	Unequal	Satterthwaite	0.58	No
Nicotine	0.52	Equal	Pooled	0.21	No

The ASHRAE 62.2 standard is intended to establish a minimum ventilation rate in a dwelling unit. However, often time previous studies have relied on design intent to comply with ASHRAE rather than measured results. This study further demonstrates the challenges of relying on intent alone. If design intent alone was used, the sample size would remain 43 compliant units and 42 non-compliant units. Of the 77 units tested for compliance, only 14 were at least 100 percent of the ASHRAE 62.2 requirements (less than 33 percent of units that were intended to comply). Additional analysis was conducted for the definition of compliance at a minimum effective compliance percentage starting with formaldehyde because it was the only contaminant that was statistically significant different between the compliance groups classified on intent alone. The results are shown below in Figure 7. There was a general linear trend that was observed from compliance percent greater than zero to approximately 70 percent compliant. After 70 percent compliant there was a plateauing that occurred. The other indoor air contaminants were also examined to check for consistency of effect. Those plot results are shown in Appendix B.

The operationalized definition of compliance became units that completed ventilation testing and were determined to be at least 70 percent compliant with the ASHRAE 62.2 standard. This was primarily determined from the plot of formaldehyde against concentration and supported by similar trends of the other indoor air contaminants plotted against percent compliance found in Appendix B. Non-compliant units were then defined as units that were tested and determined

to below 70 percent compliance with the ASHRAE 62.2. Table X below shows the new statistics for these two groups below based on this new compliant definition. These values can be compared to Table VII above to explore how the population sizes changed based on this new definition. The 70 percent compliance cut-off definition was determined by plotting percent compliance against formaldehyde, the only significant finding from the air contaminant concentrations above.

Figure 6: Plot of compliance percent with formaldehyde concentration



The same analyses as above were performed again with the new 70 percent cut-point definition. The Wilcoxon Scores Rank Sums results are shown in Table XI for the raw environmental concentration data. Again, formaldehyde was significantly decreased in ASHRAE 62.2 compliant units compared to units that were not compliant with the standard. There was also a statistically significant difference reported in CO2 concentrations between the two compliance groups. A trend continued to emerge showing that as compliance with the ASHRAE 62.2 increased, the concentration of air contaminant decreased. A noted exception to this was with COmax in this dataset; however, there was no statistically significant difference noted. The t-test results for the log-transformed environmental sampling data are shown in Table XII.

Formaldehyde remained the only statistically significant difference between the two compliance groups. Since there were some contaminants that were statistically different among the zip codes and geographic location, it was hypothesized that location bias was skewing the impact ASHRAE compliance had on the indoor air concentrations based on the binary classification analysis.

Therefore, a sub-analysis was performed looking at ASHRAE compliance with the 70 percent compliance cut point in the three zip codes located within the Chicago city limits only. The study size in sub analysis was 64 homes. Of these homes 27 were over 70 percent compliant with the ASHRAE 62.2 standard and 37 were not compliant with the ventilation standard according to the classification definition.

Table XII below shows the average concentration of air contaminants and

between the two groups using the 70 percent definition through Wilcoxon analysis. Table XIV shows the differences between the two compliance groups under the 70 percent definition using log transformed environmental data.

H. Discussion

These results are an important contribution to the scientific literature. There are few published studies that have been able to capture direct environmental and ventilation measurements inside residences with meaningful sample sizes. Published studies measuring inside units mostly have smaller sample sizes due to the difficulty of gaining access to private homes. By collecting samples from inside the homes, this study represents a sampling time frame that is more representative of actual exposure as opposed to previous research that relied on modeling or ambient estimations. Many previous studies have only had sample sizes under 40 homes with a few reporting around hundred or more. Some metanalysis studies have been performed to try and combine these small sampling studies to get more statistical power, but often resulted in a range of sampling contaminants and methodology. On average, the 85 Chicagoland metropolitan homes in this study represents a moderate sampling size compared to other studies that have sampled indoor air contaminants in dwelling units.

TABLE X: UNITS IN COMPLIANCE GROUPS BASED ON 70 PERCENT DEFINITION

I laste	Units	Minimum Compliance	Maximum	Units that were at least
Units	Tested	(%)	Compliance (%)	70%
Total	77	0	383	28
ASHRAE Designed	40	0	383	28
Non-ASHRAE				
Designed	37	0	68	0

TABLE XI: AVERAGE CONTAMINANT CONCENTRATION BY ASHRAE COMPLIANCE WITH 70 PERCENT DEFINITION

Contaminant	Compliant	Non-	P values	Significant
	(n=28)	Compliant		
		(n=49)		
Formaldehyde	14.66	22.41	3.0E ⁻⁴	Yes
(ppb)	14.00	22.41	3.0L	163
NO ₂ (ppb)	25.36	25.91	0.67	No
PM _{2.5} (μg/m³)	16.99	22.08	0.24	No
COmean (ppm)	0.30	0.42	0.98	No
COmax (ppm)	5.10	4.67	0.54	No
CO ₂ (ppm)	700.32	862.49	0.04	Yes
Nicotine	0.52	0.61	0.09	No

TABLE XII: LOG TRANSFORMED CONCENTRATIONS BETWEEN THE TWO COMPLIANCE GROUPS WITH THE 70 PERCENT DEFINITION

Contaminant	Folded F	Group	Method	p value	Significant
	test	Variances			
Formaldehyde	8.0E ⁻⁴	Unequal	Satterthwaite	<1.0E ⁻⁴	Yes
NO ₂	0.73	Equal	Pooled	0.75	No
PM _{2.5}	0.77	Equal	Pooled	0.17	No
COmean	0.49	Equal	Pooled	0.95	No
COmax	1.0	Equal	Pooled	0.58	No
CO ₂	4.7E ⁻³	Unequal	Satterthwaite	0.08	No
Nicotine	0.28	Equal	Pooled	0.13	No

TABLE XIII: AVERAGE CONTAMINANT CONCENTRATION BY ASHRAE COMPLIANCE WITH 70 PERCENT DEFINITION WITHIN CITY ZIP CODES

Contaminant	ASHRAE	ASHRAE	P values	Significant
	Compliant	Non-		
		Compliant		
Formaldehyde	14.76	23.62	<.0001	Yes
(ppb)	14.70	23.02	<.0001	163
NO ₂ (ppb)	25.56	26.23	0.61	No
PM _{2.5} (µg/m³)	17.32	18.12	0.97	No
COmean (ppm)	0.31	0.50	0.74	No
COmax (ppm)	5.12	4.77	0.70	No
CO ₂ (ppm)	717.94	894.5	8.7E ⁻³	Yes
Nicotine	0.52	0.40	0.76	No

TABLE XIV: LOG TRANSFORMED CONCENTRATIONS BY ASHRAE COMPLIANCE WITH 70 PERCENT DEFINITION WITHIN CITY ZIP CODES

Contaminant	Folded F	Group	Method	p value	Significant
	test	Variances			
Formaldehyde	7.3E ⁻³	Unequal	Satterthwaite	<.0001	Yes
NO ₂	0.22	Equal	Pooled	0.56	No
PM _{2.5}	0.54	Equal	Pooled	0.83	No
COmean	0.50	Equal	Pooled	0.83	No
COmax	0.55	Equal	Pooled	0.76	No
CO ₂	<.0001	Unequal	Satterthwaite	0.20	No
Nicotine	1.0	Equal	Pooled	0.81	No

This study measured concentrations inside participants' homes in one location, typically the living room. It is possible that air flow and anthropogenic activities impacted a sampler's ability to accurately measure contaminants. In addition, people are mobile and were likely not in the domicile for the entire sampling period adding complexity to the exposure estimates. However, people spend the majority of their time inside their homes according to the reported activities pattern [55]. Some might believe individual breathing zone samplers to be more accurate to capture personal exposures [64, 65, 66]. However, the methodology used in this study is an economical and practical substitute while still capturing a more accurate representation of home-based exposures than from modeling ambient concentrations. It has also been shown in the literature

that single indoor air monitors are more appropriate surrogates for measuring individual exposures instead of attaching samplers to participants directly to measure their breathing zones [64, 65]. The sampling methods employed by this study would also make it possible to study the exposure for all occupants in the house generating larger participant sample sizes without any additional sampling costs.

The sampling time in this study is also important to recognize as a strength. This study accounted for four continuous sampling days. This was conducted over two weekend days and two weekdays to capture any change in activity patterns during the two categories. For example, more people may have been home during the day during a weekend or vice versa. Perhaps cooking and cleaning patterns were different between the two categories of days. By sampling over both groupings of days, I was able to account for these differences and offer a more robust pattern of exposure. The approximately 96-hour sampling period from each unit is a relatively long sampling time compared to the majority of previous literature that also utilized direct measurements inside homes.

This study also was meticulously designed to try and control for exposure patterns inside the homes. All the homes in the study had an operational gas stove inside the dwelling unit. This allowed for all the units to have the same contaminant generation profile in order to elucidate levels that occupants were exposed to inside of homes. However, it is possible that individual habits also contributed to indoor air contaminants inside these units. For example, the

majority of the units (79) had recirculating hood fans or no exhaust above the stove while others (6) had fans that exhausted to the outside. The units with externally exhausted vent fans potentially had better capture and removal of contaminants generated while using the stove, provided occupants utilized these appliances.

Another strength of this study is that all units were in the same phase of renovation work. Previous studies have found mixed results for how indoor air contaminants change after green weatherization work. Some have suggested that by making units energy efficient, the ventilation in units decreases, which can result in a higher concentration of contaminants especially if there are meaningful generation sources. Published literature has found some decreases in indoor air contaminants immediately following renovation work, but others have found small increases or no reported effect [110, 125, 23]. All of the homes in this study all completed a green renovation project within the previous five years from enrollment.

This study was able to perform a sub analysis on the units based on geographic location. While all the units were in the same metropolitan area, a small subset were located outside of city limits. Previous literature has shown some air contaminants vary based on geographic setting. Such is the case with traffic patterns and exposure to PM_{2.5} [126]. An additional strength of this study was to be able to control for this potential bias by conducting additional analyses based on location within city limits or outside of city limits. It was determined that

PM_{2.5} was statistically higher in units located outside of the city of Chicago. While initial hypotheses were that dense traffic patterns could have lessened PM_{2.5} exposure in units outside of city limits, it is possible that the higher concentrations of nicotine in these units showed higher levels of environmental tobacco smoke. Environmental tobacco smoke is a known contributor of indoor air contaminants including PM_{2.5}. It could be argued that the indoor air contaminants in these units would have had better indoor air quality metrics and skewed that data regardless of ASHRAE 62.2 compliance. However, the differences in air contaminant concentrations between ASHRAE 62.2 compliant units were explored just within city of Chicago limits as well controlling for this potential bias. The indoor air contaminant differences did not significantly change when removing the units that were outside the city of Chicago limits. There was one additional statistically significant finding for CO₂ when the raw data were explored through Wilcoxon Scores Test. These results suggest that there are potentially more impacts in indoor air quality from ASHRAE 62.2 compliance than were captured in this study. Additional research is needed to explore how ASHRAE 62.2 compliance impacts indoor air quality in residential units.

When contaminant concentrations were examined between the two compliance groups, few differences were noted. Air contaminant concentrations were generally lower in the ASHRAE compliant units compared to the non-compliant units for formaldehyde, PM_{2.5}, and CO₂. However, only a statistically significant finding was found for formaldehyde. Concentrations for NO₂, COmean,

and COmax were marginally higher in ASHRAE compliant units compared to non-compliant units when using a 0 percent compliance cut point, but none of the differences were statistically significant. When a 70 percent cut-point was used, all of the concentrations of air contaminants were lower in ASHRAE 62.2 compliant units compared to those that were not compliant except for CO.

These have some other possible explanations. It was hypothesized that air contaminants would have been lower in homes that were compliant with the ventilation standard. However, ambient air in this geographic area may have been more contaminated than primary sources from the indoor environment. These units were enrolled in this study because they contained similar indoor air contaminant sources such as a gas stove inside the units. Therefore, it could also be argued that the generation patterns are similar inside the units so there were no statistical differences observed even with the ventilation components. Another facet of these data is the percent compliance with the ASHRAE 62.2 standard.

The range of compliance within the ASHRAE 62.2 group was quite large for a modest sample size. The lowest value in the compliant group was 22 percent compliant with the standard and the highest reported value was 386 percent compliant with the standard. With an average compliance percent of almost 114 for the 36 units. A unit that reported only a 22 percent compliance with the standard may in fact be much different than the unit that reported over 300 percent compliance with the standard. Improved maintenance to repair bathroom exhaust fans is a clear need. A strength of this study was that it attempted to

explore different compliance cut-points to explore how ASHRAE 62.2 classification impacted indoor air contaminants in units based on data-driven decisions. This study could be used in the future to help additional research determine a proper operational definition of an ASHRAE 62.2 compliant home.

The simple dichotomous classification of either compliant with the standard or not presented limitations to further explore how the contaminant levels varied within these units for this study. The cohort size for compliant units was drastically decreased by increasing the compliance cut point to 70 percent. The study size of compliant units varied greatly based on the definition. There were 43 units that were intended to comply with the ventilation standard, 38 that completed blower-door testing, 36 that achieved any positive percent compliance, 14 that achieved a minimum compliance of 100 percent, and 28 that achieved a minimum compliance of 70 percent. Additional research and analysis would be able to further investigate these potential associations, ideally with larger sample sizes. By design this study expected to enroll approximately half of the participants from units that had zero percent ASHRAE 62.2 compliance as they were not intended to comply based on renovation designs.

Homes were enrolled into this study based on the recent green renovation work that was completed within the last five years prior to enrollment. It was determined that roughly half of the homes (43) were designed to comply with the ASHRAE ventilation standard, while half (42) of the homes were not designed to comply with the standard. However, quantitative results from testing 77 of the

bathroom exhaust ventilation that was designed to operate in the units was not functioning while in another scenario it was found that a unit had mechanical ventilation even though the design was not intended to comply. The blower door results were a preferred method of classification of the units based on compliance because it provided actual results rather than just design intent.

The only statistically significant air contaminant difference was found for formaldehyde. This suggests that ASHRAE compliance does have some impact on resident exposure. This current study was also restricted to Phase 1 visits, and it is possible that inclusion of visits at four and eight months after enrollment could yield different results. Even though natural gas appliances have some release of formaldehyde, most exposure in homes is not from anthropogenic or activity related point-source emissions [106]. Formaldehyde exposure in homes can be primarily attributed to off gassing [106]. It may be unrealistic to expect bathroom exhaust fans to ventilate an entire home. Bathroom exhaust may also be effective for broad ventilation of air contaminants but not for air contaminants in the home that are largely related to point sources. If bathroom doors were not kept open for most of the time, the effect of the exhaust would be diminished. Better ventilation is likely to reduce indoor air contaminants in homes.

This study also had limitations. A key component was that this study did not account for seasonal variations. All these baseline data were collected between November 2018 and August 2019. The Chicago metropolitan area is

known for wide seasonal temperature and weather patterns. Some of the units in this study were sampled during winter months when the furnace was more likely to be in use. In addition, some units also had gas furnaces located inside the dwelling units, usually in a utility closet. Conversely, units sampled during warmer months may have been more likely to have windows open, thus increasing ventilation and air changes inside the units. As part of the sampling protocol, participants were asked to keep windows shut to ensure true indoor air contaminants present inside the housing units were captured. However, some units did not have central air and windows had to be opened in order to maintain occupant comfort.

There was variation within the composition of the equipment of the units that could also be further investigated to explore any meaningful changes in indoor air contaminant levels. As previously mentioned, some units in this study had additional gas appliances inside the homes themselves while others had these in equipment rooms in the basement. The units also varied in the number of levels, windows, shared envelopes with other units, and number of occupants. All of these additional variables could also impact the air contaminant levels inside dwellings but were outside the scope of this research. However, these results presented are important as they contribute to the scientific literature for indoor air exposure profiles inside dwellings.

VI. INDOOR AIR QUALITY AND HEALTH-RELATED QUALITY OF LIFE

A. <u>Hypothesis</u>

Health-related quality of life is associated with air quality metrics inside residential dwellings. There are negative associations with SF-36 domains and increased concentrations of PM_{2.5}, NO₂, CH₂O, CO₂, and CO. There is a positive association with SF-36 domains and increased percent compliance with the ASHRAE 62.2 standard.

B. Introduction

This research was able to investigate how indoor air quality impacts resident health related quality of life. Prior to this study, there was minimal scientific literature regarding direct measures of indoor air quality and occupant health indices. It has generally been hypothesized that general comfort improves with proper ventilation, but there is a lack of actual investigation into the occupants' health especially in residential settings. While there have been some studies that explored exposure to indoor air contaminants in homes and health, there are even fewer that have investigated outcomes pertaining to health-related quality of life. The objective of this aim was to explore how health-related quality of life is related to ASHRAE compliance as well as other air contaminants in residential settings. The working hypothesis was that health-related quality of life was positively impacted when green renovation projects met the ASHRAE 62

standard, and that health-related quality of life was negatively impacted in the presence of indoor air contaminants. The <u>approach</u> included interviewing residents in both ASHRAE compliant and noncompliant dwellings using the reliable and validated SF-36 and conducting environmental sampling within the units.

C. <u>Justification and Feasibility</u>

The quality-of-life scale, SF-36, has proved to be a useful tool and has been shown to be psychometrically sound in research applications. This scale has been employed in several different research areas including how indoor air quality impacts health related quality of life [87]. The SF-36 is divided into the following eight domain scales: physical functioning, role physical, bodily pain, general health, vitality, social function, role emotional, and mental health [87]. These contribute to two separate summary scores known as the Physical Component Summary (PCS) and the Mental Component Summary (MCS) [97]. These two separate but related domains will help to evaluate construct validity during data analysis as well.

Air quality impacts health-related quality of life. One study in Japan estimated environmental exposures for nitrogen oxides and particulate matter and compared levels to the eight domains of the SF-36 with over 3,000 participants [127]. They reported a significant association between nitrogen oxide levels and the vitality domain from the scale [127]. Another study found that

asthmatics who were exposed to higher levels of PM_{2.5} had decreased quality of life when assessed with the Asthma Quality of Life Questionnaire [128]. Another study looked at occupant health pre and post renovation work in 37 homes. These researchers reported that occupants had statistically significant improvements in five of the eight SF-36 domains after improvements were made to the home [129].

Nitrogen dioxide is a known respiratory irritant. Pulmonary edema may occur at high levels of NO₂, but continuous low-level exposure may result in bronchitis [130]. Such lower levels in some studies have demonstrated that there can be increased bronchial reactivity, increased rate of infections, and decreased lung function [130]. Most study populations demonstrate no immediate health effects for acute exposures less than 1,000 ppb. However, sensitized populations like those with lung diseases may notice pulmonary function changes at lower levels around 300 ppb [79]. There have also been health effects on several other biologic endpoints from chronic exposure to NO₂ that may be reversible or irreversible [79].

Formaldehyde has a range of documented physiological health effects. The EPA designated formaldehyde under Group B1 (probable human carcinogen) in 1989 based on sufficient animal modeling evidence, but the agency noted a lack of human epidemiologic studies [72, 130]. Some sensitized populations report an odor threshold as low as 24 ppb [79]. Between ten and 20 percent of the total US population may be more vulnerable to health effects of formaldehyde due to differences in sensitivity [130]. Increasing concentrations can result in irritation of

eyes, nose, and throat [130, 27]. At a range of just under 49,000 ppb to almost 102,000 ppb death becomes possible from a short single exposure period [79]. There is also more recent evidence supporting the carcinogenic action of formaldehyde exposure and the International Association for Research on Cancer (IARC) designated formaldehyde and PM_{2.5} as carcinogenic to humans (Group 1) [131, 56, 79].

Other scientific literature has demonstrated an association between healthrelated quality of life and exposure to air contaminants. Studies have shown that decreases in PM_{2.5} are associated with an increase in average life expectancy and that exposure to PM_{2.5} has an increased relative risk for total mortality [132, 79]. Particle size and aerodynamic diameter greatly influence the potential health effects as these characteristics generally determine deposition locations in the lung. Particles that are 10 micrometers (µm) or greater tend to settle out of the air relatively quickly and are classified as "coarse" [133]. Smaller particles of 2.5 µm or less are considered "fine" and can travel deeper into the respiratory tract causing health effects [133]. There are large population-based estimates for the impacts of PM on health. The WHO has estimated that there are 800,000 deaths and almost a million disability adjusted life years (DALYs) annually from PM exposure worldwide [134]. Outside of carcinogenic and mortality statistics, PM_{2.5} exposure has also been associated with bronchitis, decreased lung function, and cardiovascular deficiencies [79].

The physiological impacts of PM_{2.5} have been well documented in the scientific literature, but there are less published studies also exploring the health-related quality of life. One study in Mongolia looked at PM measurements, fuel types, and season to explore both qualitative and quantitative health measurements. Researchers reported that participants with a FEV1/FVC ratio less than 0.7 of the predicted value and who had increased PM exposure from indoor fuel sources had significantly lower health related quality of life compared to those who were above this predictive benchmark [39].

There have also been investigations as to how PM_{2.5} exposure could also impact mental health and quality of life factors outside of physical health endpoints. A study of over 4,000 elderly people showed an association between increased PM_{2.5} and increased depressive outcomes and some anxiety symptoms [135]. This association was strongest in participants with lower socioeconomic status and with comorbidities, but the exposure to PM_{2.5} was assessed through modeling [135]. These outcomes are supported by previous research that has demonstrated the biologic plausibility of this relationship through an association of neurological effects and PM_{2.5} exposure [135].

Health impacts of CO have been well documented. Carbon monoxide binds to hemoglobin to form carboxyhemoglobin [79]. Even at low levels it has been documented that CO binds more readily with hemoglobin in the body and impacts the oxygen transport pathway [79]. Although acute exposures may be transient and reversible, even low levels of CO have resulted in decreased weight at birth,

birth defects, increased mortality, cardiovascular problems, cerebrovascular interruption, diseases of the lung, and other negative health outcomes [23, 79].

Most NO₂ studies have focused on physical health, but one study explored health-related quality of life. Researchers reported that NO₂ did not impact quality of life through two different scales [40]. However, they were focused on NO₂ concentrations from traffic pollution and used modeling techniques for exposure estimates [40]. This current study instead measured exposures inside the actual homes from both primary and secondary sources. The study by Colton et al. (2014) reported that homes rehabbed using a green methodology reported lower levels of NO₂ than homes that had a conventional rehab. They also reported that residents in green homes reported less symptoms associated with sick building syndrome [116]. Their study did not specifically look at how NO₂ impacted health-related quality of life.

Carbon dioxide health effects traditionally were explored at levels much higher than ambient occurrences. Recent studies have focused on potential impacts at levels most experienced by humans. A study by Satish et al. (2012) found there were statistically significant decreases in decision making abilities for those exposed to 1,000 ppm CO₂ compared to exposure at 600 ppm [20]. These differences were larger at exposure to 2,500 ppm of CO₂. Research in the occupational setting has used CO₂ concentrations as a surrogate measure to explore symptoms of sick building syndrome and inadequate fresh air supply [45]. Other smaller studies have reported declines in participants' abilities to perform

proofreading capabilities with increases in CO₂ exposure [20]. One study also found that there was an association between CO₂ levels and depressive symptoms [136]. A review study examined the impacts of ventilation on CO₂ in office buildings. These findings showed increases in ventilation were associated with decreases in CO₂ levels, prevalence of SBS, and respiratory illness [137].

Environmental tobacco smoke (ETS) has also been classified as a known human carcinogen (Group A) by the EPA [130]. Some estimates have concluded that an estimated 3,000 deaths in the US from lung cancer occur even among individuals who do not smoke [130, 78]. There have also been correlations between PM generated from ETS and labored breathing, respiratory disease, cardiovascular disease, and immune system impairment [138, 130]. There is also research to show that ETS can directly impact indoor air quality metrics [41, 79]. These results from previous research support the potential of ETS to have a direct negative impact on health-related quality of life and indoor air quality metrics.

Exposure to formaldehyde has also been found to have negative health outcomes. Indoor exposures have been found to present an increased risk of asthma diagnosis in children at levels equal to or greater than 49 ppb [139]. Some research has been able to find an association between indoor air measures like formaldehyde and general weariness and malaise [111]. A study among home based daycare workers reported increased symptoms with higher exposures to formaldehyde concentrations [140].

Ventilation standards were historically designed to control for odors; however, it is now known that there are numerous common indoor air contaminants that can impact human health well below the odor threshold [27]. It has been documented in the scientific literature that the quest to decrease residential energy demand often resulted in worse indoor air quality due to decreases in ventilation. There has been a dispute among the ASHRAE community as to whether standards should incorporate health outcomes and wellbeing as guidance for indoor air quality [141]. Studies have generally supported the benefits of increased ventilation in indoor environments. It is widely accepted that decreased ventilation in homes leads to an increase concentration of indoor air contaminants [24]. The extent to which ventilation truly impacts these specific concentrations has been varied in the literature. One particular study did not find that differences in ventilation systems influenced formaldehyde concentrations in homes [142]. However, the study did not collect actual ventilation measurements and instead relied on design information for classification. Another study reported that increasing ventilation did reduce formaldehyde concentrations in homes, but at less efficiency than predicted under a constant emission rate theory [143]. A study of 77 homes in Boston found that ventilation rates had statistically significant associations with NO2 concentrations [144].

Few studies have explored the relationship between ventilation and health outcomes in homes. A randomized controlled study of asthmatic children in

Canada explored how increasing the ventilation rates in homes impacted asthma symptoms in children. Researchers reported no significant decrease in the number of days with asthma symptoms with increased ventilation, but they did note there was a difference in wheezing and decrease in formaldehyde with increased ventilation [145]. Another study in the United Kingdom found some improvement in parent reported quality of life for asthmatic children with increased ventilation [146]. In Sweden, a research study concluded low ventilation rates were a risk factor for asthma and allergy symptoms among children [147]. A review of different HVAC systems found inconclusive results in regard to reports of Sick Building Syndrome (SBS) [148]. While a report measuring ventilation rates in Chinese homes, researchers found decreased ventilation rates associated with an increase in SBS symptoms [149]. Compliance with the ASHRAE 62.2-2010 standard has been associated with decreases in physical symptoms among children and improved psychological outcomes in adults [150].

This current study was unique in that it investigated how the ASHRAE ventilation standard in the US can impact health-related quality of life in adults. Homes were similar in socio-economic status as well as having undergone a "green" renovation. The primary point of investigation was how compliance with ASHRAE 62.2 impacted health-related quality of life. This study was important because to-date there has been limited investigation into how the ASHRAE 62.2 standard impacts occupant health-related quality of life outside of controlling for odors.

D. Research Design

A cross-sectional study was conducted in which both indoor air samples and health data were collected at the same time. A group of 85 primary participants living in low-income multifamily Chicago metropolitan area homes were interviewed utilizing the SF-36. In each home a sampling apparatus was deployed to collect PM_{2.5}, CO₂, CO, NO₂, formaldehyde, and nicotine samples. Active PM_{2.5} sampling collected particulate matter with a Personal Modular Impactor. Passive sampling was conducted for formaldehyde with a UMEx 100 sampler, NO₂ with a UMEx 200 sampler, nicotine with treated filter prepared by a lab at Johns Hopkins, CO with a Lascar EL-USB-CO data logger, and CO₂ with a Telaire attached to a HOBO datalogger. These methods are described in more detail above.

E. Methods

Study participants were enrolled after an informed consent process that discussed risks and benefits, duties associated with participation and other matters. All participants had all their questions answered. They were also informed of their right to withdraw at any time during the study. Trained field staff administered the reliable and valid SF-36 survey on paper and recorded the responses into Research Electronic Data Capture (REDCap) software from Vanderbilt University. This application was chosen because it was HIPPA compliant, accessed easily during home visits through internet connection, and

required minimal staff training or knowledge of coding operations. Interviewers were trained to administer the questionnaire in a consistent manner that was respectful of participants.

Residents were asked to refrain from opening windows to prevent oversampling of ambient air and to optimize performance of the building ventilation systems. They were also asked not to smoke inside to prevent skewing sampling data. Bathroom doors were left open when not in use to ensure ventilation measures were operating as intended within the units. Ventilation measures in the homes were measured by field expert contractors to determine the percent compliance with the ASHRAE standard 62.2.

F. Data Analysis

Data were coded for analyses utilizing the preceding methods. Sampling data for formaldehyde, NO₂, PM_{2.5}, COmax, COmean, and CO₂ were left as their numeric values. Nicotine was coded as a dichotomous variable to either be above the limit of detection (1) or below the limit of detection (0). ASHRAE 62.2 compliance was examined through both the numeric percent compliant result and a dichotomous variable. The dichotomous ASHRAE compliance variable was established from the method above in Aim 1. Units that showed a minimum of 70 percent compliance with the standard were designated as ASHRAE 62.2 compliant units while those that were under 70 percent compliance were designated as non-compliant units. The following other categorical variables were also coded

utilizing a dichotomous method to help control for confounding: sex (0=male; 1=female), asthma diagnosis (0=no; 1=yes), education (0=high school or less; 1= greater than high school), income (0=<10,000; 1=>10,000), Hispanic (0= no;1=yes), race (0= other, 1=Black). Age was left as its numeric value for analyses.

Correlation tests were run on all the potential dependent and independent variables to explore relations. Spearman correlations were run using raw environmental data to investigate associations between the above variables given non-parametric assumptions. Pearson correlations were run using log transformed environmental data and binary coding where appropriate. These associations provided exploratory results for the relationship between health-related quality of life for residents and indoor air quality metrics as well as guidance for additional analyses.

Both Wilcoxon Rank Sums Scores and t-test values were run to explore any differences in SF-36 scores between the two ASHRAE 62.2 compliance groups given the minimum 70 percent compliance definition. The Wilcoxon test used raw environmental concentration data, while the t-test used log transformed data.

Statistical analysis also included logistic regression to explore the potential association between indoor air quality and health related quality of life. Pearson coefficients were calculated for continuous variables and chi square tests were performed for the dichotomous variables for PCS and MCS. These were able show potential relationships and some directionality of the relationships between indoor air quality and health related quality of life. The indoor air measurements

were the independent predictor variables and the results from the two scores of the SF-36 were the dependent environmental outcome variables (Equation 3). To control for other potential biases, the variables of sex, age, asthma, education, income, ethnicity, and race were also included in this equation as well.

Equation 3

$$Y_{SF36} = \beta_o + \beta_1 X_{PM2.5} + \beta_2 X_{CO2} + \beta_3 X_{NO2} + \beta_4 X_{CH2O} + \beta_5 X_{ETS} + \beta_6 X_{CO} + \beta_7 X_{ASHRAE} + \epsilon$$

Model selection was utilized to further investigate how these variables impacted resident health-related quality of life. To help eliminate issues of multicollinearity, only one variable was selected from environmental data that were strongly correlated. For this research, the final independent variables were log of formaldehyde, log of NO₂, log of PM_{2.5}, log of COmean, log of CO₂, nicotine (yes/no), compliance percent, sex (male/female), age, asthma (yes/no), education (high school or less/more than high school), income (under \$10,000/greater than \$10,000), Hispanic (yes/no), race (black/other), and city (yes/no). The outcome variables modeled were the PCS and MCS domains from the SF-36 results.

An automatic stepwise model selection was conducted to determine which variables were statistically significant for predicting the PCS and MCS results from the SF-36. A significance level for entry (SLE) of 0.10 and a value of stay significance level (SLS) of 0.15 were used for both models predicting the PCS and MCS from the SF-36. For MCS the city variable was kept in the model because of

the hypothesis that nicotine was operating as a collider in the relationship pathway with MCS. Both forward and backward elimination were also analyzed to test the validity of the stepwise model under the same inclusion and exclusion criteria. Additionally, the variables were added individually into a model based on significance to control for loss of observations and as an additional validation step.

It is widely known that air contaminants do not exist in isolation. It may be plausible that ASHRAE 62.2 compliance is better at controlling for some forms of air pollution than others or that larger concentrations of multiple contaminants had greater impacts on health-related quality of life. As such this study also investigated total air contaminant load. To perform this analysis the raw contaminant concentrations were ranked from smallest to largest across all the units. These rank results were then summed for each of the units individually to create a sum air contaminant. Each home had one total air contaminant sum score. These total load scores were then divided into tertiles for general low total contaminant exposures, medium total contaminant exposure, and high total contaminant exposure. The air contamination tertiles were then compared against the numeric MCS, PCS, and ASHRAE 62.2 compliance scores from the respective homes to look for differences. This methodology has been utilized in previous literature [151].

G. Results

In total, 85 primary participants were interviewed representing 85 unique independent housing units in the Chicago metropolitan area. These homes were spread across four zip codes. The demographics represented from the participant sampling pool are presented below in Table XV. The average age of the participants enrolled in this study was 48 years old and ranged from 18 to 81 years old. The majority of the population interviewed identified as non-Hispanic Black women. There was a relatively equal spread between education levels among participants and most study participants reported a household income below 10,000 dollars per year. This income finding was expected since all the homes were multifamily low-income housing units. Asthma was of particular interest in this study as air contaminants are known to exacerbate symptoms as well as the potential to impact responses for participant health-related quality of life. These statistics provide important information for the potential to generalize findings from this study to larger ecological settings or similar populations.

The two SF-36 scores used in these analyses for the objectives of this study were relatively normally distributed as shown below. Normality was not improved when transforming the data so raw values were used for the remaining analyses. The mean, median, minimum, and maximum values for the PCS and MCS scores are displayed below in Table XVI. Higher scores indicated a more positive health related quality of life. There were no perfect scores which meant every participant had some impairment in their health-related quality of life.

To explore how ASHRAE 62.2 compliance impacted health related quality of life differences in scores between the two groups were examined. Based on previous research reported in Aim 1 above, a minimum cut-off value of 70 percent compliant was used. Table XVII below shows the proc t-test results while the Table XVIII shows the results from the Wilcoxon. Two tailed test assumptions were used because it is not known if ASHRAE 62.2 compliance increased concentration of air containments or contributed to a decrease in concentration through exhaust from the unit. There were no statistically significant differences noted. However, the PCS was slightly lower, but the MCS was slightly higher in the ASHRAE 62.2 compliant units.

When examining the differences in the SF-36 component scores based on ASHRAE 62.2 compliance, there were no statistically significant differences found from either the Wilcoxon scores test with raw environmental data or the t-test operation with log transformed data. The mean scores for both composite constructs were comparable for both the compliant units and the non-compliant units. However, additional analyses were conducted to further investigate how ASHRAE 62.2 compliance and indoor air contaminants impacted health-related quality of life among participants. Correlation tables were created to explore any relationships with the PCS and the MCS results from the SF-36. Spearman correlation coefficients were calculated for the raw environmental data and Pearson correlation coefficients were calculated for the log transformed data.

TABLE XV: DEMOGRAPHICS OF STUDY POPULATION

		% (n)
Sex		
	Female	87.1% (74)
	Male	12.9% (11)
	Refused	0.0% (0)
Race		,
	Black	68.2% (58)
	White	16.5% (14
	Other	11.8% (10)
	Refused	3.5% (3)
Hispanic	1101000	0.070 (0)
тпаратис	No	67.1% (57)
	Yes	32.9% (28)
	Refused	0.0% (0)
Education		0.0 /8 (0)
Euucatioi		
	Never completed high school	20.00/ /17\
		20.0% (17)
	High school or GED	29.4% (25)
	Some college	38.8% (33)
	College degree	10.6% (9)
	Refused	1.2% (1)
Income		()
	Less than \$10,000	62.4% (53)
	\$10,000 to \$30,000	27.1% (23)
	\$30,000 to \$50,000	7.1% (6)
	Greater than \$50,000	2.4% (2)
	Refused	1.2% (1)
Asthma		
	Yes	27.1% (23)
	No	72.9% (62)

TABLE XVI: SCORE SUMMARIES FOR THE PCS AND MCS OF THE SF-36

SF-36	Mean Score	Median Score	Minimum	Maximum
Component			Score	Score
(n=85)				
PCS	46.9	49.1	17.7	62.7
MCS	51.5	54.0	24.4	71.6

TABLE XVII: AVERAGE CONTAMINANT CONCENTRATION BY ASHRAE COMPLIANCE FROM WILCOXON SCORES TEST USING THE 70 PERCENT DEFINITION

SF-36	ASHRAE	Non-	P values	Significant
Component	Compliant	Compliant		
	n=28	N=49		
PCS	45.03	46.52	0.67	No
MCS	52.02	51.16	0.92	No

TABLE XVIII: RESULTS FROM THE T-TEST BETWEEN GROUPS BASED ON ASHRAE COMPLIANCE WITH THE 70 PERCENT DEFINITION

SF-36	Folded F test	Group	Method	p value	Significant
Component		Variances			
PCS	0.84	Equal	Pooled	0.60	No
MCS	0.19	Equal	Pooled	0.71	No

Pearson correlation coefficients were used to explore bivariate associations with the variables and log-normal environmental data as shown in Table XIX.

There was a significant weak negative association between PCS and CO₂ and a significant moderate negative association between PCS and age. There was a statistically significant weak negative association between MCS and CO₂.

Compliance had a significant weak negative association with formaldehyde and CO₂. Other statistically significant weak and moderate positive associations were seen between formaldehyde/NO₂, formaldehyde/CO mean, formaldehyde/CO₂, NO₂/CO mean, NO₂/CO max, PM_{2.5}/CO mean, and PM_{2.5}/CO₂.

To explore the dichotomous variables with the outcome variable of interest from the SF-36, ttests were used. Table XX shows how PCS results varied for the dichotomous variables while Table XXI shows the differences of the dichotomous variables for MCS results. The boxplot figures for the statistically significant findings are displayed in Appendix C. The boxplot figures show the directionality of the significant relationships. Asthma was negatively associated with PCS. Education was positively associated with PCS. The presence of nicotine was positively associated with MCS.

This study does not conclude that the presence of nicotine in homes has a positive association with mental health-related quality of life. Units outside of the city were more statistically likely to also have nicotine present. It was hypothesized that the presence of nicotine was functioning as a collider between MCS and features of neighborhood partly captured by being located in city limits.

TABLE XIX: PEARSON CORRELATION COEFFICIENTS OF CONTINUOUS VARIABLES

	Pearson Correlation Coefficients Prob > r under H0: Rho=0 Number of Observations										
	MCS	PCS	logCH2O	logNO2	logPM	logCOmean	logCOmax	logCO2	Compliance	Age	
MCS	1.00000	0.07332	-0.14156	-	0.00539	0.12397	0.10683	-	0.03598	0.04689	
MCS		0.5049	0.1962	0.14337	0.9612	0.2642	0.3364	0.21316	0.7561	0.6700	
	85	85	85	0.1905	84	83	83	0.0501	77	85	
				85				85			
PCS	0.07332	1.00000	-0.14822	0.02198	0.03915	-0.17472	-0.14373	ı	-0.02776	-	
PCS	0.5049		0.1758	0.8418	0.7236	0.1141	0.1949	0.21840	0.8106	0.42335	
	85	85	85	85	84	83	83	0.0446	77	<.0001	
								85		85	
logCH2O	-	-	1.00000	0.26223	0.13855	0.33398	0.19079	0.54258	-0.28959	0.10622	
logCH2O	0.14156	0.14822		0.0153	0.2088	0.0020	0.0840	<.0001	0.0106	0.3333	
	0.1962	0.1758	85	85	84	83	83	85	77	85	
	85	85									
logNO2	-	0.02198	0.26223	1.00000	0.09074	0.40293	0.42937	0.16743	0.06450	-	
logNO2	0.14337	0.8418	0.0153		0.4117	0.0002	<.0001	0.1256	0.5773	0.11590	
	0.1905	85	85	85	84	83	83	85	77	0.2909	
	85									85	
logPM	0.00539	0.03915	0.13855	0.09074	1.00000	0.24849	0.13126	0.21867	-0.08819	-	
logPM	0.9612	0.7236	0.2088	0.4117		0.0244	0.2398	0.0457	0.4456	0.12394	
	84	84	84	84	84	82	82	84	77	0.2613	
										84	

TABLE XIX: PEARSON CORRELATION COEFFICIENTS OF CONTINUOUS VARIABLES (continued)

logCOmean	0.12397	_	0.33398	0.40293	0.24849	1.00000	0.84708	0.42525	0.12982	0.13115
logCOmean	0.2642	0.17472	0.0020	0.0002	0.0244		<.0001	<.0001	0.2670	0.2373
	83	0.1141	83	83	82	83	83	83	75	83
		83								
logCOmax	0.10683	-	0.19079	0.42937	0.13126	0.84708	1.00000	0.25616	0.13532	0.05480
logCOmax	0.3364	0.14373	0.0840	<.0001	0.2398	<.0001		0.0194	0.2471	0.6227
	83	0.1949	83	83	82	83	83	83	75	83
		83								
logCO2	-	-	0.54258	0.16743	0.21867	0.42525	0.25616	1.00000	-0.04003	0.06407
logCO2	0.21316	0.21840	<.0001	0.1256	0.0457	<.0001	0.0194		0.7296	0.5602
	0.0501	0.0446	85	85	84	83	83	85	77	85
	85	85								
Compliance	0.03598	-	-0.28959	0.06450	-	0.12982	0.13532	-	1.00000	0.12914
Compliance	0.7561	0.02776	0.0106	0.5773	0.08819	0.2670	0.2471	0.04003		0.2630
	77	0.8106	77	77	0.4456	75	75	0.7296	77	77
		77			77			77		
Age	0.04689	-	0.10622	-	-	0.13115	0.05480	0.06407	0.12914	1.00000
Age	0.6700	0.42335	0.3333	0.11590	0.12394	0.2373	0.6227	0.5602	0.2630	
	85	<.0001	85	0.2909	0.2613	83	83	85	77	85
		85		85	84					

Therefore, for additional analysis of MCS, nicotine was not included. Instead, the city variable was implemented for additional investigation.

The results from the PCS stepwise model in SAS are show in Table XXII below. The results for the MCS stepwise model are shown below in Table XXIII. The models included 75 observations with no missing data. In the shown results below, having asthma, participant age, and logCO₂ levels had statistically significant associations with PCS as a response variable with 75 observations used. The model was also run with just forward selection and just backward selection. Both additional analyses resulted in the same model

In the results from Table XXIII logCO₂, logCOmean, and logNO₂ had statistically significant associations with MCS as a response variable with 75 used. The same variables were statistically significant utilizing forward modeling selection. When backward elimination was used the following variables were significant: logNO₂, logCOmean, logCO₂, education, Hispanic, and race. Both variables for race and ethnicity had variance inflation factors close to three while the other significant variables had lower variance inflation factors..

In order to provide additional analysis for model selection. Individual variables were added based on significance. There were four variables that had individual significant associations with PCS in the above analysis. These variables were age, asthma, education, and logCO₂ in descending significance. When variables were added one at a time starting with age in a stepwise methodology

only age and asthma remained in the model with PCS with all 85 observations used even after adding in non-significant variables as well

Similarly, MCS was examined through model selection adding variables individually based on significance. The city variable was kept in the model again. When the variables were put through model selection, logCO2 became a significant regressor with all 85 observations used. No other variables were significant on an individual basis in the model with MCS and city. When added to the model with city and logCO2, logCOmean became significant as well with 83 observations used. Both NO2 and Hispanic variables became positive when added to the model next still with 83 observations used. No additional significant variables were found after this step. The final model included city, logCO2, logCOmean, logNO2, and Hispanic.

The contaminants that were combined for total load scores inside units were concentrations of formaldehyde, NO₂, COmean, CO₂, and nicotine. The individual air concentrations were ranked from low to high for each unit sampled. The individual rank scores of contaminants were then summed for each unit. These summed rank load scores were divided into tertiles for the following analysis where 0 was the lowest rank tertile and 2 was the highest rank tertile. This meant that a unit within the higher exposure tertile generally had higher total air contaminant exposure compared to a unit that was in the lowest rank tertile.

TABLE XX: TTEST RESULTS BETWEEN DICHOTOMOUS VARIABLES AND PCS

Contaminant	Folded F	Group	Method	p value	Significant
	test	Variances			
NicotineYN	0.78	Equal	Pooled	0.83	No
Sex	0.54	Equal	Pooled	0.13	No
Asthma	0.91	Equal	Pooled	1.9E ⁻³	Yes
Education	0.86	Equal	Pooled	0.02	Yes
Income	0.85	Equal	Pooled	0.25	No
Hispanic	0.84	Equal	Pooled	0.08	No
Race	0.60	Equal	Pooled	0.19	No
City	0.72	Equal	Pooled	0.08	No

TABLE XXI: TTEST RESULTS BETWEEN DICHOTOMOUS VARIABLES AND MCS

Contaminant	Folded F	Group	Method	p value	Significant
	test	Variances			
NicotineYN	0.05	Equal	Pooled	0.03	Yes
Sex	0.75	Equal	Pooled	0.43	No
Asthma	0.87	Equal	Pooled	0.70	No
Education	0.09	Unequal	Satterthwaite	0.70	No
Income	0.72	Equal	Pooled	0.84	No
Hispanic	0.55	Equal	Pooled	0.07	No
Race	0.92	Equal	Pooled	0.18	No
City	0.30	Equal	Pooled	0.16	No

TABLE XXII: PCS STEPWISE MODEL SELECTION RESULTS

Variable	Parameter Estimate	Standard Error	Type II SS	F Value	Pr > F
Intercept	93.41200	18.67061	2423.14626	25.03	<.0001
logCO2	-10.98190	6.49068	277.11878	2.86	0.0950
Age	-0.27527	0.07370	1350.56313	13.95	0.0004
Asthma	-9.17850	2.53417	1269.88528	13.12	0.0005

TABLE XXIII: MCS STEPWISE MODEL SELECTION RESULTS

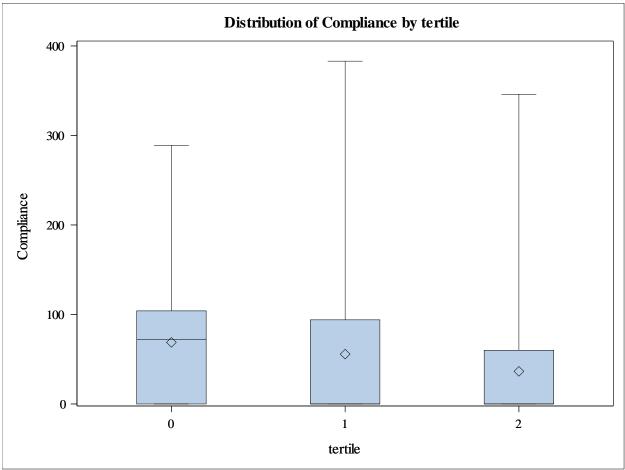
	Variable	Parameter Estimate	Standard Error	Type II SS	F Value	Pr > F			
	Intercept	129.31774	21.99866	3044.72062	34.56	<.0001			
*	City	4.89606	2.98412	237.18341	2.69	0.1053			
	logNO2	-12.45224	5.62493	431.80145	4.90	0.0301			
	logCOmean	5.08742	1.55918	938.05053	10.65	0.0017			
	logCO2	-21.70281	6.66920	933.05649	10.59	0.0018			
	* Forced into the model by the INCLUDE= option								

Figure 8 shows the difference in ASHRAE 62.2 compliance between these air contamination load tertiles. There was a general trend that ASHRAE 62.2 compliance was greater among the tertile with the lowest reported air contaminant loads. However, none of the differences between the tertile groups were statistically significant as determined through analysis of variance.

Similarly, the health-related quality of life composite scores from the SF-36 were also examined. Figure 8 below shows the differences in PCS while Figure 9 shows the differences in MCS outcomes based on total air contaminant tertiles. There were no statistically significant differences between any of the tertiles. However, the same general trend remained. Higher average scores for both PCS and MCS were observed in the tertiles with the lowest air contaminant loads. As total air contaminant load increased there were decreases in average PCS and MCS results for the participants.

Additionally, the health-related quality of life composite scores from the SF-36 were also examined. Figure 8 below shows the differences in PCS while Figure 9 shows the differences in MCS outcomes based on total air contaminant tertiles. There were no statistically significant differences between any of the tertiles. However, the same general trend remained. Higher average scores for both PCS and MCS were observed in the tertiles with the lowest air contaminant loads. As total air contaminant load increased there were decreases in average PCS and MCS results for the participants.





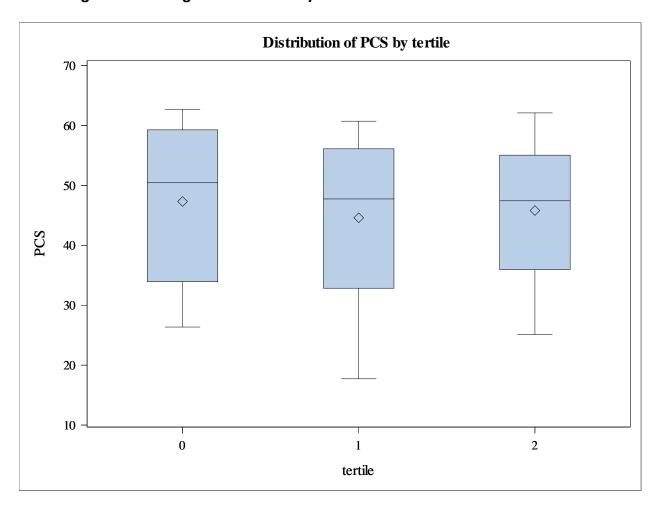
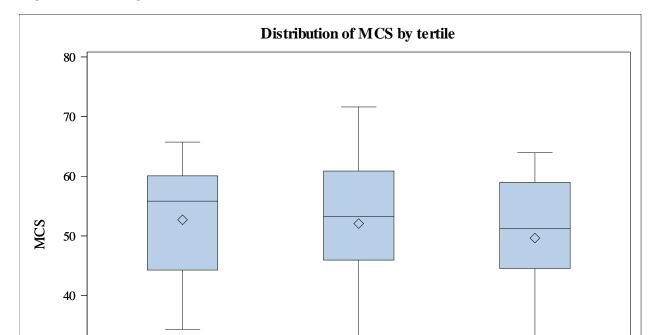


Figure 8: Average PCS results by contaminant tertile

2



tertile

Figure 9: Average MCS results by contaminant tertile

30 -

20 -

0

A. Discussion

This study provided valuable insight into how indoor air quality in homes can influence health-related quality of life for residents. The primary strength of this study was that there were multiple measures of indoor air quality inside occupants' homes along with results from psychometrically validated health-related quality of life scale. This study was able to provide quantitative results for both exposure and outcome metrics to explore associations and relationships. By utilizing actual measurements instead of modeling, a more accurate picture of true exposure levels and outcomes was achieved.

The SF-36 has been widely used across populations to quantify health-related quality of life. From 2005 to 2006 it was administered to over 3,800 adults through the National Health Measurement Study. The report resulted in a mean PCS score of 49.22 and MCS score of 53.78 [152]. The results were used to determine normal ranges within the US general population. In the results from this current study mean scores of 46.9 and 49.1 were reported for PCS and MCS respectively. Both component scores had slightly lower mean results compared to a nationally representative sample for the SF-36. However, as previously noted, the population in this study was not intended to be representative.

While this study did not find any statistically significant differences in PCS or MCS results from the SF-36 surveys between the ASHRAE 62.2 compliance groups, there were some interesting results that should be noted. The individual correlation coefficients and chi square results suggested that PCS was statistically

associated with age, asthma, education, and logCO₂. These associations showed that there were potential interactions between physical health and air quality in homes. The significance of asthma is important because it is well established in the scientific literature that indoor air quality does impact asthma in occupants. Therefore, even though there were limited findings for statistically significant interactions between PCS and individual air contaminants it can be argued that indoor air quality should be improved especially for those diagnosed with asthma.

Historically, CO₂ has been the primary pollutant of control for ASHRAE standards in indoor environments because it has been regarded as a surrogate measure of adequate outdoor air supply into the indoor environment. Based on the findings of this study, there is some evidence to suggest that reducing these levels in homes may result in improved physical responses for occupant health-related quality of life. It is important to limit indoor air pollution in homes to protect occupant health-related quality of life. It should also be noted that there was a statistically significant correlation between CO₂ levels and ASHRAE 62.2 compliance percentage.

Similarly, there were no statistical differences in MCS results between the ASHRAE 62.2 compliant and non-compliant units. There were statistically significant associations found with other predictors for MCS. In looking at bivariate correlations, MCS had a statistically significant association with the presence of nicotine in the living area and logCO₂. There was also a significant association between MCS and nicotine; however, through additional analysis it

was determined that the presence of nicotine operated as a collider. Mental health and stress are greatly impacted through neighborhood characteristics [153, 154, 155]. While there was not a statistically significant difference found with the city variable, there was evidence of variation in MCS based on geographic location. Therefore, it was probable that the presence of nicotine was acting as a collider in this regard for a variable not fully captured through the scope of this study [156]. To control for the potential of collider bias, the presence of nicotine was excluded from MCS model selection, and the city variable was included to capture the neighborhood effect.

When modeling selection was used in SAS 9.4 for the PCS outcome variable, consistent results were achieved through stepwise, forward, and backward elimination methods. The three variables that remained statistically significant in predicting PCS were asthma diagnosis, participant age, and logCO₂. Even though the ASHRAE 62.2 percent compliance variable was not included in the final models, it is still important to consider the implications of compliance given the significance of CO₂ concentrations. The results from Aim 1 above showed that ASHRAE 62.2 compliance resulted in statistically lower concentrations of CO₂ compared to the units that did not comply. Therefore, complying with the ventilation standard may still influence physical health-related quality of life.

There were some differences in the results from the three modeling selection procedures that were performed for the MCS results. The presence of

nicotine again remained significant along with logCO₂, logCOmean, and logNO₂. Again, it was probable that there was a different construct being captured by nicotine presence because the direction of the relationship was opposite to what previous literature supports. However, even though the ASHRAE 62.2 percent compliance variable remained insignificant, more individual air contaminant variables were significant in the models for MCS. There have been studies that have shown that ASHRAE 62.2 compliance does help to reduce air contaminants in homes [150]. Therefore, this research concluded that by designing homes to comply with ASHRAE 62.2 there can be improvements in occupant health-related quality of life through the improvement of indoor air quality.

A strength of this study was the examination of general trends of summed rank air contaminants in homes divided into tertiles. This allowed the ranked exposures for the units to be examined for the variables of PCS, MCS, and compliance to examine trends. There was a consistent trend for PCS, MCS, and compliance that as total ranked air contaminants increased health-related quality of life and compliance decreased. These findings support the hypothesis that controlling indoor air quality concerns is important to improve occupant health-related quality of life. It also suggests that, in general, as compliance with the ASHRAE 62.2 ventilation standard increases, the overall exposure to indoor air contaminants decreases.

This study was limited in its ability to determine how ASHRAE 62.2 compliance impacted health-related quality of life. Bivariate correlations were

calculated for both composite scores from the SF-36 scale and the numerical percent compliance with ASHRAE 62.2 and the dichotomous variable under the minimum 70 percent compliant definition. There were no significant results, and the associations were inconsistent and weak. All indoor air quality metrics were used in addition to demographic variables to control for bias during model selection. In total, there were 15 variables used to predict health-related quality of life. One variable was used for ASHRAE 62.2 compliance; however, based on the lack of associations from the bivariate calculations it was not surprising that compliance was not included in any of the final models. However, it is important to consider that some individual indoor air contaminants were significantly associated with health-related quality of life outcomes and were included in final models. In Aim 1, some of these indoor air contaminants were found to be statistically lower in ASHRAE 62.2 units such as CO₂. Therefore, ASHRAE 62.2 compliance may still have meaningful impacts on health-related quality of life.

Additional studies are needed to further explore how ASHRAE 62.2 compliance impacts health-related quality of life among occupants. Previous literature has shown a link to improved health outcomes of residents given healthy homes initiatives in dwellings [12, 150, 157]. Larger study sizes would provide greater power to detect statistical findings. It may also be that the contributions of individual indoor air contaminants were greater than the ASHRAE 62.2 percent compliance variable. For example, logCO₂ was included in the final model for both PCS and MCS. The results from Aim 1 demonstrated that ASHRAE

62.2 compliance generally resulted in lower levels of CO₂ in homes. Even though lower CO₂ concentrations were associated with increases in PCS and MCS outcomes, the reason houses had lower contaminant concentrations may be attributed to ASHRAE 62.2 compliance.

Additional recommended research was further supported through the examination of air contamination loads compared to compliance, MCS, and PCS. There was a consistent trend that increases in compliance resulted in decreases in air contaminant load as well as lower air contaminant loads in homes resulted in higher average PCS and MCS results. These trends offered important insights into how indoor air in homes were impacted through compliance with the ASHRAE 62.2 standard. It also supported the fact that resident health-related quality of life may be improved by focusing on improving indoor air quality. Again, larger study sample sizes with a diverse population would allow for a more robust analysis of these variables.

VII. DISEASE FROM MOLD AND MOISTURE EXPOSURE

A. Objective and Hypothesis

Quantify the burden of disease from pediatric asthma attributable to mold and moisture exposure in US homes. There are large monetary amounts that can be attributable to the disease burden from pediatric asthma from mold and moisture exposure in US homes.

B. Introduction

This study investigated the burden of disease that can be linked to substandard housing in the US. It is hypothesized that the housing stock in the US contributes greatly to disease and injury status but has previously been poorly enumerated in the literature. There have been minimal scientific reports regarding the etiology of housing related diseases until recently. Time studies have clearly demonstrated that the greatest exposure duration period is spent inside homes. The objective of this aim was to investigate the burden of disease from pediatric asthma given dampness and mold exposures. The working hypothesis was that there was meaningful burden of disease that could be addressed through focusing on improved housing. By completing this case-study, this methodology could be applied to additional areas in the future. The approach estimated the disease burden through Disability Adjusted Life Years (DALYs) and mortality statistics.

These burden of disease estimates were then monetized through accepted relationships between DALYs and cost to society to explore their economic impacts. These data were significant because few studies have previously looked at the diseases that can be linked to US based homes and then monetized to show the related economic burdens facing the US population. This was the first study to utilize the American Housing Survey (AHS) as a nationally representative sample of the US housing stock to assess home-based exposures such as mold and moisture.

C. <u>Justification</u>, Feasibility, and Preliminary Data

Previous literature has explored the best way to quantify disease burden from environmental disease burden factors. There has been some discussion in the literature surrounding the construct validity of DALY calculations [158, 159]. These equations have inherent assumptions and even minor changes to these variables can lead to a wide range in results [158, 159]. However, DALYs remain the preferred statistic throughout the literature. The World Bank has concluded that DALYs should be used to for ambient air pollution disease burden [160]. They also concluded that DALYs can be monetized through economic equations to further explore the cost of disease due to exposures [160]. Previous research has aimed to report a monetary value of a life year based on European Union surveys. These results have estimated that on average the value of a life year is equal to

40,000 euros or almost 47 thousand US dollars [161]. This is one indication that mortality statistics from substandard housing in the US need investigation.

Globally, asthma is a disease that impacts a large proportion of the population both in disease status and monetary expenditures. There is no cure for asthma; therefore, treatment requires continued maintenance and control of symptoms and triggers. It was estimated that over 339 million people had asthma worldwide and that asthma accounted for 23.7 million DALYs [162]. The cost of treating patients varies greatly based on the country. The lower range has been reported at an estimated 150 US dollars per patient annually in the United Arab Emirates while the upper range has bene reported at over 3,000 US dollars per patient annually in the US [162].

The CDC estimated that over 25 million people in the US alone currently have asthma [163]. This represented approximately eight percent of the total population that is impacted by this disease [163]. Almost 4.9 million of those with asthma in the US were between the ages of zero and 14 years of age [163]. It is therefore important to explore these contributing factors to asthma well as any potential interventions that may alleviate this burden since there is no cure.

There have been some studies in the scientific literature investigating how exposures in the home may contribute to occupant asthma. These studies have focused both on potential causation factors as well as exacerbation of symptoms [164]. A report of over 16,000 cases from the Respiratory Health in Northern Europe project showed that persons who lived in damp homes were more likely

to report respiratory and asthma symptoms [165]. Another cohort study in Sweden found an increase in asthma and rhinitis for adolescents who were exposed to mold or dampness in infancy [166]. An evidence review of housing interventions showed that mitigating dampness in homes and removing mold was significantly associated with a reduction in respiratory symptoms [167].

A randomized prospective study in Ohio compared two groups of asthmatic children. One group received an educational intervention while the study group also received remediation work plus education in the homes to remove sources of dampness. Researchers reported a significant improvement in asthma symptom days in the study group compared to the control group [168]. All homes in this study were eventually remediated.

Many of these aforementioned studies were included in a review conducted by Mendell et al. (2011). These researchers did not find a causal relationship between identified health outcomes and dampness or mold; however, they did find statistically significant associations [164]. The large cohorts and smaller sample sizes in their review demonstrated the existence of a potential link between mold and moisture exposure in homes and disease outcome. Emerging research investigated if there was a dose response relationship between health outcomes and indoor dampness or mold in homes [169]. Some published research has found an increased odds ratio for asthma symptoms and atopic dermatitis with increased moisture content in walls and water damage in homes [169].

The environmental burden of disease attributable to US based housing characteristics can be determined using the WHO's methodology. In 2011, the WHO published the *Environmental burden of disease associated with inadequate housing* to address ubiquitous housing quality issues and related health outcomes in Europe. This report utilized an accepted methodology to calculate the environmental burden of disease related to a health outcome for substandard housing circumstances [170]. Each chapter of the report is divided into background of the exposure component, calculation of the population attributable fraction (PAF), determination of the best relative risk (RR) factor supported by the global scientific literature of exposure and adverse health outcomes, and then the calculation of the housing related burden of disease [170].

The purpose of this research was to investigate the burden of disease from pediatric asthma from dampness and mold exposures in homes in the US following the accepted WHO methodology. This study did not attempt to assess temporal causality of asthma from mold and moisture, but instead explored the disease burden that can be attributed to these specific US housing stock conditions.

D. Research Design

This research estimated the burden of disease attributable to housing in the US through calculations of DALYs, mortality statistics, and economic equations.

The PAF needed to calculate the associated burden of disease was determined

through Equation 4 [170]. Where **p** was the proportion exposed and **RR** was the relative risk of the health outcome given the exposure. In this case, RR was used for both relative risks and odds ratios to determine the PAF.

Equation 4

$$PAF = \frac{p(RR - 1)}{p(RR - 1) + 1}$$

The American Housing Survey (AHS) was initially implemented in 1973 [171]. Its purpose is to collect a standardized series of nationally representative housing data. The AHS has core components that measure vacancies, size and composition, physical conditions, resident information, mechanical concerns, fuel supply, renovations, home costs, housing assistance, value, and recent occupant information [171]. Data are used to inform policy regarding current housing needs [171]. Results are used to help inform budgetary appropriation decisions to determine needs and allocations [171]. The survey can also be utilized to measure the effectiveness of current programs for vulnerable populations such as the elderly [171]. In 2015 and 2017 there were also questions to determine mold presence in homes. The AHS is the one of the largest nationally representative samples of the US housing stock.

The WHO formula for calculating the environmental burden of disease included the following components: the distribution of the risk factor in the

sample population, the outcome response for the identified risk factor relative risk, and the DALYs related to the risk factor [172]. The risk factor was combined with the exposure-response relationship to form the variable of impact fraction [172]. This variable represented the proportion of the risk in the population from the exposure [172].

The two estimates of disease burden in this study were mortality and DALYs. The DALY estimates for asthma were taken from the WHO Department of Measurement and Health Information published in 2009 from 2004 data [173, 174]. Mortality data was pulled from the CDC Wide-ranging online data for epidemiologic research (CDC WONDER) [175].

The exposure data for mold and moisture were determined from the 2017 AHS. The AHS is collected in odd-numbered years through US Census Bureau and supported through HUD [176]. It is considered the most representative and comprehensive housing survey for the United States [176]. Surveys were collected through computer-assisted personal interviewing and are available in English and Spanish [176].

Economic burden was assessed through published literature estimations on disease related costs. A range of estimates of dollar per DALY and mortality were collected for comparison of asthma related costs attributable to substandard housing. The total economic burden of disease from asthma was also used as an estimation point with high and low ranges.

E. Data Analysis

The WHO methodology and statistics were used to determine the epidemiology statistics of disease from exposure. Supplemental literature reviews were conducted to ensure there was no new additional information regarding odds ratios or relative risks that would largely alter the estimation calculations. Exposure prevalence to mold and moisture in homes was determined from the publicly available AHS. Point estimates were presented with 95 percent confidence intervals to provide both a lower and upper range to portray the PAF. These PAF estimates were then multiplied by the economic estimates to provide approximations of the monetary burden from asthma due to substandard housing conditions. Publicly available data from the WHO and the CDC provided the data for pediatric asthma and mortality in the US. Economic studies provided monetary estimates.

F. Results

Literature searches provided the proportion of housing exposed to mold and damp environments, DALY estimates, mortality estimates, and relative risk of developing asthma given the exposure of concern. The estimates for DALY and mortality for asthma among children aged zero to 14 in the United States of America was 333,000 DALYs in 2004 and 128 deaths in 2017 respectively [177, 178, 175].

1. Mold and Moisture in Homes

The AHS included results from 121,600 housing units that reported characteristics on housing quality in 2017, a representative sample of the approximately 137 million housing units in the US in 2017 [179, 180]. Of those units in the survey, 3,775 (3.1%) reported the presence of mold. The AHS reported 9,945 units had water leaks within the dwelling structure in 2017. This was approximately 8.2 percent of the homes that were surveyed; units that did not report were assumed to not have leaks present. The AHS also reported 12,320 units had leaks from outside the structure or 10.2 percent of the units surveyed. If these moisture sources are considered mutually exclusive the summed results were 18.4 percent of units surveyed.

Risk estimates for children developing asthma were comparable across countries during the WHO literature review process. A study by Jaakkola et al., (2005) provided a risk estimate of 2.4 (1.1, 5.6) for children who were exposed to mold in their dwellings. A case-control study from Finland by Pekkanen et al., (2007) was used as the main risk statistic for dampness [181]. The odds ratio of children developing asthma when living in damp environments was approximately 2.2 (1.3, 4.0). These risk estimates presented in the WHO methodology were also used in this present study for comparison of disease burden with US based housing data.

Below are the solved formulas from Equation 4 to determine the PAFs of asthma for mold, interior, exterior, and cumulative sources of dampness given the

exposure metrics from the AHS. The PAF for mold present in the home was 0.042. The PAF for dampness present from interior sources was 0.090 and 0.109 for exterior sources. The summed estimate of the PAF for interior and exterior water sources was 0.181.

Calculated PAF Formulas from Equation 4 for Mold and Moisture Home Exposures

Mold sources:

$$PAF = \frac{.031(2.4-1)}{.031(2.4-1)+1} = 0.042$$

Interior water sources:

$$PAF = \frac{.082(2.2-1)}{.082(2.2-1)+1} = 0.090$$

Exterior water sources:

$$PAF = \frac{.102(2.2-1)}{.102(2.2-1)+1} = 0.109$$

Interior and exterior water sources:

$$PAF = \frac{.184(2.2-1)}{.184(2.2-1)+1} = 0.181$$

The PAF was multiplied by the WHO DALY and CDC WONDER mortality statistics attributable to asthma for both males and females aged zero to 14. Mold accounted for 13,870 (1,034 low estimate; 41,732 high estimate) DALYs and approximately 5 (0.4; 16) deaths. Interior leaks accounted for 29,839 (7,997 low estimate; 65,760 high estimate) DALYs and 11 (3 low estimate; 25 high estimate) deaths. Exterior sources of dampness in housing units contributed to 36,198 (9853 low estimate; 77,808 high estimate) DALYs and almost 14 (4 low estimate; 30 high estimate) deaths in 2008. A combined statistic for interior and exterior sources of

moisture combined in the home contributed to 60,136 (17,389 low estimate; 118,296 high estimate) DALYs and 23 (7 low estimate; 45 high estimate) deaths.

2. Monetization of Environmental Burden of Disease

Literature searches have reported several estimates as to the cost of asthma and mortality. One study found that the direct, indirect, and societal cost per asthmatic per year was approximately 3,100 dollars in the US. [182] This is similar to another study that found 3,259 dollars were spent per asthmatic per year. [183] These studies also reported a total cost of asthma to be 56 billion dollars societal cost from asthma and a mortality cost of 2.1 billion dollars and morbidity at 3.8 billion dollars from lost productivity, based on 2009 US dollars. [183]. Another study aimed to quantify the total cost of asthma between the years 2008 to 2013. They found that roughly 33 percent of the asthmatic population was between the ages of zero to 14 [184]. The CDC most recent national asthma data reported that this population was approximately 19 percent in 2017 [163]. For this current study, the most recent CDC estimate was used to align with the recent AHS prevalence estimates of mold and moisture in dwellings.

All monetary estimations were calculated using 2009 dollars from Barnett and Nurmagambetov (2011). For the study population of asthmatics aged zero to 14, the monetary estimations were 10.6 billion dollars for the total cost of asthma with 722 million dollars attributed to morbidity and 399 million dollars attributed to mortality in 2009 dollars. These estimates were used to determine the burden

that could be attributed to substandard housing of mold and moisture exposure in US homes. In total, based on AHS results, moisture inside the home accounted for an estimated 953 million dollars with morbidity and mortality estimates being 64.7 million dollars and 3.58 million dollars, respectively. Moisture from exterior sources was 1.16 billion dollars for total asthma costs, 136 million dollars for asthma morbidity, and 4.34 million dollars for asthma mortality. The combined moisture estimations were 1.92 billion dollars for the total cost of asthma with 130 million due to morbidity and 72.1 million attributable to mortality. Mold accounted for 443 million dollars for total asthma cost with 30.1 million and 16.6 million from morbidity and mortality, respectively.

The figures below depict the costs of asthma that can be attributed to exposure to mold and moisture in the US housing stock for children under 15 years old separated by the following four housing conditions: dampness from interior sources, dampness from exterior sources, dampness from combined sources, and mold. Figure 11 shows the cost of asthma for children aged zero to 14 in billions of 2009 dollars by housing exposure. Figure 12 shows the cost of asthma morbidity for children aged zero to 14 in billions of 2009 dollars by housing exposure. Figure 13 shows the cost of asthma mortality for children aged zero to 14 in billions of 2009 dollars by housing exposure.

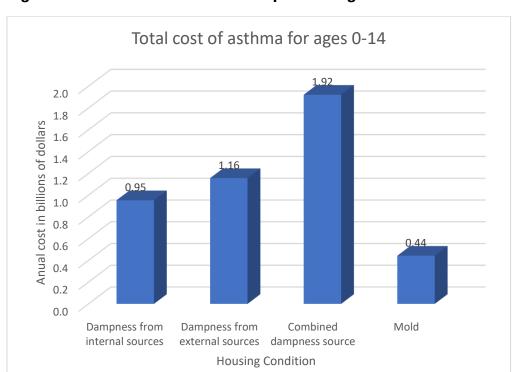


Figure 10: Total cost of asthma for persons aged 0 to 14

Figure 11: Cost of asthma due to morbidity from mold and moisture exposure in homes

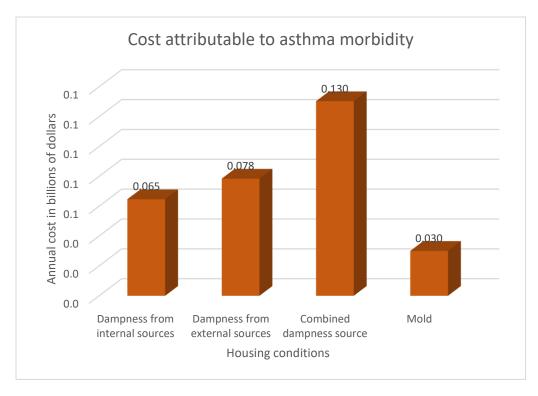
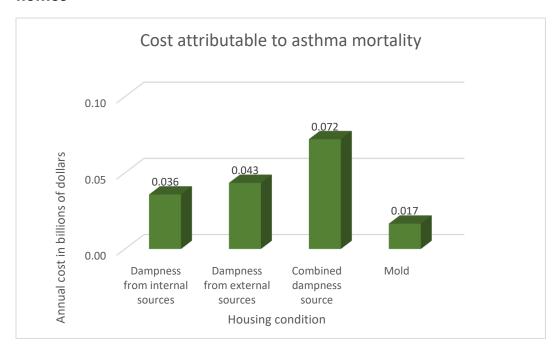


Figure 12: Cost of asthma due to mortality from mold and moisture exposure in homes



G. Discussion

This study showed the potential fraction of cost attributable to substandard housing in the US due to mold and moisture in homes. The total cost of asthma for those aged 0 to 14 was almost 2.4 billion dollars for mold and moisture in homes. This is a staggering figure that justifies exploring interventions that can address these issues in the housing stock to reduce this monetary burden. As previously cited, some research estimates the total societal cost of asthma to be approximately 56 billion dollars [183]. There was a large monetary and public health problem from childhood asthma that required investigation into ways to alleviate the burden. This study presented the specific asthma burden for children aged 0-14 that can be attributed to mold and moisture exposure in homes.

The WHO study resulted in substantial burden of disease estimates for European member countries. The housing exposure statistics were slightly higher from the AHS report than the estimation used from the European data. The environmental exposures were estimated at a median prevalence of dampness in homes at 15 percent which would be closest to this study's 18 percent estimate of combined exterior and interior sources of water in the home, which is fairly good agreement in estimations [181]. This would suggest that the burden presented in this study by the individual interior and exterior sources of water are conservative estimations. It also supports that a combined estimate of interior and exterior water sources from the AHS may be closer to the actual exposure of dampness in homes by occupants than considering each factor separately.

Mold was also reported to occur in 10 percent of homes based on European data while this study only found approximately three percent of US homes reported mold. These variations in conditions could be based on climate, structural composition, and age of housing stock. Mold and dampness may both be present in dwellings which is not accounted for in this current study. They are examined as separate entities to explore monetary burden of disease from each environmental exposure associated with childhood asthma. This allows for policy decisions to be determined as to where interventions should be targeted. However, there interventions aimed at addressing mold and moisture in homes have meaningful crossover as moisture is critical in the mold development pathway.

The European report from the WHO estimated mortality ranging from 72 to almost 156 deaths attributable to dampness in homes in 2008 [181]. The US estimates for mortality ranged from approximately seven to 45 when looking at combined potential sources of dampness inside the homes. The European DALY estimate for 2008 ranged from 48,578 to 104,874 [181]. This current study reported a combined interior and exterior dampness sources DALY estimate of US homes of 17,389 to 118,296. The mortality and DALY estimates could be higher in the WHO report due to the greater population pool in the WHO study and the lower exposure prevalence reported in the AHS.

This current study was presented with limitations and potential introductions of bias as well as strengths. This current research used the AHS

because it has been shown to be a representative national sample. By using the AHS this study was able to provide the most accurate estimations for nationwide exposure data in housing units while other studies utilized other exposure estimates from samples that have not been shown to represent the US housing stock. This study also examined mold and dampness as two separate entities in accordance with the prescribed WHO methodology. To date, most scientific inquiry has grouped these two variables together since it can be argued that mold does not exist without the presence of moisture. It is possible that homes experience both dampness and microbial growth simultaneously.

The study by Mudarri and Fisk (2007) argued that results from the AHS are conservative and underestimate the prevalence of exposures in US housing stock. They used other studies to estimate the prevalence of moisture and mold in homes. However, it is unknown how nationally representative the other studies are. Therefore, it could also be argued that their study overestimates the prevalence of mold and moisture in US homes. The report also examined the presence of mold and moisture as a single exposure metric. Their study found that 47 percent of homes in the US have mold and/or excessive moisture present [185]. They reported approximately 400 million dollars for mortality, 2.6 billion dollars from morbidity, and 3.5 billion dollars from total asthma costs in 2004 dollars could be attributable to mold in moisture in homes [185]. These estimates are not that far removed from the results presented in this study even though the

researchers used combined statistics for mold and moisture and included total asthmatic population in the US.

There were a few limitations and potential introduction of bias faced by this study. Housing conditions may not be accurately reported in the AHS leading to inconsistent estimations of exposure prevalence for mold and moisture. Housing conditions also change over time leading to variance in exposure status. The AHS only asked respondents about water leakage and mold in the dwelling during the last 12 months [171]. It is possible that previous exposures to mold or dampness in the homes were not captured by the most recent AHS results especially since mold is not a regular item that is tracked.

The formulas used in these analyses were also susceptible to great variation in results from inherent assumptions and risk characterization. Even a small change in the RR inputs result in great differences in burden of disease estimates. This is evident in the statistics reported for the low, medium, and high RR inputs that were used to look at morbidity and DALY estimates. While variation is inevitable, it is important to realize that this study utilized previously validated methods and risk estimates supported by peer reviewed scientific literature to ensure robust estimations.

This study also used the assumption of occupancy by the target population from homes surveyed in the AHS. This assumption was necessary for the completion of the estimation equations. However, it was probable that units surveyed were not occupied by persons zero to 14. It was also probable that some

units had more than one person from the target population. Therefore, the exposure metrics used in this study had some inherent biases.

Based on the results of this study, the burden of disease caused by asthma among children aged zero to 14 in the US can be partially attributed to dwelling conditions. The estimates presented in this study demonstrate that DALYs and mortality from asthma among US children could be alleviated through homebased interventions. Most local housing codes cover mold and leaks under building violations through local adaptations of the International Property Maintenance Code. By addressing housing issues within the US, it is possible to improve the health of children as well as provide meaningful return on investment for housing stock repairs. This is an important policy issue when considering resource allocation.

This study also highlighted several areas for additional research. There is no standard definition for defining mold and moisture exposure in homes.

Definitions that were standardized across studies, surveillance, and remediation initiatives would increase data integrity. There is also a need to comprehensively evaluate the cost of mold and moisture remediation in homes to conduct a cost benefit analysis. This study demonstrated the disease burden and monetary consequences for asthma among persons aged zero to 14 from mold and moisture. However, it did not investigate other age groups or other confounding variables. It also did not look at other disease outcomes or housing exposures.

VIII. CONCLUSION

The environment comprised of housing conditions represents an important field of scientific investigation needed to protect and promote public health. This is supported by the belief that everyone has the right to a healthy dwelling.

National and international organizations agree that a healthy home is essential in ensuring occupant health and well-being. This concept is included in Article 25(1) of the Universal Declaration of Human Rights [186]. This dissertation research epitomizes an important contribution to support the benefits of focusing on healthy homes for occupants.

As exhibited throughout the report, there is no standardized definition of a healthy home although a few have been proposed. This makes comparing and generalizing findings difficult in the scientific literature. In addition, previous research studies have had relatively small sample sizes with an n around 30. Therefore, each study is uniquely important as it can offer guidance as to what aspects make homes healthy and offer the greatest benefit. The concept of what makes a home healthy has evolved through the scientific literature and continues to be influenced based on results from studies similar to the one presented here. Most direct exposure studies have historically been relatively small in sample sizes. Therefore, additional studies like this current one is critical to better understand home-based exposures.

The research presented here offered several unique strengths. There was a moderate sample size of 85 homes in the Chicago metropolitan area which greatly

increased the ability to describe the presence and magnitude of air contaminants in homes as well as offer greater power to examine statistical inferences. It was one of the few studies that measured ASHRAE 62.2 compliance as a variable. In addition, this study was able to quantitatively assess indoor air contaminants as well as health-related quality of life outcomes for occupants. It was also able to monetize direct and indirect costs associated with substandard housing conditions. Occupant health, well-being, and economic benefits of healthy homes are corner stones for supporting additional initiatives.

There were several key findings presented in this study. First, ASHRAE 62.2 does improve indoor air quality in homes. This study was able to find some statistically significant differences for specific contaminants, and it also showed a general decrease in indoor air contaminants with increased ASHRAE 62.2 compliance. There was evidence that indoor air quality impacted health-related quality of life for adult occupants. Specifically, the concentration of CO₂ seemed to be consistently linked to both PCS and MCS results from the SF-36 scale. There was additional evidence that increased CO₂ and NO₂ predicted lower MCS scores. While ASHRAE 62.2 compliance was not statistically associated with either health-related quality of life outcomes, it was associated with CO₂ levels in the home. Therefore, it can be argued that ASHRAE 62.2 compliance did have an impact on occupant health-related quality of life.

This study also enumerated the DALY, YLL, and monetary estimates attributed to pediatric asthma from substandard housing conditions in the US.

This research used scientifically proven methodology to implement a case study to develop estimates of attributable disease burden from pediatric asthma from mold and moisture exposure in homes. The results from this study were an important step for proof of concept. The AHS has been greatly underutilized in current literature and it remains the only nationally representative sample of the US housing stock. Additional exposure prevalence data can be pulled from the AHS and compared to other disease prevalence rates to further explore how substandard housing in the US is contributing to DALYs and YLLs. These disease estimates can be combined with economic estimates to determine the monetary burden that can be attributed to substandard housing conditions.

While this study offered important contributions to the current literature, it also pointed to additional needs. Larger sample size studies are needed to continue to explore ASHRAE 62.2 compliance on indoor air contaminants and health-related quality of life of occupants. By design, approximately half of the population enrolled in this study were thought to have zero ASHRAE 62.2 compliance. This meant that the compliance variable did not have as great of variation for statistical exploration. Larger and more diverse samples of both housing characteristics and occupants would allow for greater generalizability of findings.

The estimates of time spent in homes consistently presented in the literature are now considered unrepresentative of true exposure time and duration in response to the COVID-19 pandemic. There were orders for populations to

remain at home and restrict travel even to schools and workplaces beyond what was considered essential. Therefore, people's home-based exposures have become much greater and more important than ever to address. There are already reports showing increases in childhood lead poisoning even though screening rates have fallen drastically during the pandemic [187]. These early reports could serve as sentinel events portraying the greater importance of focusing on keeping the indoor home environment healthy for all. Studies like this one are more important than ever to explore the indoor environment and provide additional insight to the current scientific literature.

Based on comparison values alone, this study found that some indoor air contaminants were exceeded in study homes. All homes sampled had formaldehyde levels greater than 7 ppb and over half of the homes had levels of NO₂ greater than 21 ppb. Without formal limits established, it is difficult to properly address these areas of concern in homes. There has been a recent push through independent and local organizations to address similar obstacles by recommending standardized threshold values. Some cities in California have already banned natural gas appliances in new construction homes and the state is aiming to do the same [188]. This is intervention is aimed at reducing pollution generating sources. Greater exposure studies are needed to better describe indoor air contamination in homes and universal standards are needed to protect occupant health.

There are many physical and mental health outcomes that have been linked to exposures in the home. There should be more guidance and standardization for recommended exposure limits in homes for air contaminants. There are levels for occupational exposures and ambient exposures, but there is no consistent benchmark for which to compare indoor air contaminants in homes to. The literature provides some guidance and recommendations; however, these are usually presented as a wide range or modeled based on exposure distribution rather than based on health or wellbeing outcomes. By establishing indoor air contaminant limits in homes, it would be possible to improve exposure and risk assessments of occupants. It would also allow for implementation of interventions and evaluation of those interventions.

CITED LITERATURE

- 1 K. R. Smith, C. F. Corvalán and T. Kjellström, "How much global ill health is attributable to environmental factors," *Epidemiology*, vol. 10, no. 5, pp. 509-515, 1999.
- 2 S. T. Holgate, E. Davies, R. M. Powell, P. H. Howarth, H. M. Haitchi and J. W. Holloway, "Local genetic and environmental factors in asthma disease pathogenesis: Chronicity and persistence mechanisms," *European Respiratory Journal*, vol. 29, pp. 7830=-803, 2007.
- 3 C. Sengler, S. Lau, U. Wahn and R. Nickel, "Interactions between genes and environmental factors in asthma and atopy: New developments," *Respiratory Research*, vol. 7, 2001.
- 4 A. B. Mukherjee and Z. Zhang, "Allergic asthma: Influence of genetic and environmental factors," *Journal of Biological Chemistry*, vol. 286, no. 38, pp. 32883-32889, 2011.
- World Health Organization Europe, "Air Quality Guidelines: Global Update 2005," 2006. [Online]. Available: https://www.euro.who.int/__data/assets/pdf_file/0005/78638/E90038.pdf.
- 6 A. H. Maslow, "A theory of human motivation," *Psychological Review,* vol. 50, no. 4, pp. 370-396, 1943.
- Office of the United States Surgeon General, "The Surgeon General's Call to Action to Promote Healthy Homes," Rockville, MD, 2009. [Online]. Available https://www.ncbi.nlm.nih.gov/books/NBK44192/pdf/Bookshelf_NBK44192.pdf.
- A. Moreland, C. Herlihy, M. A. Tynan, G. Sunshine, R. McCord, C. Hilton and et. al., "Timing of the state and territorial COVID-19 stay-at-home orders and changes in population movement- United States, March 1- May 21, 2020," *Morbidity and Mortality Weekly Report*, vol. 69, no. 355, pp. 1198-1203, 2020.
- 9 J. Q. Koenig, T. F. Mar, R. W. Allen, K. Jansen, T. Lumley, J. H. Sullivan, C. A. Trenga, T. Larson and L. J. Liu, "Pulmonary effects of indoor- and outdoor-generated particles in children with asthma," *Environmental Health Perspectives*, vol. 113, no. 4, pp. 499-503, 2005.

- 10 L. K. Baxter, T. M. Barzyk, A. F. Vette, C. Croghan and R. W. Williams, "Contributions of diesel truck emissions to indoor elemental carbon concentrations in homes in proximity to Ambassador Bridge," *Atmospheric Environment*, vol. 42, no. 40, pp. 9080-9086, 2008.
- 11 S. S. Mitter, R. Vedanthan, F. Islami, A. Pourshams, H. Khademi, F. Kamangar, C. C. Abnet, S. M. Dawsey, P. D. Pharoah, P. Brennan, V. Fuster, P. Boffetta and R. Malekzadeh, "Household fuel use and cardiovascular disease mortality: Golestan cohort study," *Circulation*, vol. 133, pp. 2360-2369, 2016.
- 12 A. P. Jones, "Indoor air quality and health," *Atmospheric Environment,* vol. 33, no. 28, pp. 4535-4564, 1999.
- 13 T. Gotschi, L. Oglesby, P. Mathys, C. Monn, N. Manalis, K. Koistinen, M. Jantunen, O. Hanninen, L. Polanska and N. Kunzli, "Comparison of black smoke and PM2.5 levels in indoor and outdoor environments of four European cities," *Environmental Science & Technology*, vol. 36, no. 6, pp. 1191-1197, 2002.
- 14 International Agency for Research on Cancer, Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. 105, 2013. [Online]. Available: https://www.ncbi.nlm.nih.gov/books/NBK294269/pdf/Bookshelf_NBK294269.pdf.
- 15 M. H. Sherman and N. E. Matson, "Reducting indoor residential exposures to outdoor pollutants," 2003.
- 16 R. Williams, J. Suggs, A. Rea, L. Sheldon, C. Rodes and J. Thornburg, "The Research Triangle Park particulate matter panel study: Modeling ambient source contribution to personal and ambient residential PM mass concentrations," *Atmospheric Environment*, vol. 37, no. 38, pp. 5365-5378, 2003.
- 17 A. Kousa, L. Oglesby, K. Koistinen, N. Kunzli and M. Jantunen, "Exposure chain of urban air PM2.5: Associations between ambient fixed site, residential outdoor, indoor, workplace and personal exposures in four European cities in the EXPOLIS-study," *Atmospheric Environment*, vol. 36, pp. 3031-3039, 2002.
- 18 A. Dėdelė and A. Miškinytė, "Seasonal variation of indoor and outdoor air quality of nitrogen dioxide in homes with gas and electric stoves," *Environmental Science and Pollution Research International*, vol. 23, no. 17, pp. 17784-92, 2016.
- 19 M. M. Abdel-Salam, "Seasonal variation in indoor concentrations of air pollutants in residential buildings," *Journal of the Air and Waste Management Association*, 2021.

- 20 U. Satish, M. J. Mendell, K. Shekhar, T. Hotchi, D. Sullivan, S. Streufert and et al., "Is CO2 an indoor pollutant? Direct effects of low-to-moderate CO2 concentrations on human decision-making performance," *Environmental Health Perspectives*, vol. 120, no. 12, pp. 1671-1677, 2012.
- 21 D. W. Bearg, Indoor Air Quality and HVAC Systems, CRC Press, 1993.
- 22 S. Laville, "Ella Kissi-Debrah: How a mother's fight for justice may help prevent other air pollution deaths," *The Guardian,* 16 December 2020.
- World Health Organization (WHO), WHO guidelines for indoor air quality: Selected pollutants, Copenhagen, Denmark, 2010. [Online]. Available: https://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf. [Accessed 5 November 2020].
- 24 Environmental Protection Agency (EPA), "Indoor Air Quality (IAQ)," [Online]. Available: https://www.epa.gov/indoor-air-quality-iaq/what-average-level-carbon-monoxide-homes. [Accessed 5 November 2020].
- 25 National Center for Biotechnology Information, National Library of Medicine, "Compound Summary: Formaldehyde," PubChem, [Online]. Available: https://pubchem.ncbi.nlm.nih.gov/compound/Formaldehyde. [Accessed 5 November 2020].
- 26 Agency for Toxic Substances and Disease Registry (ATSDR), "Toxicological profile for formaldehyde," US Department of Health and Human Services, Atlanta, 1999. [Online]. Available:https://www.atsdr.cdc.gov/toxprofiles/tp111.pdf. [Accessed 5 November 2020].
- 27 T. Salthammer, S. Mentese and R. Marutzky, "Formaldehyde in the indoor environment," *Chemical Reviews*, vol. 110, pp. 2536-2572, 2010.
- 28 T. J. Kelly, D. L. Smith and J. Satola, "Emission rates of formaldehyde from materials and consumer products found in California homes," *Environmental Science and Technology*, vol. 33, pp. 81-88, 1999.
- 29 National Center for Biotechnology Information, National Library of Medicine, "Compound Summary: Carbon monoxide," PubChem, [Online]. Available: https://pubchem.ncbi.nlm.nih.gov/compound/281. [Accessed 5 November 2020].

- 30 Agency of Toxic Substances and Disease Registry (ATSDR), "Toxicological Profile for Carbon Monoxide," US Department of Health and Human Services, Atlanta, 2012.
- 31 National Center for Biotechnology Information, National Library of Medicine, "Compound Summary: Carbon dioxide," PubChem, [Online]. Available: https://pubchem.ncbi.nlm.nih.gov/compound/280. [Accessed 5 November 2020].
- 32 Environmental Protection Agency, "Overview of greenhouse gases: Carbon dioxide emissions," [Online]. Available: https://www.epa.gov/ghgemissions/overview-greenhouse-gases#carbon-dioxide. [Accessed 5 November 2020].
- 33 A. Rodero and D. A. Krawczyk, "Carbon dioxide human gains: A new approach of the estimation," *Sustainability*, vol. 11, no. 24, pp. 7128-7140, 2019.
- 34 L. Yang, X. Wang, M. Li, X. Zhou, S. Liu, H. Zhang and et. al., "Carbon dioxide generation rates of different age and gender under various activity levels," *Building and Environment,* vol. 186, 2020.
- 35 C. A. Erdmann, K. C. Steiner and M. G. Apte, "Indoor carbon dioxide concentrations and sick building syndrome symptoms in the BASE study revisited analyses of the 100 building dataset," *Indoor Air,* vol. 3, pp. 443-448, 2002.
- 36 M. G. Apte, W. J. Fisk and J. M. Daisey, "Associations between Indoor CO2 concentrations and sick building syndrome symptoms in U.S. office buildings: An analysis of the 1994-1996 BASE study data," *Indoor Air,* vol. 10, pp. 246-257, 2000.
- 37 D.-H. Tsai, J.-S. Lin and C.-C. Chan, "Office workers' sick building syndrome and indoor carbon dioxide concentrations," *Journal of Occupational and Environmental Hygiene*, vol. 9, no. 5, pp. 345-351, 2012.
- 38 M. G. Apte, W. J. Fisk and J. M. Daisey, "Associations between indoor CO2 concentrations and sick building syndrome symptoms in US office buildings: An analysis of the 1994-1996 BASE study data," *Indoor Air,* vol. 10, pp. 246-257, 2000.
- 39 A. Afshari, U. Matson and L. E. Ekberg, "Characterization of indoor sources of fine and ultrafine particles: A study conducted in a full-scale chamber," *Indoor Air,* vol. 15, pp. 141-150, 2005.

- 40 D. Mueller, J. Schulze, H. Ackermann, D. Klingelhoefer, S. Uibel and D. A. Groneberg, "Particulate matter (PM) 2.5 levels in ETS emissions of a Marlboro Red cigarette in comparison to the 3R4F reference cigarette under open- and closed-door condition," *Journal of Occupational Medicine and Toxicology*, vol. 26, no. 7, 2012.
- 41 G. Invernizzi, A. Ruprecht, R. Mazza, E. Rossetti, A. Sasco, S. Nardini and et al., "Particulate matter from tobacco versus diesel car exhaust: an educational perspective," *Tobacco Control,* vol. 13, no. 3, pp. 219-221, 2004.
- 42 U.S. Department of Housing and Urban Development, "HUD's Healthy Homes Program," [Online]. Available: https://portal.hud.gov/hudportal/HUD?src=/program_offices/healthy_homes/hhi.
- 43 P. J. Ashley, "HUD's Healthy Homes Program: Progress and future directions," *Journal of Environmental Health,* vol. 78, no. 2, pp. 50-53, 2015.
- 44 Centers for Disease Control and Prevention, "Healthy Homes," 16 March 2015. [Online]. Available: https://www.cdc.gov/nceh/lead/healthyhomes.htm.
- 45 National Center for Healthy Housing, "NCHH: 25 Years of Better Housing, Better Health," 2017. [Online]. Available: http://www.nchh.org/WhoWeAre/NCHHs25thAnniversary.aspx.
- 46 National Environmental Health Association, "HHS Credential," [Online]. Available: https://neha.org/professional-development/credentials/hhs-credential.
- 47 Enterprise Community Partners, "What we do," 2017. [Online]. Available: http://www.enterprisecommunity.org/about/what-we-do.
- 48 Enterprise Community Partners, "Green Communities," 2017. [Online]. Available: http://www.enterprisecommunity.org/solutions-and-innovation/green-communities.
- 49 Enterprise, Enterprise Green Communities Criteria, Enterprise Community Partners, Inc., 2015.
- 50 American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE), "ANSI/ASHRAE Standard 62.2-16: Ventilation and Acceptable Indoor Air Quality in Residential Buildings," Atlanta, 2016.

- 51 L. Zhong, M. S. Goldberg, Y.-T. Gao and F. Jin, "Lung cancer and indoor air pollution arising from Chinese-style cooking among nonsmoking women living in Shanghai, China," *Epidemiology*, vol. 10, no. 5, pp. 488-484, 1999.
- D. W. Dockery, A. Pope, X. Xu, J. D. Spengler, J. H. Ware, M. E. Fay, B. G. Ferris, Jr. and F. E. Speizer, "An association between air pollution and mortality in six U.S. cities," *New England Journal of Medicine*, vol. 329, pp. 1753-1759, 1993.
- 53 G. Hoek, R. M. Krishnan, R. Beelen, A. Peters, B. Ostro, B. Brunekreef and J. D. Kaufman, "Long-term air pollution exposure and cardio- respiratory mortality: A review," *Environmental Health*, vol. 12, no. 43, pp. 1-15, 2013.
- 54 T. K. Takaro, J. Krieger, L. Song, D. Sharify and N. Beaudet, "The Breathe-Easy Home: The impact of asthma-friendly home construction on clinical outcomes and trigger exposure," *American Journal of Public Health,* vol. 101, no. 1, pp. 55-62, 2011.
- 55 N. E. Kleepis, W. C. Nelson, W. R. Ott, J. P. Robinson, A. M. Tsang, P. Switzer and et al., "The National Activity Pattern Survey (NHAPS): A resource for assessing exposure to environmental pollutants," *Journal of Exposure Analysis and Environmental Epidemiology*, vol. 11, pp. 231-252, 2011.
- 56 International Agency for Research on Cancer (IARC), Outdoor Air Pollution, vol. 109, Lyon, France, 2013.
- 57 Environmental Protection Agency (EPA), "The total exposure assessment methodology (TEAM) study: Summary and analysis," Washington, DC, 1987.
- National Center for Healthy Housing (NCHH), "2013 State of Healthy Housing," Columbia, MD, 2013. [Online]. Available: https://nchh.org/tools-and-data/data/state-of-healthy-housing/.
- 59 World Health Organization (WHO), "Burden of Disease from Household Air Pollution for 2012," Geneva, Switzerland, 2014.
- 60 World Health Organization (WHO), "The Right to Healthy Indoor Air: Report on a WHO Meeting," Netherlands, 2000.
- 61 I. Annessi-Maesano, D. Moreau, D. Caillaud, F. Lavaud, Y. Le Moullec, A. Taytard, G. Pauli and D. Charpin, "Residential proximity fine particles related to allergic sensitisation and asthma in primary school children," *Respiratory Medicine*, vol. 101, no. 8, pp. 1721-1729, 2007.

- 62 G. B. Diette, N. N. Hansel, T. J. Buckley, J. Curtin-Brosnan, P. A. Eggleston, E. C. Matsui, M. C. McCormack, D. L. Williams and P. N. Breysse, "Home indoor pollutant exposures among inner-city children with and without asthma," Environmental Health Perspectives, vol. 115, no. 11, pp. 1665-1669, 2007.
- 63 P. Lajoie, D. Aubin, V. Gingras, P. Daigneault, F. Ducharme, D. Gauvin, D. Fugler, J. M. Leclerc, D. Won, M. Courteau, S. Gringas, M. È. Héroux, W. Yang and H. Schleibinger, "The IVAIRE project A randomized controlled study of the impact of ventilation on indoor air quality and the respiratory symptoms of asthmatic children in single family homes," *Indoor Air*, vol. 25, no. 6, pp. 582-597, 2015.
- 64 V. Lazenby, A. Hinwood, A. Callan and P. Franklin, "Formaldehyde personal exposure measurements and time weighted exposure estimates in children," *Chemosphere*, vol. 88, pp. 966-973, 2012.
- 65 C. P. Weisel, J. Zhang, B. J. Turpin, M. T. Morandi, S. Colome, T. H. Stock and et al., "Relationships of indoor, outdoor, and personal air (RIOPA). Part I. Collection methods and descriptive analyses," *Research Report (Health Effects Institute)*, vol. 130, no. 1, pp. 1-107; discussion 109-27, 2005.
- 66 P. Gustafson, L. Barregård, R. Lindahl and G. Sällsten, "Formaldehyde levels in Sweden: personal exposure, indoor, and outdoor concentrations," *Journal of Exposure Analysis and Environmental Epidemiology*, vol. 15, no. 3, pp. 252-60, 2005.
- 67 National Institute for Occupational Safety and Health (NIOSH), "Flavoring Related Lung Disease," Centers for Disease Control and Prevention, 28 June 2018. [Online]. Available: https://www.cdc.gov/niosh/topics/flavorings/limits.html. [Accessed 27 October 2020].
- 68 Environmental Protection Agency (EPA), "NAAQS Table," 2016. [Online]. Available: https://www.epa.gov/criteria-air-pollutants/naaqs-table. [Accessed 30 September 2020].
- 69 S. Wilbur, M. Williams, R. Williams, F. Scinicariello, J. M. Klotzbach, G. L. Diamond and et. al., Toxicological Profile for Carbon Monoxide, Atlanta: Agency for Toxic Substances and Disease Registry (ATSDR), 2012.
- 70 Occupational Safety and Health Administration, "OSHA Annotated Table Z-1," US Department of Labor, 2019. [Online]. Available: https://www.osha.gov/dsg/annotated-pels/tablez-1.html. [Accessed 27 October 2020].

- 71 Agency for Toxic Substances and Disease Registry (ATSDR), "Minimal Risk Levels (MRLs)," CDC, 2018. [Online]. Available: https://www.atsdr.cdc.gov/mrls/index.html. [Accessed 30 September 2020].
- 72 Environmental Protection Agency (EPA), "Formaldehyde; CASRN 50-00-0," Integrated Risk Information System (IRIS), 1990.
- 73 U.S. Environmental Protection Agency, "Basic Information about the Integrated Risk Information System," 21 September 2020. [Online]. Available: https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system. [Accessed 27 October 2020].
- 74 California Office of Environmental Health Hazard Assessment, "Appendix D. Individual Acute, 8-Hour, and Chronic Reference Exposure Level Summaries," 2014.
- 75 J. Spengler, M. Schwab, P. B. Ryan, S. Colome, A. L. Wilson, I. Billick and et al., "Personal exposure to nitrogen dioxide in the Lose Angeles Basin," *Air & Waste,* vol. 44, no. 1, pp. 39-47, 1994.
- 76 National Institute for Occupational Safety and Health (NIOSH), "Carbon dioxide," 30 October 2019. [Online]. Available: https://www.cdc.gov/niosh/npg/npgd0103.html. [Accessed 17 December 2020].
- 77 The American Society of Heating, Refrigerating and Air-Conditioning Engineers, "What is the allowable level of carbon dioxide in an occupied space?," in *ASHRAE Technical FAQ: 35*, 2016.
- 78 Environmental Protection Agency (EPA), "Respiratory health effects of passive smoking: Lung cancer and other disorders," Washington, D.C., 1992.
- 79 World Health Organization (WHO) for Europe, Air Quality Guidelines for Europe, 2 ed., Copenhagen, 2000.
- 80 M. Sherman, "How ASHRAE set the rates for residential ventilation," *ASHRAE Journal*, pp. 20-23, 2015.
- 81 Residential Building Systems, Ventilate Right: Ventilation Guide for New and Existing California Homes, Berkeley: U.S. Department of Energy.
- 82 M. Karimi and J. Brazier, "Health, health-related quality of life, and quality of life: What is the difference?," *PharmacoEconomics*, vol. 34, pp. 645-649, 2016.

- 83 Center for Disease Control and Prevention (CDC), "Health-Related Quality of Life (HRQOL)," 31 October 2018. [Online]. Available: https://www.cdc.gov/hrqol/index.htm. [Accessed 29 January 2021].
- 84 Healthy People 2020, "Foundation Health Measurement Report: Health-Related Quality of Life and Well-Being," 2010.
- 85 Centers for Disease Control and Prevention (CDC), "HRQOL Concepts," 31 October 2018. [Online]. Available: https://www.cdc.gov/hrqol/concept.htm. [Accessed 29 January 2020].
- 86 D. Khanna and J. Tsevat, "Health-related quality of life: An introduction," *Rheumatoid Arthritis,* vol. 13, no. 9-S, 2007.
- 87 RAND Corporation, "36-Item Short Form Survey (SF-36)," [Online]. Available: https://www.rand.org/health/surveys_tools/mos/36-item-short-form.html.
- 36): I. Conceptual framework and item selection," *Medical care,* vol. 30, no. 6, pp. 473-483, 1992.
- 89 J. E. Ware, M. Kosinski and S. D. Keller, "SF-36 physical and mental health summary scales: A user's manual," Health Assessment Lab, Boston, MA, 1994.
- 90 J. E. Brazier, J. M. B. Jones, A. O'Cathain, K. J. Thomas, T. Usherwood and L. Westlake, "Validating the SF-36 health survey questionnaire: New outcome measure for primary care," *British Medical Journal*, vol. 305, pp. 160-164, 1992.
- 91 C. A. McHorney, J. E. Ware and A. E. Raczek, "The MOS 36-item short-form health survey (SF-36): II. Psychometric and clinical tests of validity in measuring physical and mental health constructs," *Medical Care*, vol. 31, no. 3, pp. 247-263, 1993.
- 92 H. Wittink, D. Turk, D. Carr, A. Sukiennik and W. Rogers, "Comparison of the redundancy, reliability, and responsiveness to change among SF-36, Oswestry Disability Index, and Multidimensional Pain Inventory," *Clinical Journal of Pain*, vol. 20, no. 3, pp. 133-142, 2004.
- 93 M. E. (. Maruish, User's manual for the SF-36v2 Healthy Survey, vol. 3rd ed, Lincoln, RI: QualityMEtric Incorporated, 2010.

- 94 E. O. Meltzer, R. A. Nathan, J. C. Seiner and W. Storms, "Quality of life and rhinitic symptoms: Results of a nationwide survey with the SF-36 and RQLQ questionnaires," *Journal of Allergy and Clinical Immunology,* vol. 99, no. 6, pp. S815-S819, 1997.
- 95 E. F. Juniper, G. R. Norman, F. M. Cox and J. N. Roberts, "Comparison of the standard gamble, rating scale, AQLQ and SF-36 for measuring quality of life in asthma," *European Respiratory Journal*, vol. 18, pp. 38-44, 2001.
- 96 J. Bousquet, J. Knani, H. Dhivert, A. Richard, A. Chicoye, J. E. Ware and F. B. Michel, "Quality of life in asthma. I. Internal consistency and validity of the SF-36 questionnaire.," *American Journal of Respiratory and Critical Care Medicine*, vol. 149, no. 2, pp. 371-375, 1994.
- 97 RAND Corporation, "36-Item Short Form Survey (SF-36) Scoring Instructions," [Online]. Available: https://www.rand.org/health/surveys_tools/mos/36-item-short-form/scoring.html.
- 98 J. M. Logue, N. E. Klepeis, A. B. Lobscheid and B. C. Singer, "Pollutant exposures from natural gas cooking burners: A simulation-based assessment for southern California," *Environmental Health Perspectives*, vol. 43, pp. 43-50, 2014.
- 99 L. K. Baxter, J. E. Clougherty, F. Laden and J. I. Levy, "Predictors of concentrations of nitrogen dioxide, fine particulate matter, and particle constituents inside of lower socioeconomic status urban homes," *Journal of Exposure Science & Environmental Epidemiology*, vol. 17, pp. 433-444, 2006.
- 100 US Energy Information Administration, "Residential Energy Consumption Survey (RECS)," May 2018. [Online]. Available: https://www.eia.gov/consumption/residential/data/2015/hc/php/hc1.1.php. [Accessed 11 11 2020].
- 101 J. Engelberg and E. Brassell, "Differences in fuel usage in the United States housing stock: American Housing Survey report," US Department of Commerce, 2019.
- 102 S. C. Doll, E. L. Davison and B. R. Painting, "Weatherization impacts and baseline indoor environmental quality in low income single-family homes," *Building and Environment*, vol. 107, pp. 181-190, 2015.

- 103 Ó. G. Algar, S. Pichini, X. Basagana, C. Puig, O. Vall, M. Torrent and et al., "Concentrations and determinants of NO2in homes of Ashford, UK and Barcelona and Menorca, Spain," *Indoor Air,* vol. 14, pp. 298-304, 2004.
- 104 G. J. Raw, S. K. Coward, M. V. Brown and D. R. Crump, "Exposure to air pollutants in English homes," *Journal of Exposure Science & Environmental Epidemiology*, vol. 14, pp. 85-94, 2004.
- 105 J. I. Levy, K. Lee, J. D. Spengler and Y. Yanagisawa, "Impact of residential nitrogen dioxide exposure on personal exposure: An international study," *Journal of the Air & Waste Management Association,* vol. 48, no. 6, pp. 553-560, 1998.
- 106 N. L. Gilbert, D. Gauvin, M. Guay, M.-E. Heroux, G. Dupuis and et al., "Housing characteristics and indoor concentrations of nitrogen dioxide and formaldehyde in Quebec City, Canada," *Environmental Research*, vol. 102, pp. 1-8, 2006.
- 107 J. Xu, H. Jian, H. Zaho and B. Stephens, "Exposures during scripted daily activities in Chicago, IL," *Aerosol and Air Quality Research*, vol. 17, pp. 1999-2009, 2017.
- 108 P. L. Kinney, S. N. Chillrud, J. Ramstrom and J. D. Spengler, "Exposures to multiple air toxics in New York City," *Environmental Health Perspectives,* vol. 110, no. 4, pp. 539-546, 2020.
- 109 M. H. Garrett, M. A. Hooper, B. M. Hooper, P. R. Rayment and M. J. Abramson, "Increased risk of allergy in children due to formaldehyde exposures in homes," *Allergy*, vol. 54, pp. 330-337, 1999.
- 110 K. C. Coombs, G. L. Chew, C. Schaffer, P. H. Ryan, C. Brokamp, S. A. Grinshpun and et. al,, "Indoor air quality in green-renovated vs. non-green low-income homes of children living in a temperate region of US (Ohio)," *Science of the Total Environment*, Vols. 554-555, pp. 178-185, 2016.
- 111 J. Rovira, N. Roig, M. Nadal, M. Schuhmacher and J. L. Domingo, "Human health risks of formaldehyde indoor levels: An issue of concern," *Journal of Environmental Science and Health, Part A,* vol. 51, no. 4, pp. 357-363, 2015.
- 112 N. L. Gilbert, M. Guay, D. Gauvin, R. N. Dietz, C. C. Chan and B. Lévesque, "Air change rate and concentration of formaldehyde in residential indoor air," Atmospheric Environment, vol. 42, pp. 2424-2428, 2008.

- 113 E. L. Hult, H. Willem, P. N. Price, T. Hotchi, M. L. Russell and B. C. Singer, "Formaldehyde and acetaldehyde exposure mitigation in US residences: In-home measurements of ventilation control and source control," *Indoor Air*, vol. 25, pp. 523-535, 2015.
- 114 A. R. Pickett and M. Bell, "Assessment of indoor air pollution in homes with infants," *International Journal of Environmental Research and Public Health,* vol. 8, pp. 4502-4520, 2011.
- 115 E. D. Patino and J. A. Siegel, "Indoor environmental quality in social housing: A literature review," *Building and Environment,* vol. 131, pp. 231-241, 2018.
- 116 M. D. Colton, P. MacNaughton, J. Vallarino, J. Kane, M. Bennett-Fripp, J. D. Spengler and e. al., "Indoor air quality in green vs conventional multifamily low-income housing," *Environmental Science & Technology*, vol. 48, no. 14, pp. 7833-7841, 2014.
- 117 M. M. Abdel-Salam, "Investigation of PM2.5 and carbon dioxide levels in urban homes," *Journal of the Air & Waste Management Association*, vol. 65, no. 8, pp. 930-936, 2015.
- 118 National Center for Healthy Housing (NCHH), "Studying The Optimal Ventilation for Environmental Indoor Air Quality (The STOVE IAQ project)," Study Protocol Version 1.4, 2019.
- 119 SKC, Inc., "UMEx 100 Passive Sampler for Formaldehyde," [Online]. Available: https://www.skcinc.com/catalog/index.php?cPath=200000000_20300000_2030005 50. [Accessed 11 May 2020].
- 120 C. Kuhlman, N. Zovack and L. Coyne, "Validation of nitrogen dioxide using the SKC UMEx 200 Passive Sampler cat. No. 500-200," SKC, Inc., Eighty Four, 2018.
- 121 SKC, Inc, "UMEx 200 Passive Samplers, for Sulfur Dioxide and Nitrogen Dioxide," 2020. [Online]. Available: https://www.skcltd.com/images/pdfs/3760.pdf. [Accessed 17 December 2017].
- 122 S. K. Hammond and B. P. Leaderer, "A diffusion monitor to measure exposure to passive smoking," *Environmental Science & Technology,* vol. 21, pp. 494-497, 1987.

- 123 Onset, "Telaire 7001 CO2 Sensor: Specifications," [Online]. Available: https://www.onsetcomp.com/products/sensors/tel-7001/. [Accessed 2 October 2020].
- 124 Lascar electronics, "EasyLog: Specifications," [Online]. Available: https://www.lascarelectronics.com/easylog-el-usb-co. [Accessed 2 October 2020].
- 125 S. E. Frey, H. Destaillats, S. Cohn, S. Ahrentzen and M. P. Fraser, "The effects of an energy efficiency retrofit on indoor air quality," *Indoor Air,* vol. 25, no. 2, pp. 210-219, 2015.
- 126 N. R. Martins and G. C. da Graça, "Impact of PM2.5 in indoor urban environments: A review," *Sustainable Cities and Society,* vol. 42, pp. 259-275, 2018.
- 127 S. Yamazaki, H. Nitta, Y. Murakami and S. Fukuhara, "Association between ambient air pollution andhealth-related quality of life in Japan: Ecological study," *International Journal of Environmental Health Researc,* vol. 15, no. 5, pp. 383-391, 2005.
- 128 M. Ścibor, A. Galbarczyk and G. Jasienska, "Living well with pollution? The impact of the concentration of PM2.5 on the quality of life of patients with asthma," *International Journal of Environmental Research and Public Health,* vol. 16, no. 14, p. 2502, 2019.
- 129 G. Karani, M. Bradburn and M. Evans, "SF-36 health survey, indoor environment and housing renovation," *WSEAS Transactions on Environment and Development*, vol. 10, no. 6, pp. 709-720, 2010.
- 130 Environmental Protection Agency (EPA), Indoor air pollution: An introduction for health professionals, 2015.
- 131 International Agency for Research on Cancer (IARC), "Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxypropan-2-ol," Lyon, France, 2006, pp. 37-325.
- 132 C. A. Pope, III, M. Ezzati and D. W. Dockery, "Fine-particulate air pollution and life expectancy in the United States," *New England Journal of Medicine*, vol. 360, pp. 376-386, 2009.
- 133 O. J. Anderson, J. G. Thundiyil and A. Stolbach, "Clearing the air: A review of the effects of particulate matter air pollution on human health," *Journal of Medical Toxicology*, vol. 8, no. 2, pp. 166-175, 2012.

- 134 World Health Organization (WHO), "Reducing Risks, Promoting healthy life," in *The World Health Report*, Geneva, 2002.
- 135 V. C. Pun, J. Manjourides and H. Suh, "Association of ambient air pollution with depressive and anxiety symptoms in older adults: Results from the NSHAP study," *Environmental Health Perspectives*, vol. 125, no. 3, pp. 342-348, 2017.
- 136 A. Acikgoz, B. Baykara, U. M. Camsari, H. Ellidokuz and N. Uysal, "Preliminary investigation of association between indoor air quality and emergence of depressive symptoms," *Sustainable Environment Research,* vol. 23, no. 6, pp. 385-391, 2013.
- 137 O. A. Seppänen, W. J. Fisk and M. J. Mendell, "Association of ventilation rates and CO2 concentrations with health and other responses in commercial and institutional buildings," *Indoor Air*, vol. 9, no. 4, pp. 226-252, 1999.
- 138 C. A. Pope III, J. Schwartz and M. R. Ransom, "Daily mortality and PM 10 pollution in Utah, Salt Lake, and Cache Valleys," *Archives of Environmental Health,* vol. 46, pp. 90-96, 1992.
- 139 K. B. Rumchev, J. T. Spickett, M. K. Bulsara, M. R. Phillips and S. M. Stick, "Domestic exposure to formaldehyde significantly increases the risk of asthma in young children," *European Respiratory Journal*, vol. 20, pp. 403-408, 2002.
- 140 J. H. Olsen and M. Døssing, "Formaldehyde induced symptoms in day care centers," *American Industrial Hygiene Association Journal*, vol. 43, no. 5, pp. 366-370, 1982.
- 141 A. Persily, "Challenges in developing ventilation and indoor air quality standards: The story of ASHRAE Standard 62," *Building and Environment,* vol. 91, pp. 61-69, 2015.
- 142 J. S. Park and K. Ikeda, "Variations of formaldehyde and VOC levels during 3 years in new and older homes," *Indoor Air*, vol. 16, no. 2, pp. 129-135, 2006.
- 143 E. L. Hult, H. Willem, P. N. Price, T. Hotchi, M. L. Russell and B. C. Singer, "Formaldehyde and acetaldehyde exposure mitigation in US residences: in-home measurements of ventilation control and source control," *Indoor Air,* vol. 25, no. 5, pp. 523-535, 2014.

- 144 A. Zota, G. Adamkiewicz, J. I. Levy and J. D. Spengler, "Ventilation in public housing: Implications for indoor nitrogen dioxide concentrations," *Indoor Air,* vol. 15, no. 6, pp. 393-401, 2005.
- 145 P. Lajoie, D. Aubin, V. Gingras, P. Daigneault, F. Ducharme, D. Gauvin, D. Fugler, J.-M. Leclerc, D. Won, M. Courteau, S. Gingras, . M. Héroux, W. Yang and H. Schleibinger, "The IVAIRE project- A randomized controlled study of the impact of ventilation on indoor air quality and the respiratory symptoms of asthmatic children in single family homes," *Indoor Air*, vol. 25, no. 6, pp. 582-597, 2015.
- 146 L. Woodfine, R. D. Neal, N. Bruce, R. T. Edwards, P. Linck, L. Mullock, N. Nelhans, D. Pasterfield, D. Russell and I. Russell, "Enhancing ventilation in homes of children with asthma: Pragmatic randomised controlled trial," *British Journal of General Practice*, vol. 61, no. 592, pp. 724-732, 2011.
- 147 C. G. Bornehag, J. Sundell, L. Hägerhed-Engman and T. Sigsgaard, "Association between ventilation rates in 390 Swedish homes and allergic symptoms in children," *Indoor Air*, vol. 15, no. 4, pp. 275-280, 2005.
- 148 O. Seppänen and W. J. Fisk, "Association of ventilation system type with SBS symptoms in office workers," *Indoor Air,* vol. 12, no. 2, pp. 98-112, 2002.
- 149 Y. Sun, J. Hou, R. Cheng, Y. Sheng, X. Zhang and J. Sundell, "Indoor air quality, ventilation and their associations with sick building syndrome in Chinese homes," *Energy and Buildings*, vol. 197, pp. 112-119, 2019.
- 150 P. W. Francisco, D. E. Jacobs, L. Targos, S. L. Dixon, J. Breysse, W. Rose and S. Cali, "Ventilation, indoor air quality, and health in homes undergoing weatherization," *Indoor Air*, vol. 27, no. 2, pp. 463-477, 2016.
- 151 D.-H. Lee, M. W. Steffes, A. Sjödin, R. S. Jones, L. L. Needham and D. R. Jacobs, Jr, "Low dose of some persistent organic pollutants predicts type 2 diabetes: A nested case-control study," *Environmental Health Perspectives*, vol. 118, no. 9, pp. 1235-1242, 2010.
- 152 G. A. Maglinte, R. D. Hays and R. M. Kaplan, "US general population norms for telephone administration of the SF-36v2," *Journal of Clinical Empidemiology,* vol. 65, no. 5, pp. 497-502, 2012.
- 153 K. D. Truong and M. Sai, "A systematic review of relations between neighborhoods and mental health," *Journal of Mental Health Policy and Economics*, vol. 9, no. 3, pp. 137-154, 2006.

- 154 A. Wandersman and M. Nation, "Urban neighborhoods and mental health: Psychological contributions to understanding toxicity, resilience, and interventions," *American Psychologist*, vol. 53, no. 6, pp. 647-656, 1998.
- 155 G. W. Evans, "The built environment and mental health," *Journal of Urban Health,* vol. 80, pp. 536-555, 2003.
- 156 K. J. Rothman, L. T. Lash and S. Greenland, Modern Epidemiology, Lippincott Williams & Wilkins, 2008.
- 157 J. Breysse, D. E. Jacobs, W. Weber, S. Dixon, C. Kawecki and et al., "Health outcomes and green renovation of affordable housing," *Public Health Reports,* vol. 126, no. Supplement 1, pp. 64-126, 2011.
- 158 J. Fox-Rushby and K. Hanson, "Calculating and presenting disability adjusted life years (DALYs) in cost-effectiveness analysis," *Health Policy and Planning,* vol. 16, no. 3, pp. 326-331, 2001.
- 159 R. Parks, "The rise, critique and persistence of the DALY in global health," *The Journal of Global Health,* vol. 4, no. 1, pp. 28-32, 2014.
- 160 M. Cropper and S. Khanna, "How should the World Bank estimate air pollution damages?," Resources for the Future, Washington, DC, 2014.
- 161 B. Desaigues, D. Ami, A. Bartczak, M. Braun-Kohlová, S. Chilton, M. Czajkowski and et al., "Economic valuation of air pollution mortality: A 9-country contingent valuation survey of value of a life year (VOLY)," *Ecological Indicators,* vol. 11, pp. 902-910, 2011.
- 162 Global Asthma Network, "The Global Asthma Report," Auckland, 2018.
- 163 Centers for Disease Control and Prevention (CDC), "National Current Asthma Prevalence (2017)," 15 May 2018. [Online]. Available: https://www.cdc.gov/asthma/most_recent_national_asthma_data.htm. [Accessed 24 January 2020].
- 164 M. J. Mendell, A. G. Mirer, K. Cheung, M. Tong and J. Douwes, "Respiratory and allergic health effects of dampness, mold, and dampness-related agents: A review of the epidemiologic evidence," *Environmental Health Perspectives,* vol. 119, no. 6, pp. 748-756, 2011.

- 165 M. I. Gunnbjörnsdóttir, K. A. Franklin, D. Norbäck, E. Björnsson, D. Gislason, E. Lindberg and et al., "Prevalence and incidence of respiratory symptoms in relation to indoor dampness: the RHINE study," *Thorax*, vol. 61, no. 3, pp. 221-225, 2006.
- 166 J. D. Thacher, O. Gruzieva, G. Pershagen, E. Melén, J. C. Lorentzen and e. al, "Mold and dampness exposure and allergic outcomes from birth to adolescence: data from the BAMSE cohort," *Allergy*, vol. 72, no. 6, pp. 967-974, 2017.
- 167 J. Krieger, D. E. Jacobs, A. Peter, A. Baeder, G. Chew, D. Dearborn and et al., "Housing interventions and control of asthma-related indoor biologic agents: A review of the evidence," *Journal of Public Health Management and Practice,* vol. 16, no. 5, pp. S11-S20, 2010.
- 168 C. M. Kercsmar, D. G. Dearborn, M. Schluchter, L. Xue, H. L. Kirchner, J. Sobolewski and e. al., "Reduction in asthma morbidity in children as a result of home remediation aimed at moisture sources," *Environmental Health Perspectives*, vol. 114, no. 10, pp. 1574-1580, 2006.
- 169 M. J. Mendell, J. M. Macher and K. Kumagai, "Measured moisture in buildings and adverse health effects: A review," *Indoor Air*, vol. 28, no. 4, pp. 488-499, 2018.
- 170 World Health Organization (WHO), "Environmental burden of disease associated with inadequate housing: A method guide to the quantification of health effects of selected housing risks in the WHO European Region," Copenhagen, Denmark, 2011.
- 171 United States Census Bureau, "American Housing Survey (AHS)," 2017. [Online]. Available: https://www.census.gov/programs-surveys/ahs.html.
- 172 World Health Organization (WHO), "Introduction methods: Assessing the environmental burden of disease at national and local levels," *Environmental Burden of Disease Series*, vol. 1, pp. 573-584, 2003.
- 173 World Health Organization (WHO): Department of Measurement and Health Information, "Disease and Injury Country Estimates," February 2009. [Online]. Available:
 - http://www.who.int/healthinfo/global_burden_disease/estimates_country/en/.

- 174 World Health Organization (WHO): Department of Measurement and Health Information, "Disease and Injury Country Estimates," April 2011. [Online]. Available: http://www.who.int/healthinfo/global_burden_disease/estimates_country/en/.
- 175 Center for Disease Control and Prevention (CDC) WONDER, "Underlying cause of death, 1999-2017," U.S. Department of Health & Human Services, Atlanta, 2017.
- 176 United States Census Bureau, "American Housing Survey (AHS)," 2016. [Online]. Available: https://www.census.gov/programs-surveys/ahs.html.
- 177 World Health Organization (WHO), "Death and DALY estimates for 2004 by cause for WHO Member States: Persons, 3 age groups," http://www.who.int/healthinfo/global_burden_disease/estimates_country/en/., February 2009. [Online]. Available: http://www.who.int/healthinfo/global_burden_disease/estimates_country/en/.. [Accessed 15 July 2016].
- 178 World Health Organization (WHO), "Death estimates for 2008 by cause for WHO Member States: By sex and age: Disease and injury country estimates," Department of Measurement and Health Information, April 2011. [Online]. Available: http://www.who.int/healthinfo/global_burden_disease/estimates_country/en. [Accessed 15 July 2016].
- 179 United States Census Bureau, "American Housing Survey (AHS): Methodology," 30 August 2018. [Online]. Available: https://www.census.gov/programs-surveys/ahs/about/methodology.html. [Accessed 29 March 2019].
- 180 United States Census Bureau, "Annual estimates of housing unis for the United States, regions, divisions, states, and counties: April 1, 2010 to July 1, 2017 population estimates," May 2018. [Online]. Available: https://factfinder.census.gov/faces/tableservices/jsf/pages/productview.xhtml?src=bkmk. [Accessed 29 March 2019].
- 181 World Health Organization (WHO), "Environmental burden of disease associated with inadequate housing: A method guide to the quantification of health effects of selected housing risks in the WHO European Region," Copenhagen, 2011.
- 182 C. Nunes, A. M. Pereira and M. Morais-Almeida, "Asthma costs and social impact," *Asthma Research and Practice*, vol. 3, no. 1, pp. 1-11, 2017.

- 183 S. B. Barnett and T. A. Nurmagambetov, "Costs of asthma in the United States: 2002-2007," *Journal of Allergy and Clinical Immunology,* vol. 27, no. 1, pp. 145-152, 2011.
- 184 T. Nurmagambetov, R. Kuwahara and P. Garbe, "The economic burden of asthma in the United States, 2008-2013," *Annals of the American Thoracic Society,* vol. 15, no. 3, pp. 348-356, 2018.
- 185 D. Mudarri and W. J. Fisk, "Public health and economic impact of dampness and mold," *Indoor Air*, vol. 17, pp. 226-235, 2007.
- 186 UN General Assembly, "Universal Declaration of Human rights," 1948.
- 187 E. Anthes, "More childhood lead poisoning is a side effect of COVID lockdowns," *The New York Times,* 11 March 2021.
- 188 A. C. Mulkern, "California is closing the door to gas in new homes," *Scientific American,* 4 January 2021.
- 189 M. R. Asato, R. Manjunath, D. R. Seth, S. J. Phelps, J. W. Wheless, C. A. Hovinga, J. E. Pina-Garza, S. L. Haskins and W. M. Zingaro, "Adolescent and caregiver experiences with epilepsy," *Journal of Child Neurology,* vol. 24, no. 5, pp. 562-571, 2009.
- 190 T. Espanol, J. Prevot, J. Drabwell, S. Sondhi and L. Olding, "Improving current immunoglobulin therapy for patients with primary immunodeficiency: quality of life and views on treatment," *Patient Preference and Adherence*, vol. 8, pp. 621-629, 2014.
- 191 M. Nakao, K. Yamauchi, Y. Ishihara, H. Omori, D. Ichinnorov and B. Solongo, "Effects of air pollution and seasons on health-related quality of life of Mongolian adults living in Ulaanbaatar: Cross-sectional studies," *BMC Public Health,* vol. 17, no. 594, pp. 1-12, 2017.
- 192 Syracuse Research Corporation, Toxicological Profile for Formaldehyde, Atlanta: Agency for Toxic Substances and Disease Registry, 1999.
- 193 J. N. Sommar, A. Ek, R. Middelveld, A. Bjerg, S.-E. Dahlén, C. Janson and B. Forsberg, "Quality of life in relation to the traffic pollution indicators NO2 and NOx: results from the Swedish GA2LEN survey," *British Medical Journal Open Respiratory Research*, vol. 1, no. 1, 2014.

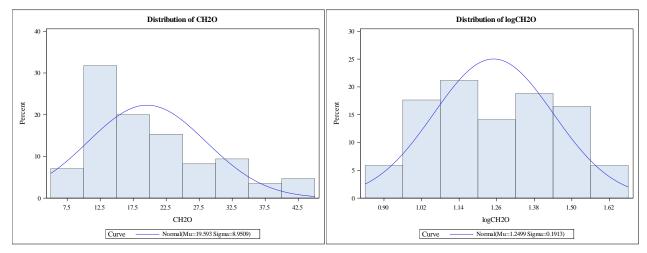
- 194 Optum, "SF-12v2 Health Survey," 2017. [Online]. Available: https://campaign.optum.com/content/optum/en/optum-outcomes/what-we-do/health-surveys/sf-12v2-health-survey.html.
- 195 J. E. Ware Jr., M. Kosinski and S. Keller, "A 12-item short-form health survey: Construction of scales and tests of reliability and validity," *Medical Care*, vol. 34, no. 3, pp. 220-233, 1996.
- 196 Utah Department of Health, "Interpreting the SF-12," Utah Department of Health, 2001.
- 197 World Health Organization (WHO), "The Right to Healthy Indoor Air," Geneva, 2010.
- 198 C. Meisner, D. Gjorgjev and F. Tozija, "Estimating health impacts and economic costs of air pollution in the Republic of Macedonia," *South Eastern European Journal of Public Health,* vol. 4, pp. 22-29, 2015.
- 199 D. Mudarri and W. J. Fisk, "Public health and economic impact of dampness and mold," *Indoor Air,* vol. 17, no. 3, pp. 226-235, 2007.
- 200 U.S. Department of Housing and Urban Development: Office of Healthy Homes and Lead Hazard Control, "American Healthy Homes Survey: Lead and Arsenic Findings," 2011.
- 201 E. Oesterling-Owens, "Endogenous carbon monoxide production in disease," *Clinical Biochemistry*, vol. 43, no. 15, pp. 1183-1188, 2010.
- 202 Environmental Protection Agency (EPA), "Respiratory health effects of passive smoking: Lung cancer and other disorders," Vols. EPA 600-6-90-006F, 1992.
- 203 S. Semple and N. Latif, "How long does secondhand smoke remain in household air: Analysis of PM2.5 data from smokers' homes," *Nicotine and Tobacco Research,* vol. 16, no. 10, pp. 1365-1370, 2014.
- 204 G. Drago, C. Perrino, S. Canepari, S. Ruggieri, L. L'Abbate, V. Longo and &. e. al., "Relationship between domestic smoking and metals and rare earth elements concentration in indoor PM2.5," *Environmental Research*, vol. 165, pp. 71-80, 2018.
- 205 The Carpet and Rug Institute, "Green Label Plus: A Higher Standard for Indoor Air Quality," 2021. [Online]. Available: https://carpet-rug.org/testing/green-label-plus/. [Accessed 22 March 2021].

- 206 N. Suzuki, H. Nakaoka, M. Hanazato, Y. Nakayama, K. Takaya and C. Mori, "Emission rates of substances from low-volatile-organic-compound paints," *International Journal of Environmental Science and Technology*, vol. 16, pp. 4543-4550, 2019.
- 207 J. C. Chang, Z. Guoz, R. Fortmann and H. C. Lao, "Characterization and reduction of formaldehyde emissions from a low-VOC latex paint," *Indoor Air*, vol. 12, no. 1, pp. 10-16, 2002.
- 208 J. C. Chang, R. Fortmann, N. Roache and H.-C. Lao, "Evaluation of low-VOC latex paints," *Indoor Air,* vol. 9, no. 4, pp. 253-258, 2004.
- 209 P. J. Franklin, "Indoor air quality and respiratory health of children," *Pediatric Respiratory Reviews,* vol. 8, no. 4, pp. 281-286, 2007.
- 210 D. A. Otto, H. K. Hudnell, D. E. House, L. Molhave and W. Counts, "Exposure of humans to a volatile organic mixture. I. Behavioral assessment," *Archives of Environmental Health*, vol. 47, no. 1, pp. 23-30, 1992.
- 211 P. Wolkoff and G. D. Nielsen, "Organic compounds in indoor air- Their relevance for perceived indoor air quality?," *Atmospheric Environment,* vol. 35, no. 26, pp. 4407-4417, 2001.
- 212 J. H. Olsen and M. Døssing, "Formaldehyde induced symptoms in day care centers," *American Industrial Hygiene Association Journal*, vol. 43, no. 5, pp. 366-370, 1982.
- 213 L. J. Fewtrell, A. Prüss-Üstünb, P. Landrigan and J. L. Ayusos-Mateos, "Estimating the global burden of disease of mild mental retardation and cardiovascular disease from envriomental lead exposure," *Environmental Research*, vol. 94, no. 2, pp. 120-133, 2004.

APPENDICES

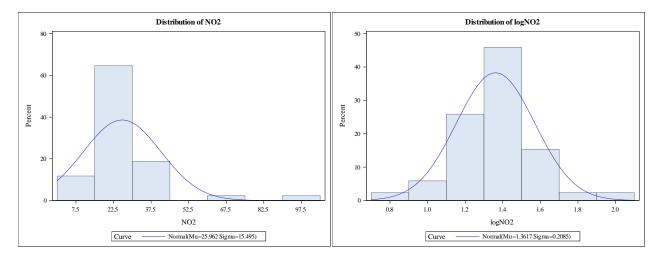
APPENDIX A

Figure 13: Distribution of formaldehyde



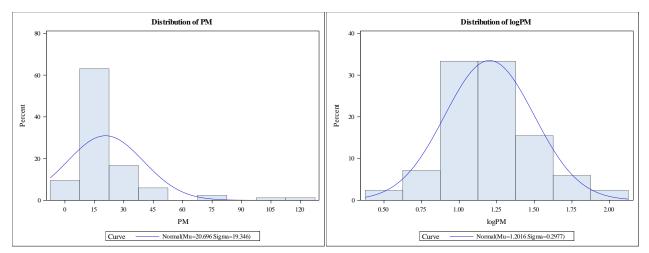
These are the histogram figures showing the distribution of the raw formaldehyde concentrations (left) and the log transformed formaldehyde concentrations (right).

Figure 14: Distribution of nitrogen dioxide



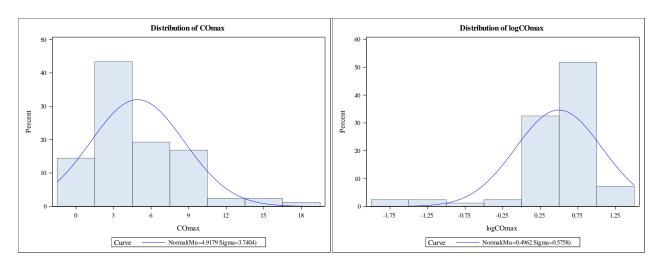
These are the histogram figures showing the distribution of the raw NO₂ concentrations (left) and the log transformed formaldehyde concentrations (right).

Figure 15: Distribution of particulate matter 2.5



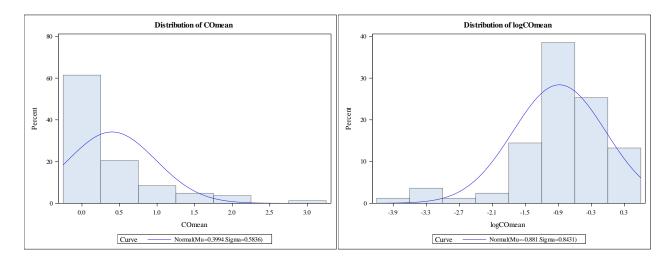
These are the histogram figures showing the distribution of the raw PM_{2.5} concentrations (left) and the log transformed PM_{2.5} concentrations (right).

Figure 16: Distribution of maximum carbon monoxide



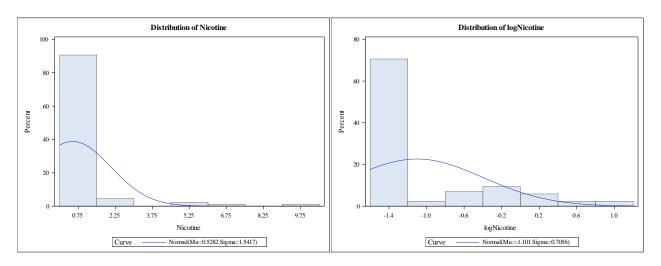
These are the histogram figures showing the distribution of the raw 15-minute maximum CO concentrations (left) and the log transformed 15-minute maximum concentrations (right).

Figure 17: Distribution of mean carbon monoxide



These are the histogram figures showing the distribution of the raw mean CO concentrations (left) and the log transformed mean CO concentrations (right).

Figure 18: Distribution of nicotine



These are the histogram figures showing the distribution of the raw nicotine concentrations (left) and the log transformed nicotine concentrations (right).

<u>APPENDIX B</u>

In order to explore a critical effective cut-off value for percent compliance with ASHRAE 62.2, indoor air contaminant concentrations were plotted against percent compliant results from the blower-door test. The results are shown below in. In general, there were large ranges of concentrations in units that had zero percent compliance with the ASHRAE 62.2 standard. However, similar trends observed in formaldehyde concentrations when plotted against percent compliance are also seen in some of these other air contaminants.

Figure 19: ASHRAE compliance against NO₂

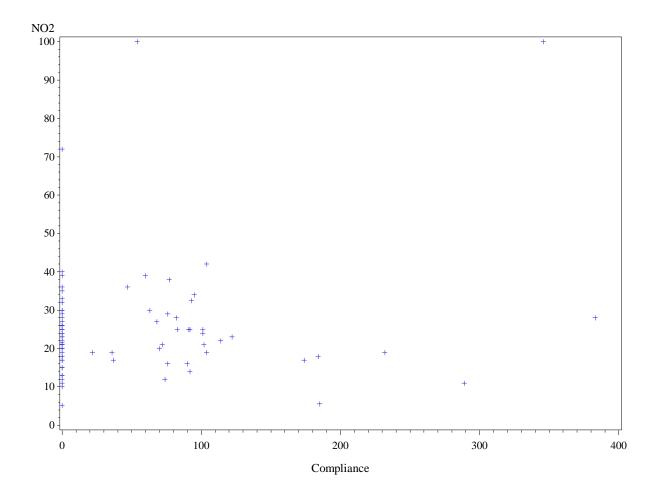


Figure 19 shows the concentration of NO_2 in ppb on the y axis plotted against ASHRAE 62.2 compliance in percent on the x axis.

Figure 20: ASHRAE compliance against PM_{2.5}

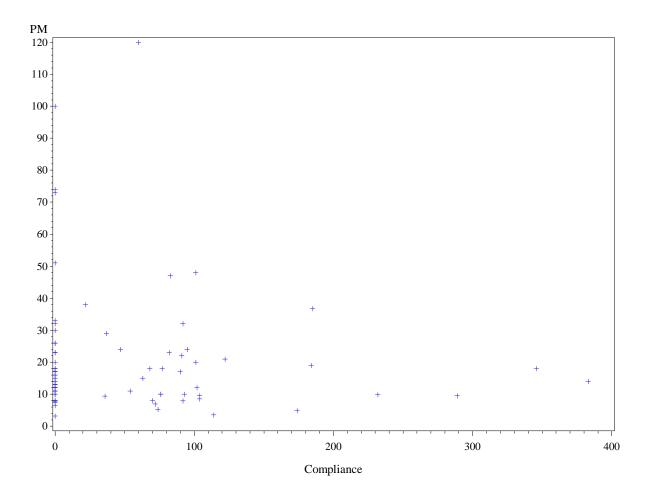


Figure 20 shows the concentration of $PM_{2.5}$ in $\mu g/m^3$ on the y axis plotted against ASHRAE 62.2 compliance in percent on the x axis.

Figure 21: ASHRAE compliance against COmean

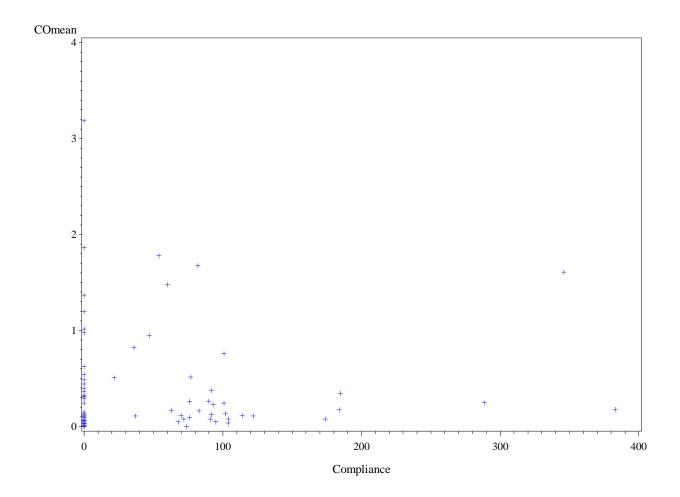


Figure 21 shows the concentration of COmean in ppm on the y axis plotted against ASHRAE 62.2 compliance in percent on the x axis.

Figure 22:ASHRAE compliance against COmax

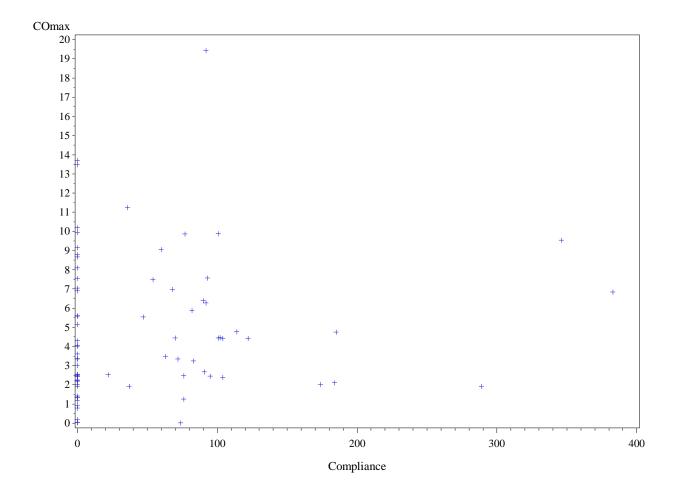


Figure 22 shows the concentration of COmax in ppm on the y axis plotted against ASHRAE 62.2 compliance in percent on the x axis.

Figure 23: ASHRAE compliance against CO₂

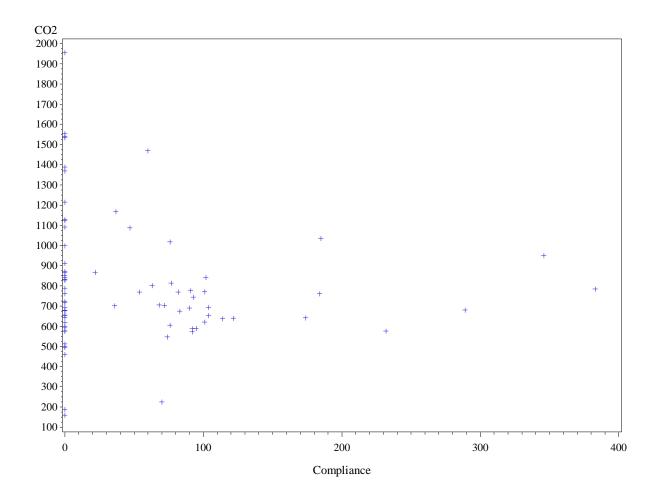


Figure 23 shows the concentration of CO_2 in ppm on the y axis plotted against ASHRAE 62.2 compliance in percent on the x axis.

APPENDIX C

Figure 24: PCS by asthma diagnosis

G

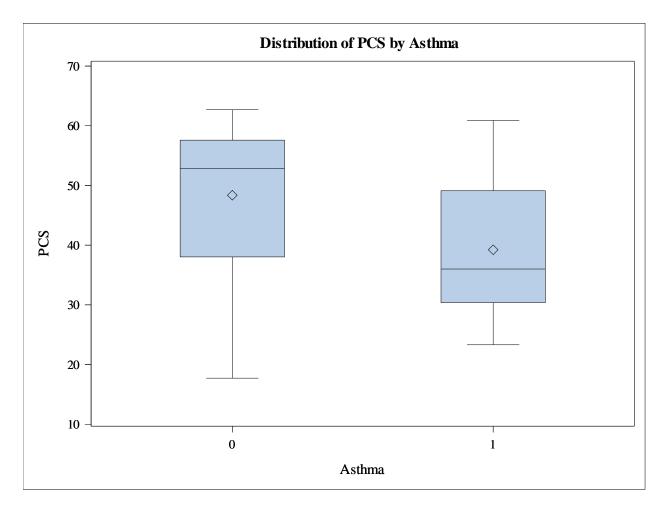


Figure 24 shows there were higher PCS results in participants that reported no asthma disease compared to participants that did report having asthma.

Figure 25: PCS by education level

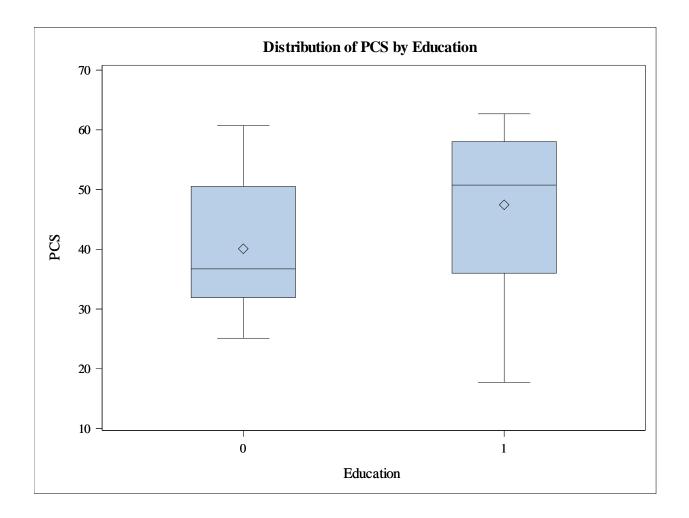


Figure 25 shows there were higher PCS results in participants that reported more than a high school education compared to those that reported a high school education or less.

Figure 26: MCS by detection of nicotine

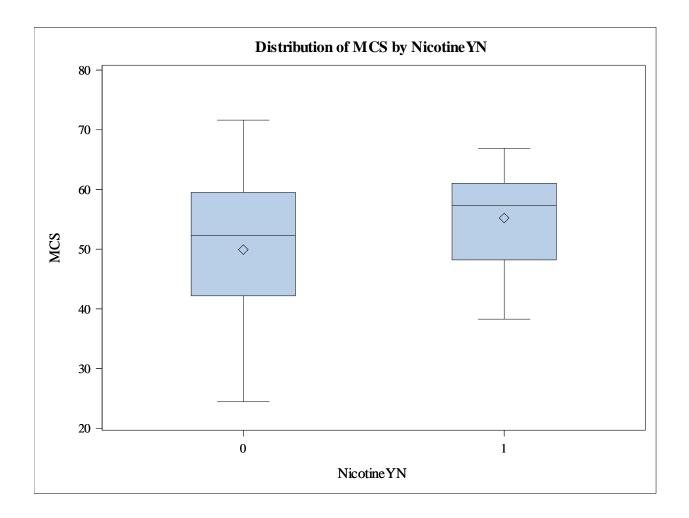


Figure 26 shows there were higher MCS results when nicotine was present in homes compared to units where no nicotine was detected.

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Using the Public Health Toolbox. October 2019

National Environmental Association's Annual Education Conference presentation on a pilot study of pesticides in subsidized low-income housing in Philadelphia and pediatric

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Wisconsin Public Health Association's Annual Conference presentation on local health departments' response to the new ACCLPPs recommendation and unique challenges as well as presenting policies and procedures to address these in May

2014

Wisconsin Environmental Health Association's Annual Education Conference presentation on local health departments' challenges to the CDC's ACCLPPs

recommendation to lower the reference level of blood leads with additional funding and departmental cuts in April 2014

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