Understanding Exposures to Volatile and Semivolatile Organic Compounds in Indoor Environments

By

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ABSTRACT

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Humans spend most of their time indoors, in residences and commercial buildings. In this thesis, I evaluate exposures to volatile (VOCs) and semivolatile organic compounds (SVOCs) in indoor environments. I use a combination of literature review and evaluation, mechanistic modeling, and skin-wipe collection and analysis to develop an understanding of the role of indoor air as an exposure medium for inhalation and passive dermal uptake of pollutants. This dissertation explores three related research topics on indoor environments and human exposures. In Chapter 2, I conduct a comprehensive review of reported measurements of pollutants found in commercial buildings. I used the literature review to estimate concentration ranges that can be compared to health-based exposure limits as basis for hazard assessment. I use the regulatory exposure limits set by government agencies to calculate hazard indices as the ratio of observed concentrations to regulatory standards. I also compare the odor and pungency thresholds of individual pollutants to observed concentrations to evaluate their potential to exceed odor thresholds. The hazard evaluation identifies the potential for health impacts at concentrations commonly found in commercial buildings. This analysis focuses exclusively on VOCs and SVOCs in commercial buildings and identified a limited set of pollutants that pose health concerns. I also characterize the selected pollutants in terms of the chemical properties that,affect partitioning to various indoor surfaces, and subsequently their fate and transport in indoor environments. Based on chemical properties and indoor fate, I grouped the pollutants into five groups. I use an hierarchical k-means analysis based on octanol-air partitioning coefficient, octanol-water partitioning coefficient, air-water partitioning coefficient, and molecular weight. The pollutants in each group are expected to behave similarly in indoor environments.

In Chapter 3, I evaluate the role of buildings operation parameters such as ventilation and filtration in limiting exposures to pollutants originating from indoor and outdoor sources. I use a simple well-mixed-air model of an indoor space to study the impact of ventilation on concentrations of ozone, nitrogen dioxide, carbon monoxide, and radon. I employ a chemical-thermodynamics-(fugacity)-based mass balance model in conjunction with a particle mass balance to study the fate and transport of particulate matter, VOCs, and SVOCs. The fugacity mass balance model accounts for chemical partitioning among air, air-borne particles, and indoor surfaces. I ran the fugacity model with indoor and outdoor source of VOCs and SVOCs and

indoor and outdoor sources of particulate matter. I evaluate the consequent inhalation exposures these sources with two outcome metrics, intake fraction (iF) for indoor sources and indoor/outdoor concentration ratio for outdoor sources. The exposure to particulate matter of indoor and outdoor origin was evaluated using the outcome metrics iF and the indoor proportion of outdoor particles (iPOP). The model evaluation shows that ventilation is most effective at controlling exposures to VOCs that have an indoor source. Filtration is seen to be effective at controlling exposures to particulate matter and SVOCs that partition preferentially onto particulate matter.

In Chapter 4, I explore the role of indoor air in delivering SVOCs to human occupants through passive dermal uptake. I collected wipe samples from thirteen subjects who were randomly chosen. For each subject, I collected three sequential wipe samples from the forehead and one sample from the palm. I analyzed the samples for a suite of SVOCs and skin lipids (squalene and sapienic acid) in an analytical laboratory using gas chromatography and liquid chromatography. All forehead wipe samples contained SVOCs indicating that air to skin transfer of pollutants for passive dermal uptake could be a significant exposure pathway for SVOCs. Because skin lipid concentrations decrease with depth the quantitation of skin lipid concentrations from each wipe allowed me to estimate the depth of sampling by each skin wipe. This is the first study to quantitatively evaluate the depth of sampling by skin wipes. I use the experimental results together with a theoretical model to explore the potential role of skin as a passive sampler for short-term personal exposures, indoors. For this I develop a metric called the equivalent time of exposure (ETE) to study the usefulness of sequential skin wipe samples as a passive sampler. I used partitioning coefficients from air to skin surface, combined with a dynamic skin mass transport model, to study the theoretical transport of pollutant through the stratum corneum. I compare the modeled concentrations to measured concentrations, at comparable depths. The ETE is the amount of time to which the subject would have to be exposed to a constant air concentration to attain the observed skin-wipe concentration depth profile in the stratum corneum. Based on the ETE, I find that skin wipe samples could be indicative of exposures up to 6 hours prior to wipe sampling, depending on the diffusion coefficient of the pollutant.

The overarching goal of this research is to evaluate the role of indoor air in mediating the transfer to human receptors of pollutants released indoors or brought indoors from outdoor sources. The indoor air mass controls the fate and transport of pollutants in indoor spaces, and the rate of delivery of pollutants for inhalation and dermal uptake. The research highlights the important role of air-to-surface and air-to-particle partitioning in facilitating or mitigating source-receptor relationships. The work illustrates future research opportunities for tracking the complex web of indoor/outdoor pathways that bring pollutants into the human environment and into the blood and other viable tissues of the human population.

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CHAPTER 1. Introduction

This dissertation explores the contribution of different exposure pathways in indoor environments for a selected set of chemical substances. This chapter provides an introduction to the field of exposure science, a discussion of indoor environments of importance in this work, and reviews the different exposure pathways. In Chapter 2, I conduct a comprehensive review of environmental stressors in commercial buildings. In Chapter 3, I model the fate and transport of stressors in commercial buildings. In Chapter 4, I evaluate the role of air as a media in delivery of chemicals for dermal uptake, by combining analytical measurements with mechanistic modeling. The research addresses important data gaps in current literature on exposures in indoor environments.

Exposure science has been described as one of pillars of public health, a critical tool to understand and mitigate health risks associated with stressors As highlighted in the document "Exposure Science in the 21st Century: A Vision and Strategy" (2012), the four major demands that drive the need for more data in exposure science are: societal demands, market demands, policy and regulatory demands and health and environmental science demands. This need for data and knowledge is addressed through combination of analytical measurements and modeling. This National Academies report also highlights the need for evolving our techniques to assess exposure, particularly in the context of introducing new chemicals into global commerce. This introduction discusses the core elements of exposure science (Figure 1-1), in the context of indoor environments: stressors, time and activity behavior, and contact between stressors and receptors.

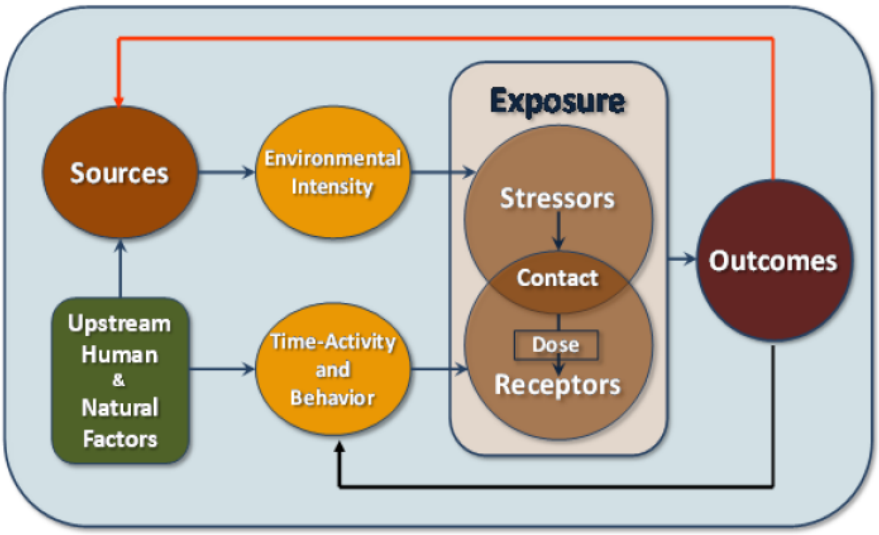

Figure 1-1: Core elements of exposure assessment (Source: Figure S1 from "Exposure Science in the 21st century: A Vision and Strategy", 2012, reproduced with permission from National Academies Press)

Indoor environments and sources

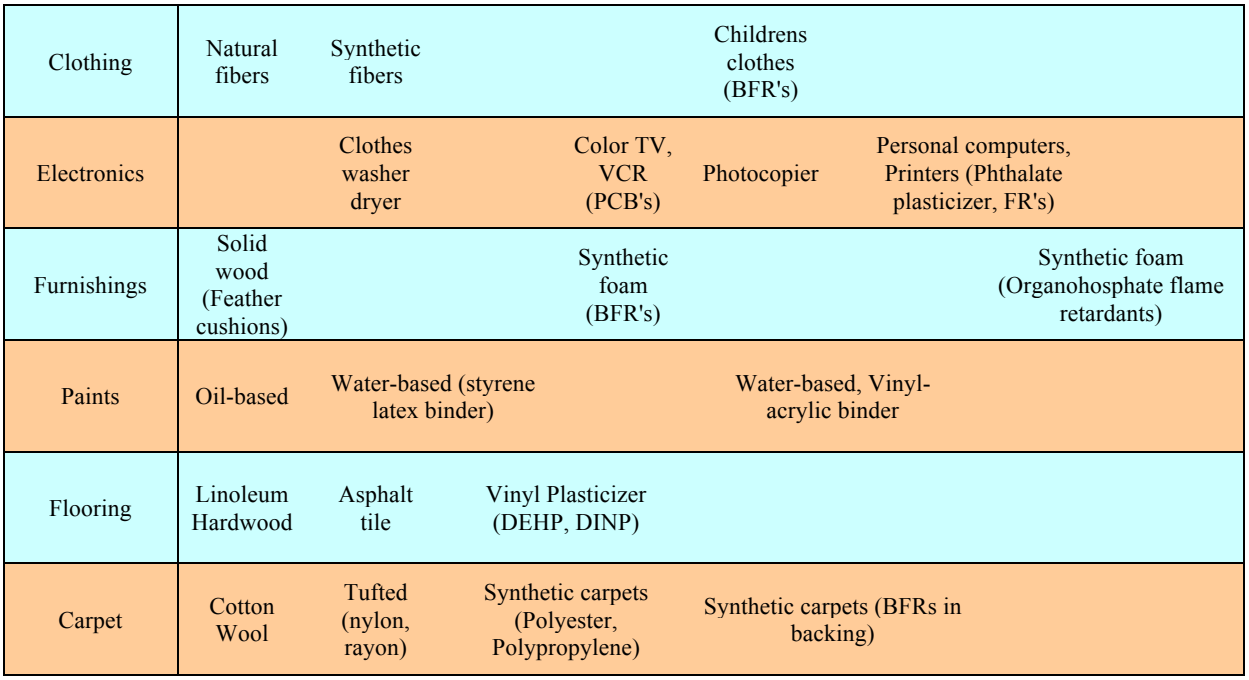

Figure 1-2: Evolution of materials in indoor environments through the years (adapted from Weschler 2009)

Indoor environments and the products used in them have constantly evolved over the past 50-60 years (Figure 1-2). These changes have a brought a paradigm shift in the nature of pollutants that are present in indoor spaces. This elicits a demand from the exposure science community to address the challenges of measuring exposures to newer pollutants. Humans spend up to 90% of their time in indoor environments such as residences (~70%), and commercial buildings (~10-15%) (Klepeis et al. 2001). Chemicals that humans contact indoors, some of which have potential for harmful effects, come from a variety of different sources. Based on the source location, they can be classified into indoor-source pollutants and pollutants originating from outdoor air but coming indoors. The term "pollutants from outdoor air" is used to describe all pollutants that come into indoor spaces from outdoor air. Sources in the outdoor environment are comprised of anthropogenic sources and biogenic sources. In urban environments anthropogenic sources come from a range of industrial and energy-generating activities and are typically dominated by combustion emissions—motor vehicular emissions in particular. Biogenic sources include plants and forests (volatile organic compounds, VOCs). Indoor sources include flooring, walls, countertops, furniture surfaces, upholstery, electronic equipment, and personal care products (Xu et al. 2011; Rudel et al. 2010; Wensing et al. 2005). Human populations are exposed to a variety of pollutants from these sources: criteria air pollutants, volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs) and biological pollutants.

The National Health and Nutrition Examination Surveys exposure reports reveal increasing levels of SVOCs in blood and urine samples of the United States population (CDC 2005-2006; CDC 2007-2008; CDC 2009-2010; CDC 2011-2012; CDC 2013-2014). As defined by the United States Environmental Protection Agency (USEPA) chemicals that are considered 'High

Production Volume' are produced or imported into the United States in excess of 1 million pounds/year. Over 1000 SVOCs are found on the high-production-volume (HPV) chemicals list (USEPA 2007; Weschler et al. 2008). Many of these reported SVOCs are used in products that find application in indoor environments, thus increasing the potential for human exposure. Apart from some classes of well-studied SVOCs such as flame-retardants and phthalates, limited toxicology and exposure data are available on many of these compounds. Halogenated flame retardants are linked to a variety of health effects such as lowering of intellectual quotient (IQ) points in children and endocrine disruption (Birnbaum et al. 2004; Hites et al. 2004; Main et al. 2007). Phthalates are linked to congenital reproductive system development issues in male children and increased occurrences of allergies (Bornehag et al. 2004; Hauser et al. 2005). Current research on the fate and transport of SVOCs in the indoor environment shows that they are present on various indoor surfaces as well as on particulate matter and have long residence times (Shin et al. 2014; Weschler et al. 2010; Zhang et al. 2009; Little et al. 2013). However, very limited studies exist on understanding exposure to SVOCs in indoor environments, both residential and commercial. Indoor environments can deliver SVOCs to human occupants at a continuous rate for years.

Interactions between humans and their indoor environments

The current literature on commercial buildings in the United States can be broadly classified into three categories that evaluate: 1) the effect of ventilation and filtration on pollutant concentration in indoor spaces, 2) the effect of ventilation on health 3) the effect of concentration on health. Very few studies examine the effect of building parameters on concentrations and subsequent health effects (Sundell et al. 1993; Jaakola et al. 1991; Bluyssen et al. 1996; Wargocki et al. 1999; Wargocki et al. 2000; Seppanen and Fisk 2006; Seppanen et al. 2006; Wargocki et al. 2007; Chao et al. 2001; Fisk et al. 2009). In addition, the study of how building parameters affect concentration and exposure in commercial spaces can be used to identify pollutants of concern for setting minimum ventilation rate standards in buildings. Understanding the relationship between exposures and ventilation in indoor environments is important for conducting any comprehensive health assessment for indoor environments.

In residential environments, prior work has included efforts to understand what drives the health risks associated with inhalation of pollutants indoors (Logue et al. 2012; Guo et al. 2004; Hoddinott et al. 2000). The results from a recent risk assessment by Logue et al. (2012) show that health risks are dominated by particulate matter (PM), formaldehyde and acrolein in residential environments. The current literature on indoor environments can be broadly summarized into a number of themes. Numerous studies have highlighted the importance of reducing indoor pollutant loads to improve occupant perceptions of indoor air quality and office worker productivity (Bluyssen et al. 1996; Wargocki et al. 1999; Wargocki et al. 2000). Increased ventilation (increased outdoor air supply) is a means of reducing concentrations of pollutants emitted indoors and studies have reported significant improvement in measures of work and school performance when ventilation rates are increased (Wargocki et al. 2007; Seppanen and Fisk 2006; Seppanen et al. 2006). Satisfaction with air quality has improved and sick building syndrome (SBS) symptoms have decreased with increased ventilation rates (Fisk et al. 2006; Chao et al. 2001; Fisk et al. 2009; Sundell et al. 1993), although not in every study. This theme was further supported by a study, which showed that doubling ventilation rates reduced absence among office workers (Milton et al. 2000). The modeled economic benefits of

improvements in acute health effects and work performance, resulting from increased ventilation rates, are large (Fisk et al. 2009). However, these studies have not directly related work performance, satisfaction with indoor air quality, or SBS symptoms with any specific indoor pollutant concentrations and have not considered potential implications on chronic health effects. Chan et al. (2015) evaluated how filtration and ventilation can change the calculated risks from inhalation exposure to pollutants in commercial buildings such as offices, schools, grocery and retail stores. Particulate matter and formaldehyde largely drive the health risks in these buildings; with high efficiency PM filters contributing to significant lowering of risks.

In indoor environments human populations are exposed to pollutants via the three exposure pathways: inhalation, dermal and ingestion (Exposure Science in the 21st Century: A Vision and Strategy, 2012). The three pathways can be further described in detail as follows. Inhalation exposure occurs through intake of bulk air, which consists of pollutants in the gas and particle phase. This distinction is especially important for compounds such as SVOCs, which partition onto organic (particle) phases (Weschler and Nazaroff 2008; Weschler and Nazaroff 2010). The ingestion pathway can be categorized as direct ingestion and indirect ingestion. Dermal uptake of chemicals occurs through active contact with surfaces or through passive uptake from air. The overarching goal of this research is to explore the role of air as a delivery medium of chemicals, which includes the inhalation of air and passive dermal uptake of chemicals from air. The ubiquitous presence of a wide suite of SVOCs in indoor environments, highlights the need for passive samplers of exposure. I highlight the use of sequential skin wipes as a passive sample of recent exposures. While most dermal exposure studies focus on contact driven uptake of chemicals, recent work has highlighted that direct-air-skin transfer of chemical can significantly contribute to overall exposure. Prior research on air-to-skin transfer of chemicals has focused on VOCs, however there is recent interest in air-to-skin movement of SVOCs (Weschler et al. 2016). To develop understanding of this topic I collect opportunity samples to study the concentrations of SVOCs in skin surface wipes.

As indoor spaces and their complex chemistry affects humans, the humans in turn also have the ability to alter the indoor space to enhance or diminish exposures. Some examples include skin and clothing reacting with indoor environment and human bio-effluent emissions into indoor spaces through breathing. An example I explore in my research, albeit briefly, is the reaction of skin-surface lipids with ozone. The skin is a complex membrane, the uppermost layer of the skin is covered with skin-surface lipids (Greene et al. 1970; Downing et al. 1974). The most dominant skin lipid by mass concentration is squalene (Greene et al. 1970). Many SVOCs partition preferentially onto the skin lipids which are on the surface of the skin membrane. Wisthaler and Weschler (2010) showed that ozone can react with squalene in skin lipids to produce a variety of oxidation products.

Overview of the dissertation

In this research, I focus on addressing data gaps in exposure science within indoor environments. This work is organized into three projects. The first project, which is reported in Chapter 2 of this dissertation, focuses on understanding the various pollutants in commercial buildings and evaluating them from a health-impact perspective. I summarize the current literature on VOCs and SVOCs in commercial buildings in the United States. SVOCs have low vapor pressure (10^{-9} to 10 Pa at 25°C), are largely lipophilic, and have high octanol-air partitioning coefficients (Weschler and Nazaroff 2008; Weschler and Nazaroff 2010). They are largely in the particle

phase in air and on indoor surfaces. Given the physicochemical properties of these compounds, dermal and ingestion exposures are expected to dominate contributions to total indoor intake for these compounds. Some examples of SVOCs include flame retardant compounds, plasticizers and pesticides (Wensing et al. 2005; Destaillats et al. 2008; Stapleton et al. 2011). Flow of consumables out of the buildings, such as clothes, trash, and other products will also alter the SVOC load indoors. Additionally, humans may serve as SVOC source/sinks and contribute to redistribution of SVOCs from their actual source. We expect that human activities such as periodic cleaning could alter the flux of chemical from the room air into surfaces such as carpet, wall, and vinyl. VOCs are volatile, have high vapor pressure (>10 Pa at 25°C) and exist largely in the gas phase in air. In contrast to SVOCs, inhalation, and potentially dermal exposure are expected to be the dominant exposure pathways.

Once measured concentrations of indoor pollutants reported in the current literature are compared to health and odor based thresholds. I also evaluate the chemical properties of pollutants, which can determine their fate and transport in indoor environments. The properties evaluated are the octanol-water partitioning coefficient, octanol-air partitioning coefficient, and air-water partitioning coefficient. The results of this work are provided to inform risk assessments used to assess impacts of pollutants in indoor commercial spaces, and to study the effect of building parameters such as ventilation and filtration on inhalation exposures.

The second project of my dissertation explores how variations of ventilation rates and filtration efficiency impact inhalation exposures to VOCs and SVOCs in indoor environments. I use a fugacity based dynamic mass-balance model to track the fate of SVOCs in the indoor environment (Bennett and Furtaw, 2004). I use this model to understand the effect of building related parameters such as ventilation/filtration and human activities (cleaning) on VOC and SVOC concentration. The work has important implications for energy use in ventilating commercial buildings. In addition, the work can also be used to inform decisions on setting minimum ventilation rates standards in buildings.

The third project of my dissertation focuses on improving our understanding of activity-based vs. passive dermal uptake of chemicals indoors. The skin is a complex membrane and there has been recent interest on understanding how different chemical classes become available for intake through the dermal pathway through either surface contact (active) and passive transport mechanisms (Weschler and Nazaroff 2012; Weschler et al. 2015; Morrison et al. 2016). The passive transport of SVOCs is driven primarily by their chemical properties, such as the diffusion coefficient in air, the diffusion coefficient in skin, and the air-lipid partitioning coefficient. The use of dust samples as a passive measure of pollutant available for uptake in indoor environments is common. I explore the idea of using skin as a passive measure of pollutant made available for intake via both, inhalation and dermal uptake. The amount of pollutant on skin can vary widely depending on sampling location. In areas such as hands and palms the pollutant is largely transferred to the skin by contact with surfaces containing chemical. However, in areas such as the forehead the chemical is transferred via air-to-skin passive pathway. Sampling from both locations provides an interesting study in the contrasts between the concentrations. To compliment the experimental work, I model the transfer of select SVOCs, di-methyl phthalate, di-ethyl phthalate and di-n-butyl phthalate through the skin. Given the various health effects associated with phthalates and their ubiquity in indoor environments makes them an interesting class of compounds to study (Bornehaag et al. 2004; Hauser and

Calafat 2005). In addition, I also study the concentration of lipids removed from the skin with each wipe sample and the ozone-squalene reaction products in skin lipids.

My research focuses on addressing the data gaps in current literature while enhancing our understanding of exposures to pollutants in indoor environments. I confront here a series of research topics on indoor exposure in the context of understanding indoor exposures to VOCs and SVOCs. I use a combination of modeling and measurements to improve our knowledge of indoor exposures. I also explore the novel idea of using skin as a passive sampler for personal exposures. In addition, I also address some of the unique challenges in exposure science that is associated with the complexity of human interactions with indoor environments. My analysis helps understand the role of air as an exposure media, which drives two exposure pathways in indoor environments (inhalation, and passive dermal). My work will help improve the understanding of the role of skin uptake relative to other pathways. In addition, this effort will help understand how consistent exposure to stressors in indoor environments can contribute to overall exposure. By increasing the knowledge of exposure pathways, focusing on the media, the findings from this study will better inform policies targeted toward minimizing indoor exposures that adversely affect human health. The research can be used to inform risk assessment studies to evaluate health burdens associated with indoor exposures in commercial buildings. This research provides insight on how to design epidemiological studies to study health effects, limit and manage exposures, thereby reducing the health burden due to indoor air pollution.

CHAPTER 2. Pollutants in commercial buildings: Overview and hazard evaluation

ABSTRACT

This chapter evaluates potential exposures and health effects of the pollutants commonly found in commercial buildings. Many pollutants found in commercial buildings can be characterized as criteria air pollutants, volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), and biological contaminants. We start with a set of contaminants measured in commercial buildings in the United States and group the pollutants according to hazard potential. This chapter focuses on volatile organic compounds and semivolatile organic compounds with results that inform our work in chapters 2 and 3 of this dissertation. We group the pollutants independently based on two methods. The first method uses the individual chemical properties (octanol-air partitioning coefficient, air-water partitioning coefficient, octanol-water partitioning coefficient and molecular weight). In order to group chemicals in terms of their capacity or recalcitrance for being removed by ventilation, we conduct a k-means analysis based on chemical properties that determine the transport behavior of chemicals in the indoor environment. The chemicals were sorted into 5 groups. Groups 4 and 5 consist of more volatile compounds, Group 2 and 3 are comprised of compounds of lower volatility then VOCs, and Group 1 consists of semivolatile compounds. The second method compares reported concentrations of pollutants to guidelines for prevention of acute and chronic health effects. The review was restricted to studies that were conducted in buildings in the United States. We also compared reported concentrations to odor thresholds and thresholds for sensory (irritation) effects. The compounds were sorted based on measured concentrations that were close to health guidelines. Sorting the chemicals independently based on these two methods allows us to simplify the process for assessing health risks associated with exposures, while also exploring the role of building characteristics such as filtration and ventilation on the transport of pollutants in indoor environments.

INTRODUCTION

This chapter evaluates potential exposures and health effects of the pollutants commonly found in commercial buildings. Many pollutants have been identified and measured in commercial buildings and include criteria air pollutants, volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), and biological contaminants. The goal of the chapter is to provide, demonstrate, and compare two approaches for screening and ranking pollutants—one based on chemical properties that, together with commercial building characteristics, determine exposure potential and the second based on hazard ranking with reported measurements.

The focus of this work on commercial buildings requires a definition of commercial buildings and identification of the types of pollutants found in these buildings. American Society of Heating, Refrigerating and Air-Conditioning Engineers has published a ventilation standards document 62.1-2010 (ASHRAE 2010), which classifies commercial buildings into the following types: correctional facilities; educational facilities; food and beverage services; hotels, motels resorts, and dormitories; office buildings; miscellaneous spaces; public assembly spaces; retail stores; and sports and entertainment. Given the range of commercial buildings under consideration, we expect contaminant sources and their concentrations to vary depending on the activity and use of the buildings. Within the last two decades, it has been shown that ventilation

rates in commercial buildings affect the prevalence and severity of a variety of acute health symptoms, often called sick-building syndrome symptoms, associated with occupancy in a building (Seppanen et al. 1999; Wargocki et al. 1999; Fisk et al. 2009). Other studies have shown that ventilation rates can affect the speed or accuracy of office work or school work (Seppanen and Fisk 2006; Wargocki et al. 2007). The pollutants that underlie the impacts of ventilation rates on symptoms and work performance have remained largely unknown. It is recognized that ventilation rates affect exposures to air pollutants that pose chronic health risks such as cancer, cardiovascular disease, and reproductive effects, but for commercial buildings we have identified no analyses of how risks of these chronic health effects vary with ventilation rates. Very few studies focus on all three key issues in buildings—ventilation rate, its effect on concentration of pollutants indoors, and subsequent health effects (Menzies et al. 1996; Sundell et al. 1993). In this Chapter we examine the common volatile and semivolatile organic compounds in commercial buildings and use the results of this chapter to inform our work in Chapters 2 and 3 where we examine the effect of ventilation and filtration on occupant health and pollutant concentrations.

Pollutants of concern in commercial buildings include volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), biological contaminants, particulate matter, and numerous inorganic gases (CO_2, CO, nitrogen oxides, sulfur oxides, O_3). Some pollutants such as the criteria air pollutants, inorganic gases, VOCs have well-established health impacts, however, in this screening we focus on VOCs and SVOCs. VOCs, many with adverse effects on occupants, are emitted by building materials, contents, and occupants, and concentrations can be reduced by ventilation as well as by source control and various air cleaning strategies (Siegel 2015). Particulate matter has indoor sources and is also brought into buildings with outdoor air. However, filtration is expected to play a major role in controlling the concentrations compared to ventilation. At this time, very little is known about SVOCs and biological contaminants.

We group the chemicals of concern based on two criteria: chemical properties and health- and odor-based thresholds. The chemical properties of the compounds were obtained from a standard database, and subjected to a k-means analysis based on their chemical properties. It is important to distinguish chemicals based on how well they are removed from a commercial building or school by ventilation. With a primary focus on volatile and semivolatile organic compounds we developed an approach to determine how building characteristics (ventilation, filtration, cleaning) impact pollutant concentration. Chemical properties are an important factor in this process. Chemicals with a high chemical affinity for carpets, walls, and furniture will not be effectively removed from the indoor environment by ventilation. We used the following chemical properties: octanol-air partitioning coefficient (K_{oa}), octanol-water partitioning coefficient (K_{ow}), and air-water partitioning coefficient (K_{aw}). The partitioning coefficients provide a measure of solubility in the different phases (air, water, octanol – which is a reasonable substitute for the organic phase). The solubility of chemicals determines the rate of transfer to and from surfaces, and quantity of chemical present the different phases in the indoor environment such as air, particulate matter, indoor surfaces, etc.

The health- and odor-based grouping is done to identify VOCs and SVOCs that are of potential interest for conducting a health risk assessment for exposures to VOCs and SVOCs indoors. This analysis considers VOCs and SVOCs concentrations and compares them to health guidelines and thresholds. For pollutants with concentrations approaching the guidelines and thresholds,

literature was reviewed to determine the primary location of the sources (indoors or outdoor air) and to determine what is known about the impact of ventilation rates on indoor concentrations. The second grouping of chemicals is conducted independently based only on chemical properties.

LITERATURE REVIEW

As a first step in identifying the contaminants of concern, we carried out and summarize here a literature review. We searched for studies that report concentrations of VOCs in commercial buildings in the US (restricted to studies published after the 1990's). Details of this review are presented in Table 2-2. The studies included were carried out in a variety of commercial buildings: office buildings (Apte and Erdmann 2002; Daisey et al. 1994; Bennett et al. 2011; Alevantis et al. 2006; Shields et al. 1996; Fisk et al. 2016), retail buildings (Loh et al. 2006; Hotchi et al. 2006; Eklund et al. 2008; Siegel et al. 2013) and schools (California Schools 2003; Hodgson et al. 2004; Shendell et al. 2004; Godwin et al. 2006;). Most of the reviewed studies were cross-sectional. Eklund et al. (2008) and Alevantis et al. (2006) are two studies that followed buildings over time to assess changes in VOC concentrations. We also included some SVOCs that have been measured in residences but have not been sampled for in commercial buildings, since they are an emerging class of compounds of interest. Sampling times varied among studies, however, the studies mostly employed active sampling methods to measure VOC concentrations. In the following paragraphs, we provide short summaries of the studies that provided key references used to select the contaminants of concern in indoor air.

Daisey et al. (1994) measured concentrations of 39 VOCs in 12 office buildings in California along with outdoor concentrations adjacent to the buildings. The sampling included thre naturally-ventilated, three mechanically-ventilated, and six air-conditioned buildings. Daisey et al. (1994), reported that total VOC concentrations in the buildings were low, but noted that some buildings with photocopiers had higher levels of C10-C11 isoparaffinic compounds. They found no significant variation in total VOC concentration associated with the types of ventilation systems used in the buildings. They found oxidized hydrocarbons such as ethanol and chlorinated hydrocarbons to be the most abundant VOCs. An analysis of indoor and outdoor concentrations, helped associate compounds such as ethanol, isopropanol, acetone, n-dodecane, n-pentanal, n-hexanal, limonene, dichloromethane, trichloroethene, trichloroethane predominantly with indoor sources. The indoor to outdoor concentration ratio of these compounds was greater than 1.35. Other compounds such as benzene, xylenes, ethyltoluenes, trimethylbenzenes, pentane, 3-methylhexane, tetrachloroethylene, benzaldehyde, 1-phenylethanone, and n-decane were associated with outdoor sources. The ratio of indoor to outdoor concentrations of these compounds was lower than 1.35.

Shields et al. (1996) measured VOC concentrations in 50 sparsely occupied telecommunications offices, nine variably occupied data centers and 11 densely occupied administrative offices. The study was carried out for 6 weeks during March and April 1991. Their use of passive samplers limited the VOCs that could be detected in the study. They found total VOC concentrations to be consistently higher indoors compared to outdoors. Telecommunications offices had the lowest indoor/outdoor concentration ratio (3.2), followed by administrative office (5.3) and data centers (8.6). Administrative offices were better ventilated than data centers—thus indicating an association between ventilation rates and indoor concentrations. Compounds such as D4 siloxane, D5 siloxane, alkanes (n-C12 to n-C16), limonene and tetrachloroethylene varied across

the building types and were strongly associated with occupant density. Concentrations of compounds such as toluene, xylenes, n-decane, n-undecane, and Texanol were fairly uniform across all buildings types.

The California Portable Classrooms study (California Schools Study) was carried out by the California Air Resources Board and the Department of Health Services between April 2001 and February 2002 (CARB 2004). The study was primarily carried out to assess conditions in California public school classrooms in order to develop and support various recommendations for improving indoor environmental conditions. The study had two phases, the first phase included a mail survey sent to 1,000 schools and the mailing of passive formaldehyde samplers to two-thirds of the schools. The second phase included site-specific samples (for aldehydes, VOCs, mold spores, pollen, biological pollutants, particle counts, pesticides, metals, PAH's, and allergens in floor dust) collected in 201 classrooms at 67 randomly selected schools in California. The second phase also involved monitoring environmental factors such as temperature, humidity, noise, ventilation and lighting. In both phases, two portable and one traditional classroom in each school were selected for the study. The passive formaldehyde sampling was carried out for 7-10 days in Phase I, and in Phase II 6-h sampling was carried out. Most of the schools were suburban. Elementary schools were sampled more than middle or high schools. The survey showed that portable classrooms had greater number of complaints about issues such as water leaks, noise, mold, odor, indoor air quality, lighting and insects. Even though ventilation rates in both types of classrooms were not significantly different (5% confidence level), the filters of HVAC units in portable classrooms were associated more strongly with presence of mold/mildew, clogging, dirty drain pans and standing water. The CO_2 levels in classrooms were also found to be significantly higher than outdoor levels.

Apte and Erdmann (2002), analyzed data from the United States Environmental Protection Agency (USEPA) Building Assessment Survey and Evaluation (BASE) study. The BASE study was carried out in 100 US office buildings that were randomly selected by the USEPA. The study included integrated 9-hour VOC samples collected in each building and representing a work day. The study reports summary statistics (mean, median, minimum, maximum, standard deviation) for 37 VOCs for which the complete dataset is also available. For the VOCs that were measured, the BASE study also identified potential sources. The median formaldehyde concentration reported across all buildings (12 ppb), exceeded the Agency for Toxic Substances and Disease Registry (ATSDR) chronic and intermediate maximum recommended limits (MRLs) of 8 ppb. The maximum benzene concentration (10 ppb) was found to exceed the ASTDR intermediate MRL of 9 ppb.

Hodgson and Levin (2003) reviewed the published data on indoor concentrations of VOCs in residential buildings (existing and new) and office buildings (primarily large buildings) in North American starting from the year 1990. Their review excluded some compounds, such as very volatile compounds and compounds with low occurrence. Thirty-five of the compounds they summarized are classified as hazardous air pollutants (HAPs). VOCs with maximum concentrations of 50 ppb or more in office buildings included ethanol, 2-propanol, n-octane, toluene, dichloromethane, 1,1,1-trichloroethane, and 2-propanone. VOCs with maximum concentrations of 50 ppb or more in existing residences included: acetic acid, formaldehyde, toluene, m/p-xylene, 1,4-dichlorobenzene, dichloromethane, 1,1,1-trichloroethane, and 2-

propanone; in new houses, acetic acid, formaldehyde, acetaldehyde, hexanal, toluene, ethylene glycol, 1,2-propanediol, 2-propanone, and α-pinene.

Hodgson et al. (2004) carried out VOC sampling in four portable classrooms located in California public schools. Two of the classrooms were built and furnished with materials that had low VOC emissions. The other two classrooms were used as controls. Hodgson et al. (2004) measured ventilation rates and made simultaneous outdoor sample measurements. HVAC units were operational during the studies. For all measurements they used 6-7 hour sampling. Hodgson et al. (2004) found that higher ventilation rates were associated with lower VOC concentrations. Of the 15 VOCs targeted in the study, the average concentrations observed were around 1 ppb. Only formaldehyde concentration was found to exceed 5 ppb.

Shendell et al. (2004) carried out indoor air sampling in seven schools in California. They sampled 20 classrooms (including 13 portable classrooms) for a range of VOCs. They used passive samplers with sampling times ranging from 1 day to 1 week and sampled during the winter and summer seasons. VOC concentrations were found to be lower during the winter compared to summer. Overall, concentrations of VOCs were found to be low in this study compared to previous studies. Acetaldehyde, formaldehyde, toluene, m,p-xylene, α-pinene, and d-limonene were the most frequently detected compounds, however none of the concentrations were found to exceed regulatory thresholds. Shendell et al. (2004) also had technicians do walk-ins to identify potential indoor sources of VOCs. Cleaning products, personal care products, indoor furnishings and finish, and teaching materials were identified as potential sources. In addition to successfully using passive samplers for measurements, the study highlighted the need to study ventilation in schools.

The state of California commissioned a 2006 study (East End Study) to assess indoor air quality in a newly constructed office-building complex (Alevantis et al. 2006). VOC and aldehyde sampling were carried out multiple times over 12 months in 5 buildings before and after they were occupied (21-site visits), allowing for an evaluation of temporal variations in concentrations. The study started with a target list of 110 chemicals. Samples were collected for 5-6 hours during each sampling event. Ventilation rates were also measured. The study reported that apart from formaldehyde and acetaldehyde, all other VOCs were at levels largely below target concentrations. The levels were compared to VOC concentrations reported in the BASE study, with a few VOCs being detected at higher concentrations compared to BASE. Concentrations of chloroform, phenol, 1-ethyl-4-methyl-benzene, texanol, α-pinene, 1,2,4- and 1,3,5-trimethyl benzene were found to exceed the BASE concentrations by more than a factor of 2. Periodic sampling also allowed to researchers to compare increases in certain VOC concentration with activities in the buildings. Acetaldehyde, formaldehyde, caprolactam, naphthalene and nonanal were identified as building-related compounds. Other compounds such as benzaldehyde, D5 siloxane, and d-limonene were linked to occupants.

Godwin et al. (2006) randomly selected four elementary schools and five middle schools in Michigan to undergo indoor pollutant sampling. The researchers selected a variety of rooms within each school for sampling sampled (one art room, miscellaneous-use room, general classrooms, science rooms, and clerical rooms). They also sampled both outdoors and indoors (in each room) for temperature, relative humidity, CO_2, VOCs and bioaerosols. Sampling was carried out over 3.5-4.5 days in the schools. Temperature, relative humidity and CO_2 were sampled every 5-minutes during the course of monitoring. VOCs were collected onto Tenax

tubes and sampled in a gas chromatograph/mass spectrometer. The researchers made a visual inspection of the rooms and recorded the method of ventilation employed in the schools. They used the difference in CO_2 levels indoors and outdoors to estimate the air exchange rate in the rooms. Sampling was carried out in portable classrooms only in one school. Benzene, toluene, ethylbenzene, xylene, α-pinene and d-limonene were the most frequently detected compounds. With the exception of α-pinene and d-limonene, the researchers found concentrations of detected compounds to be below levels in schools reported earlier by Shendell et al. (2004). Indoor/outdoor concentration ratios for α-pinene, d-limonene, ethylbenzene, xylene, 2-butanone, methyl isooctane, toluene, chloroform, 1,2,4-trichlorobenzene, styrene, phenol, naphthalene were found to be reasonably high indicating the presence of indoor sources. Benzene had a much smaller indoor/outdoor ratio highlighting that outdoor sources were significant compared to indoor sources. The presence of swimming pools appeared to account for the trace concentrations of chloroform, 1,2,3-trichlorobenzene, and trichloroethylene found in schools. High concentrations of toluene, phenol, MIBK, and 1,2,4-trichlorobenzene were found in art rooms. High concentrations of naphthalene and α-pinene were found in science rooms. The study did not find a significant difference in total VOC concentrations between middle schools and elementary schools, and total VOC concentrations were found to be fairly low. The ventilation in most schools was inadequate compared to the ASHRAE 62.1 standard of 8 L/s-person. The CO_2 levels, however, exceeded the 1,000-ppm limit recommended by ASHRAE. Median biological pollutant concentrations measured in terms of counts/m^3 were found to be comparable to values in other commercial buildings. Regression analysis indicated that carpets and occupants were positively correlated to bioaerosol levels, and α-pinene was negatively correlated. The VOC concentrations were also found to be sensitive to changes in ventilation rates. The study highlighted the spatial variations of VOC concentrations due to the presence of localized sources in schools.

Hotchi et al. (2006) carried out VOC measurements in a Target store in the San Francisco Bay Area. Their goal was to determine whether turning off some air-handling units during load-shedding impacted VOC concentrations. They reported that formaldehyde, 2-butoxyethanol, DPGME, toluene, and D5 siloxane were found in highest concentrations at the sales area. Concentrations of compounds increased after the load-shedding events with fractional increases ranging from 0.11 to 1.28 times the pre-shedding concentrations. They sampled for about 34 VOCs, during the study. Formaldehyde and acetaldehyde concentrations were found to be similar to concentrations reported in the BASE study.

Loh et al. (2006) measured VOCs in several types of stores in Boston, using personal samplers. Sampling was carried out in a variety of stores, restaurants (1.5-h sampling) and on transportation systems (3-h sampling) during summer 2003 and winter 2004. Sampling was carried in a variety of stores such as hardware, multipurpose (7-h sampling), grocery, drug, sporting goods, furniture, housewares, department, and electronics stores. Concentrations of formaldehyde were highest in housewares stores (GM=53 μg/m^3), highest levels were measured in multipurpose stores (GM=76 μg/m^3). Restaurants had high concentrations of chloroform (GM=1.1 μg/m^3). Overall, stores had high concentrations of formaldehyde, toluene, ethylbenzene, xylenes, styrene, chlorinated compounds. They also reported that benzene concentrations indoors were not found to be much higher than concentrations outdoors. Additionally, housewares stores also had high concentrations of compounds such as limonene

and unsaturated hydrocarbons. Loh et al. (2006) also reported significant differences in formaldehyde and acetaldehyde concentrations during the summer and winter sampling events.

Eklund et al. (2008) carried out sampling in 10 retail stores located in a New Jersey shopping mall. They collected more than 130 8-h time-integrated samples over a 2-year period between 2002-2005. The types of stores sampled included: jewelery, hair/nail salon, restaurants, clothing rental, dry-cleaner, video rental and optician. Eklund et al. (2008) provided summary statistics for 28 VOCs detected in 10% or more of the samples. Concentrations of acetone (31), ethanol (28), tetrachloroethylene (12), isopropyl alcohol (8), ethyl acetate (5), toluene (5), methyl ethyl ketone (1), and tetrahydrofuran (1) exceeded 1,000 $\mu g/m^{-3}$ in one or more samples—here values in parentheses are the number exceeding 1000 $\mu g/m^{-3}$. Eklund et al. (2008) highlighted that VOC concentrations are widely variable in commercial spaces depending on the type of activity and indoor sources. High average (the arithmetic mean of all measurements) levels of acetone (27000 $\mu g/m^{-3}$), ethanol (1850 $\mu g/m^{-3}$), ethyl acetate (649 $\mu g/m^{-3}$), toluene (1150 $\mu g/m^{-3}$) and isopropyl alcohol (671 $\mu g/m^{-3}$) were detected in nail salons. High average concentrations of isopropyl alcohol were measured in the jewelry stores (6320 $\mu g/m^{-3}$) and the optician store (105 $\mu g/m^{-3}$). High concentrations of tetrachloroethylene were observed in the clothes rental stores (2540 $\mu g/m^{-3}$) and dry-cleaning establishments (1010 $\mu g/m^{-3}$). High average ethanol concentrations were measured in restaurants and the optician store. Large spatial variability was associated with VOCs such as acetone, toluene, ethanol and toluene indicating that their concentrations are influenced by significant indoor sources.

The small and medium commercial buildings study (SMCB) measured concentrations of 30 VOCs in 37 California buildings (Bennett et al. 2011). Sampling was carried out in the following types of buildings (the number of buildings is listed in parenthesis): beauty salons (2), dentist offices (2), convenience stores at gas stations (2), fitness centers (2), grocery stores (2), offices (2), restaurants (4), retailers (8), religious facilities (2), and public assembly space (1). The GM concentrations of most compounds were well below the Office of Environmental Health and Hazard Assessment (OEHHA) intermediate and chronic exposure limits. Geometric means of formaldehyde concentrations in dentist offices, convenience stores, fitness centers, restaurants, retailers, religious facilities, assembly spaces, offices, and beauty salons were found to exceed the OEHHA 8-hr and chronic RELs (9 ppb), and the ATSDR chronic REL (8 ppb). The mean tetrachloroethylene concentrations exceeded the OEHHA 8-hr REL of 5 ppb at gas stations (17 ppb), dentist offices (17 ppb), and other spaces such as religious facilities or the public assembly space. The study provided insight into the variations in VOC concentrations in different types of buildings.

The Healthy Zero Energy Buildings (HZEB) Study was conducted between 2010-2014 to evaluate the indoor air quality in commercial buildings, specifically retail stores and included grocery, furniture, and apparel stores (Fisk et al. 2016). The HZEB researchers measured the ventilation rates and indoor air quality in 18 retail stores. They measured VOC concentration, size resolved PM mass and count concentrations, ozone, CO_2 and CO levels in the stores. They sampled VOCs using sorbent tubes and sampled formaldehyde and acetaldehyde using DNPH cartridges. In addition, they measured ventilation rates using the SF6 tracer method. They reported formaldehyde, acetaldehyde, octanal, and acrolein at levels that exceed regulatory thresholds, and sensory irritation thresholds. Cooking related activities, in grocery stores were

found to increase acrolein concentration. Similarly, they reported ultrafine PM concentrations to be higher in grocery stores where there were cooking emissions.

A study of retail stores (ASHRAE Retail study) measured indoor air quality at 14 retail stores (Siegel et al. 2013). The researchers used Summa canisters and sorbent tubes to sample for VOCs, di-nitro phenyl hydrazine (DNPH) tubes for formaldehyde and acetaldehyde, and polyurethane foam (PUF) tubes for SVOCs. Ventilation rates in the stores were measured using the SF6 tracer method. Overall, the retail stores were found to have lower concentration of most VOCs compared to other commercial buildings. Formaldehyde concentrations were found to range between 2.6 and 66.9 ppb, with most concentrations exceeding the OEHHA 8-hour threshold (OEHHA 2008). They also reported elevated concentrations of certain VOCs depending on the activity in the retail stores. Acetone (5 to 370 ppb range, 17 ppb median), benzene, toluene, ethyl benzene and xylene were detected in all the retail stores. The researchers also examined ventilation rate changes in some stores and measured the change in pollutant concentrations. The goal was to examine whether formaldehyde concentrations can be reduced by increasing ventilation. They used OEHHA's chronic reference level of 7.3 ppb and reported that given outdoor formaldehyde levels, ventilation rates would have to be increased by a factor of 9 to get formaldehyde concentrations below reference levels.

METHODS

Chemical Property Analysis

The literature review helped us to determine some 115 chemicals that are present in commercial buildings in the United States. A k-means cluster analysis was conducted and the chemicals were grouped based on their positions in Hansen solubility space. The algorithm first calculates centroids for the data based on the number of groups specified. The squared Euclidean distance from the centroid to each point is calculated and individual data points are assigned to the groups based on distance from the centroids. MATLAB was used to conduct all the analysis. Three parameters were used to conduct the analysis: octanol-air partitioning coefficient (K_{oa}), octanol-water partitioning coefficient (K_{ow}), and air-water partitioning coefficient (K_{aw}). The parameters were calculated using EPISUITE (USEPA EPISUITE 2011). Chemicals in each group are expected to behave similarly in the indoor environment, since the parameters they are grouped on affect their transport in the indoor environment.

Five groups of chemicals were identified, which are displayed in Figure 2-1. The first k-means analysis created five groups of chemicals, however we wanted more resolution in Groups 1 and 2 of the first iteration. So we combined Groups 3, 4, and 5 since they had similar levels of volatility and then conducted another k-means analysis on Groups 1 and 2. The new k-means analysis on Groups 1 and 2 was used to form four new groups of chemicals. The chemicals in each group are presented in Table 2-3B. In Chapter 3 representative chemicals from each group are used to conduct the analysis in the model. The results are used to inform the effect of ventilation on removal of the compound from the indoor environment is estimated.

Toxicity, odor and sensory thresholds based analysis

This analysis uses toxicity thresholds (non-cancer, reproductive, cancer) and perceptions of air quality (odor and pungency thresholds) to determine which compound concentrations exceed any

thresholds. We compared observed indoor concentrations to health-based concentration levels. The levels we used are those established to protect the general population from acute health hazards, reproductive toxicity, and cancer. Second we compared observed indoor concentrations to odor and sensory threshold levels. In the sections below we describe this analysis in more detail.

Health-related thresholds for indoor VOC contaminants of concern - Overview

To determine which compounds pose a potential health concern for indoor spaces, we compared measured air concentrations of the compounds reported in various studies to the most health protective standards set by well-established authorities. Numerous agencies such as the federal Occupational Safety and Health Administration (OSHA), the California Office of Environmental Health Hazard Assessment (OEHHA), Agency for Toxic Substances and Disease Registry (ATSDR), and the United States Environmental Protection Agency (USEPA) have established health guidelines for various compounds. OSHA's permissible exposure limits (PELs) for workers, were largely adopted from the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit values. They were adopted around 1968, and most of the numbers remain in effect till today, even though ACGIH has updated its TLVs. OSHA's PELs are geared towards protecting the "healthy workers" and do not account for variations in susceptibility and vulnerability in the general population.

The California OEHHA has established risk-based Reference Exposure Levels (RELs), following guidelines in the National Academy of Sciences report "Science and Decisions: Advancing Risk Assessment". OEHHA has developed the REL numbers based on currently available toxicology and dose-response data applicable to the general population. The USEPA (USEPA IRIS) has also applied the elements of classic risk assessment framework--(i) hazard identification, (ii) exposure assessment (iii) dose-response and (iv) risk characterization--in order to identify the reference inhalation concentrations (RfC), corresponding to virtually risk safe dose. The RfC is obtained from the no observed adverse effect levels (NOAELs) or lowest observed adverse effect levels (LOAELs) in toxicology experiments combined with safety/uncertainty factors and expected exposure factors. The ATSDR has also developed maximum recommended [exposure] levels (MRLs) for various compounds, using an approach similar to the USEPA. To make use of these data in our screening, we compiled and compared the non-cancer guidelines developed by these agencies and then selected the most limiting exposure based on a hazard ratio (the actual-dose/safe-dose ratio) to identify contaminants of concern (Table 2-3A). The lowest chronic thresholds among limits provided by various regulatory agencies (OEHHA's RELs, ATSDR's MRLs, EPA's RfCs) were determined. We used intermediate and acute thresholds when chronic thresholds were not available.

To evaluate compounds on the basis of their potential for reproductive toxicity, we employed the maximum allowable dose level (MADL) developed under Proposition 65 by OEHHA (2010). The MADLs (μg/day) were converted to 24-hour concentrations (μg/m^3) by dividing them with assumed breathing rates (15 m^3/d, Layton 1993).

To address protection against cancer risk we used the No Significant Risk Level (NSRL) standards for inhalation (μg/day) provided under Proposition 65 by OEHHA (2010). Similar to MADLs, the NSRLs are converted to 24-hour concentrations (μg/m^3) by dividing them with breathing rates (15 m^3/d, Layton 1993) and compared to concentrations of interest.

Odor and sensory thresholds-Overview

Occupants of buildings are typically exposed to a wide array of VOCs and SVOCs, and they respond to the indoor levels of these substances based on their sensory perceptions of concentration. Sensory perception is a criterion used to determine *acceptability* of air quality indoors (ASHRAE 1999). According to Cain and Schmidt (2009) there are orders of magnitude variations among odor thresholds of compounds reported in numerous studies. Cain and Schmidt (2009) hypothesize that systemic variations (experimental procedure, definition of odor threshold used by author) contribute to most of the variations in values compared to random variations. Schmidt and Cain (2010) report that odor thresholds determined using 'vapor delivery device 8' (VDD8), have been consistently found to be orders of magnitude lower than thresholds in current literature. The device allows for sampling of the actual concentration of vapor delivered to the subject, and does not allow for dilution by surrounding air hence reducing bias (see Schmidt and Cain 2010 for more details on VDD8).

However, odor thresholds have been established for very few compounds using the VDD8. Nagata (2003) employed a triangle bag odor method to establish a homogenous odor thresholds database for approximately 220 compounds. Cain and Schmidt (2009) found the odor thresholds of n- and tert-butyl acetate reported by Nagata (2003), to be closest to thresholds determined using the VDD8. Hodgson et al (2003a) conducted an analysis of odor and sensory irritation levels for substances that had been described in terms of odor/sensory irritation and non-cancer health guidelines in the archival literature. From their review, they developed a method to arrive at a reference concentration for both odor/sensor response and non-cancer health effects. These reference levels were compared to residential and office concentrations, which had been compiled earlier (Hodgson et al. 2003). Their analysis showed that some alcohols (1-octanol), carboxylic acids (acetic acid, hexanoic acid), higher molecular weight aldehydes (hexanal, heptanal, octanal, nonanal, 3-methyl butanal) were most odorous (odor threshold<10 ppb). Acrolein, butylated hydroxy toluene, diethyl phthalate, acetic acid and 2-ethyl-1-hexanol had the lowest sensory irritation thresholds.

In the current study we rely primarily on odor and pungency thresholds reported in Cain and Schmidt. (2009), Hodgson et al. (2003a) and Nagata (2003). We selected the lowest thresholds among these studies to screen compounds of concern.

We have outlined the procedure followed to develop indices using health endpoints of concern for "safe/acceptable" air. Even though the studies report different durations of short-term sampling measurements, we compared the concentrations to chronic thresholds since chronic thresholds are much lower than acute or 8- thresholds. A meta-analysis of VOC concentrations reported in various studies was first conducted to determine the representative concentration of each VOC to be used for screening. The concentrations were arrived at as follows (see Appendix A, Tables A1 and A2):

- The measure of central tendency reported (mean/median) were compared across all the studies to determine the highest concentration.
- If SD was reported along with maximum mean concentration, the 98^{th} percentile value was calculated.
- If 90^{th}/95^{th} percentile/maximum values were reported along with maximum median they were used for the analysis.

The health endpoints of concern for "safe/acceptable" air used for screening are a) chronic, (or) intermediate (or), acute non-cancer toxicity thresholds b) cancer toxicity thresholds c) reproductive toxicity thresholds d) odor and pungency thresholds.

We determined whether any VOC concentration was within 90% of the threshold of interest, and developed various indices based on the formulae listed below. The results are tabulated in Table 2-1. Table A-1 of the Appendix contains more details on the health thresholds used.

Table 2-1: Calculation of toxicity and odor indices

Formula	Index
$Conc._{voc}$ / (0.9 × Non-cancer toxicity threshold) =	Non-cancer tox. Index (NCI)
$Conc._{voc}$ / (0.9 × Reproductive toxicity threshold =	Reproductive tox. Index (RI)
$Conc._{voc}$ / (0.9 × Cancer toxicity threshold) =	Cancer tox. Index (CI)
$Conc._{voc}$ / (0.9 × Odor toxicity threshold) =	Odor Index (OI)
$Conc._{voc}$ / (0.9 × Pungency toxicity threshold) =	Pungency Index (PI)

RESULTS

This work provides a screening analysis to identify contaminants of concern in commercial buildings in California. The screening is largely based on studies, which have reported VOC concentrations in office buildings in USA. Multiple criteria were used for the screening assessment: non-cancer acute, intermediate and chronic toxicity; odor and irritancy thresholds; reproductive toxicity; and cancer potency. The most health protective guidelines issued were used for the screening. The compounds were grouped into Lists A and B. VOCs included on List A are sometimes present in commercial buildings at concentrations that may pose risks to health or degrade perceived air quality. However, their significance with respect to the setting of minimum ventilation standards will also depend on whether the primary sources are indoors, or the outdoor air. List B comprises all the other compounds: concentrations are not high enough from a health-based perspective, no health guidelines exist, no measured concentrations. The pollutants on List A are listed here, along with the indices that were exceeded in parenthesis. The pollutants on List A are: acetaldehyde (NCI, CI, OI), acrolein (OI), benzene (NCI, RI, CI), 1,3-butadiene (NCI, CI, OI), butyl acetate (OI), carbon tetrachloride (CI), chloroform (CI), decanal (OI), 1,4-di-chlorobeznene (NCI, CI, OI), di-chloromethane (CI), ethyl benzene (CI), formaldehyde (NCI, CI, PI), hexanal (OI), d-limonene (OI), naphthalene (NCI, CI, OI), nonanal (OI), octanal (OI), pentanal (OI), α-pinene (OI), tetrachloroethane (CI), tetrachloroethene (CI), trichloroethylene (CI), toluene (NCI, RI, OI), and m/p-xylene (NCI, CI, OI).

For the second screening process, we conducted a k-means analysis to group chemicals to gauge the impact of building characteristics on their concentration. The compounds were grouped into Groups 1, 2, 3, 4 and 5. The VOCs in Groups 4 and 5 have the physical characteristics that make their indoor concentration susceptible to changes in ventilation rates. As seen in Figure 2-1, the groups of chemicals are separated by their solubility in different phases. The VOCs/SVOCs in Groups 1, 2 and 3 have physical characteristics that make their indoor concentration susceptible to changes in ventilation rates and filtration efficiencies. All of the compounds in List A are also in Group 5, based on the chemical property analysis. The compounds in List A, could also have strong indoor sources. The analysis indicates that minimum ventilation rate standards should be structured around controlling indoor exposures to these compounds.

LIMITATIONS

This analysis only considered gaseous or semivolatile contaminants. Particulate contaminants and inorganic gaseous pollutants have not yet been considered. Particles emitted from indoor sources are expected to pose health risks. Much is known about the health impacts of outdoor air particles but relatively little is known about the magnitude of the risks from indoor-generated particles. In general, ventilation will be a poor strategy for controlling indoor concentrations of indoor-generated particles in commercial buildings. If the building has no particle filtration or only very low efficiency filtration, increased ventilation will remove indoor-generated particles from the indoor air but bring in outdoor air particles. If a building has a moderate or high rate of particle filtration, which is common in commercial buildings, the ventilation rate will have a small impact on indoor concentrations of particles because particle removal by filtration dominates relative to particle removal by ventilation. Inorganic gaseous pollutants such as carbon monoxide, nitrogen oxides, ozone, and radon also pose health risks. In general, the sources of these pollutants are small in commercial buildings, with outdoor air as the primary sources for all, or for all except radon. Consequently, risks from these contaminants are not expected to be a factor that drives the selection of minimum ventilation rates in most commercial buildings. However, further analyses are needed to determine if there are exceptions in which these contaminants must be considered.

FIGURE

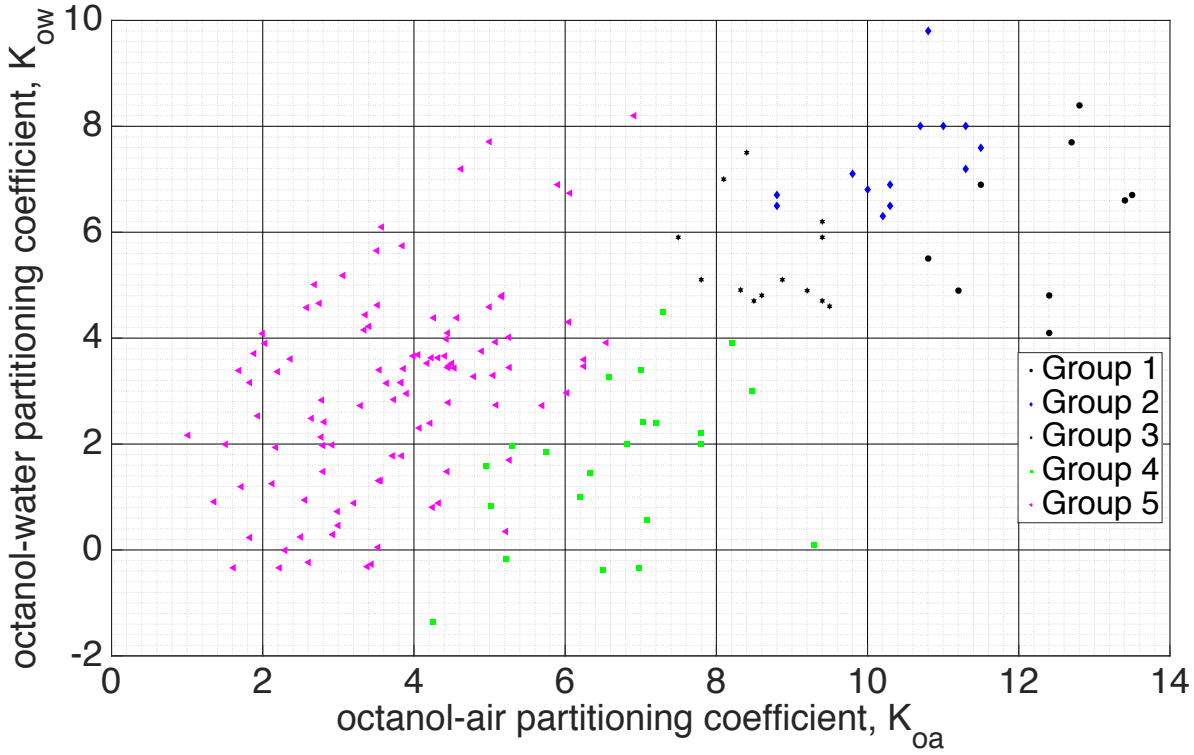

Figure 2-1: Results of hierarchical k-means analysis of chemical properties. The five chemical groups are shown

TABLES

Table 2-2: Details of key studies that report VOC and SVOC measurements

Study	Number of buildings	Sample period
Daisey et al (1994)	12 office buildings	June - September, 1990
Shields (1994)	60 Telecommunications offices, data centers and office building	March - April 1991
Apte and Erdmann (2002)	100 office buildings	1994 - 1998
California Schools (2004)	201 portable and traditional classrooms	2000-2003
Hodgson et al. (2004)	4 relocatable classrooms	Fall 2001
Shendell et al. (2004)	13 portable classrooms and 7 traditional classrooms	June 2000-June 2001
East End (Alevantis et al. 2006)	5 office buildings	2002-2004
Godwin et al. (2006)	64 elementary and middle school classrooms	March – June 2003
Hotchi et al (2006)	1 retail store	October 2005
Loh et al (2006)	12 Stores, dining	Summer 2004, Winter 2004, Winter 2005
Eklund et al (2008)	1 strip mall	October 2002 - June 2005
SMCB (Bennett et al. 2011)	37 small and medium commercial buildings	2008-2010
ASHRAE Retail Stores Study (Siegel et al. 2013)	14 retail buildings	2011-2012
Healthy Zero Energy Buildings (Fisk et al. 2016)	21 retail stores	2011-2013

Table 2-3A: Chemicals groupings based on toxicity

Compound	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index	Toxicity List
	>=0.1 ?					
Acetaldehyde	**x**		**x**	**x**		**List A**
Acetic acid						**List B**
Acetophenone						**List B**
Acrolein				**x**		**List A**
Acrylonitrile						**List B**
Benzene	**x**	**x**	**x**			**List A**
Benzaldehyde						**List B**
Benzothiazole						**List B**
Benzyl acetate						**List B**
Benzyl chloride						**List B**
Bornyl acetate						**List B**
Bromomethane (methyl bromide)						**List B**
1,3-Butadiene	**x**		**x**	**x**		**List A**
1-Butanol						**List B**
2-Butanone						**List B**
2-Butoxyethanol						**List B**
Butylacetate				**x**		**List A**
Butylated hydroxytoluene						**List B**
Butylbenzene						**List B**
t-Butyl methyl ether (MTBE)						**List B**
n-Butyraldehyde						**List B**
Camphene						**List B**
Carbon disulfide						**List B**
Carbon tetrachloride			**x**			**List A**
Chlorobenzene						**List B**
Chloroform			**x**			**List A**
Chloromethane						**List B**
a-Citral						**List B**
b-Citronellol						**List B**
Cyclohexanone						**List B**
Cyclohexyl benzene						**List B**
p-Cymene						**List B**
n-Decane						**List B**

Compound	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index	Toxicity List
	>=0.1 ?					
Decanal				x		**List A**
1,2-Dichlorobenzene						**List B**
1,3-Dichlorobenzene						**List B**
1,4-Dichlorobenzene	x		x	x		**List A**
Dichlorodifluoromethane						**List B**
1,2-Dichloroethane (ethylene dichloride)						**List B**
Dichloromethane (methylene chloride)			x			**List A**
1,2-Dichloropropane						**List B**
Diethyl phthalate						**List B**
Di(ethylene glycol) butyl ether						**List B**
Dihydromyrcenole						**List B**
7-Dimethyl-3-octanol						**List B**
1,4-Dioxane						**List B**
Di(propylene glycol)methyl ethers (DPGME)						**List B**
Dodecane						**List B**
Ethanol						**List B**
Ethyl acetate						**List B**
Ethylbenzene			x	x		**List A**
Ethylcarbonate						**List B**
2-Ethyl-1-hexanol						**List B**
2-Ethyltoluene						**List B**
3/4-Ethyltoluene						**List B**
4-Ethyltoluene						**List B**
Ethylene glycol						**List B**
Eucalyptol						**List B**
Formaldehyde	x		x		x	**List A**
n-Heptane						**List B**
1,4-Hexachloro butadiene						**List B**
n-Hexadecane						**List B**
n-Hexane						**List B**
Hexanal				x		**List A**
2-hexyloxyethanol						**List B**
Isopropylbenzene						**List B**

Compound	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index	Toxicity List
	>=0.1 ?					
d-Limonene				**x**		**List A**
Linalool						**List B**
Linalyl acetate						**List B**
Methylcarbonate						**List B**
Methylcyclohexane						**List B**
Methylcyclopentane						**List B**
3-Methylhexane						**List B**
1-Methyl-2-pyrrolidinone						**List B**
4-methyl-2-pentanone (MIBK)						**List B**
α-Methylstyrene						**List B**
Naphthalene	**x**		**x**	**x**		**List A**
Nonanal				**x**		**List A**
Nonane						**List B**
Octane						**List B**
Octanal				**x**		**List A**
n-Pentadecane						**List B**
Pentanal (valeraldehyde)				**x**		**List A**
Pentane						**List B**
a-Phellandrene						**List B**
Phenol						**List B**
4-Phenylcyclohexene						**List B**
α-pinene				**x**		**List A**
β-pinene						**List B**
2-Propanol (isopropanol)						**List B**
2-Propanone (acetone)						**List B**
n-Propylbenzene						**List B**
Styrene						**List B**
D4 Siloxane						**List B**
Terpineols						**List B**
Tetrachloroethane			**x**			**List A**
Tetrachloroethene			**x**			**List A**
n-Tetradecane						**List B**
Tetrahydrofuran						**List B**
TMPD-DIB						**List B**
TMPB-MIB						**List B**

Compound	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index	Toxicity List
	>=0.1 ?					
Toluene	**x**	**x**		**x**		**List A**
1,2,4-Trichlorobenzene						**List B**
1,1,1-Trichloroethane (Methyl chloroform)						**List B**
Trichloroethene (Trichloroethylene)			**x**			**List A**
Trichlorofluoromethane						**List B**
Trichlorotrifluoroethane						**List B**
1,2,4-Trimethylbenzene						**List B**
1,2,3-Trimethylbenzene						**List B**
1,3,5-Trimethylbenzene						**List B**
Trimethylcyclohexenone						**List B**
2,2,5-Trimethylhexane						**List B**
2,2,4-Trimethylpentane						**List B**
n-Undecane						**List B**
o-xylene						**List B**
mp-xylene	**x**			**x**		**List A**
BDE-153 (hexa BDE)						**List B**
Cyfluthrin						**List B**
Cypermethrin						**List B**
BDE-99 (penta BDE)						**List B**
Di(2-ethylhexyl) phthalate (DEHP)						**List B**
2,3,7,8-Tetrachlorodibenzo-p-dioxin						**List B**
Stearic acid						**List B**
Permethrin						**List B**
Perchloropentacyclodecane (mirex)						**List B**
BDE-47 (tetra BDE)						**List B**
Bisphenol A						**List B**
Benzo[a]pyrene						**List B**
Linoleic acid						**List B**
Piperonyl butoxide						**List B**
Butylbenzyl phthalate (BBzP)						**List B**
Di(2-ethylhexyl) adipate (DEHA)						**List B**

Compound	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index	Toxicity List
	>=0.1 ?					
PCB 153						**List B**
p,p-DDT						**List B**
Mirex						**List B**
Triphenylphosphate						**List B**
Triclosan						**List B**
Dibutyl phthalate (DBP)						**List B**
Diazinon						**List B**
Chlorpyrifos						**List B**
Chlordane						**List B**
PCB 52						**List B**
Pyrene						**List B**
Galaxolide						**List B**
Pentachlorophenol (PCP)						**List B**
4-Nonylphenol						**List B**
EtFOSE						**List B**
Propoxur						**List B**
Butylated hydroxytoluene (BHT)						**List B**
Phenanthrene						**List B**
Methyl parathion						**List B**
MeFOSE						**List B**
Tris(chloropropyl) phosphate						**List B**
Texanol 2						**List B**
Geosmin						**List B**
Nicotine						**List B**
Caryophyllene						**List B**
Pinonaldehyde						**List B**

Table 2-3B: Chemical groupings based on chemical properties

Compound	log (K_{ow})	log (K_{aw})	log (K_{oa})	Chemical Property Group
Acetaldehyde	-0.34	-2.55	2.22	**4**
Acetic acid	-0.17	-4.64	5.22	**5**
Acetophenone	1.58	-3.39	4.95	**5**
Acrolein	-0.01	-2.83	2.29	**4**
Acrylonitrile	0.25	-2.24	2.50	**4**
Benzene	2.13	-0.65	2.77	**4**
Benzaldehyde	1.48	-3.25	4.44	**4**
Benzothiazole	2.01	-4.81	6.83	**5**
Benzyl acetate	1.96	-3.23	5.30	**5**
Benzyl chloride	2.30	-1.06	4.07	**4**
Bornyl acetate	4.30	-1.74	6.05	**4**
Bromomethane (methyl bromide)	1.19	-0.45	1.71	**4**
1,3-Butadiene	1.99	0.51	1.51	**4**
1-Butanol	0.88	-3.38	4.32	**4**
2-Butanone	0.29	-2.56	2.92	**4**
2-Butoxyethanol	0.83	-5.39	5.01	**5**
Butylacetate	1.78	-1.77	3.72	**4**
Butylated hydroxytoluene	5.10	-3.77	8.87	**2**
Butylbenzene	4.38	-0.24	4.57	**4**
t-Butyl methyl ether (MTBE)	0.94	-1.08	2.56	**4**
n-Butyraldehyde	0.88	-2.30	3.21	**4**
Camphene	4.22	0.83	3.40	**4**
Carbon disulfide	1.94	0.10	2.17	**4**
Carbon tetrachloride	2.83	0.02	2.78	**4**
Chlorobenzene	2.84	-0.78	3.74	**4**
Chloroform	1.97	-0.87	2.79	**4**
Chloromethane	0.91	-0.47	1.35	**4**
a-Citral	3.45	-1.81	5.26	**4**
b-Citronellol	3.91	-2.63	6.54	**4**
Cyclohexanone	0.81	-2.67	4.24	**4**
Cyclohexyl benzene	4.81	-0.35	5.17	**4**
p-Cymene	4.10	-0.32	4.45	**4**
n-Decane	5.01	2.34	2.69	**4**
Decanal	3.76	-1.56	4.89	**4**
1,2-Dichlorobenzene	3.43	-0.91	4.54	**4**
1,3-Dichlorobenzene	3.53	-0.91	4.50	**4**

Compound	log (K_{ow})	log (K_{aw})	log (K_{oa})	Chemical Property Group
1,4-Dichlorobenzene	3.44	-0.91	4.45	**4**
Dichlorodifluoromethane	2.16	1.08	1.01	**4**
1,2-Dichloroethane (ethylene dichloride)	1.48	-0.30	2.80	**4**
Dichloromethane (methylene chloride)	1.25	-0.42	2.13	**4**
1,2-Dichloropropane	1.98	-0.17	2.92	**4**
Diethyl phthalate	2.42	-4.78	7.02	**5**
Di(ethylene glycol) butyl ether	0.56	-7.20	7.09	**5**
Dihydromyrcenole	3.47	-2.77	6.25	**4**
7-Dimethyl-3-octanol	3.60	-2.64	6.25	**4**
1,4-Dioxane	-0.27	-3.61	3.44	**4**
Di(propylene glycol)methyl ethers (DPGME)	-0.35	-7.32	6.98	**5**
Dodecane	6.10	2.59	3.58	**4**
Ethanol	-0.31	-3.63	3.38	**4**
Ethyl acetate	0.73	-2.01	2.99	**4**
Ethylbenzene	3.15	-0.48	3.64	**4**
Ethylcarbonate	-0.34	-1.94	1.61	**4**
2-Ethyl-1-hexanol	2.73	-2.89	5.70	**4**
2-Ethyltoluene	3.53	-0.44	4.18	**4**
3/4-Ethyltoluene	3.98	-0.44	4.43	**4**
4-Ethyltoluene	3.63	-0.44	4.32	**4**
Ethylene glycol	-1.36	-5.26	4.25	**5**
Eucalyptol	2.74	-2.07	5.09	**4**
Formaldehyde	0.35	-2.41	5.21	**4**
n-Heptane	4.66	1.98	2.75	**4**
1,4-Hexachloro butadiene	4.78	-0.35	5.16	**4**
n-Hexadecane	8.20	3.08	6.91	**4**
n-Hexane	3.90	1.85	2.03	**4**
Hexanal	1.78	-2.06	3.84	**4**
2-hexyloxyethanol	1.86	-5.14	5.75	**5**
Isopropylbenzene	3.66	-0.36	3.99	**4**
d-Limonene	4.38	1.20	4.27	**4**
Linalool	2.97	-2.75	6.03	**4**
Linalyl acetate	3.93	-1.14	5.08	**4**
Methylcarbonate	0.23	-1.59	1.83	**4**
Methylcyclohexane	3.61	1.15	2.37	**4**
Methylcyclopentane	3.37	1.03	2.20	**4**

Compound	log (K_{ow})	log (K_{aw})	log (K_{oa})	Chemical Property Group
3-Methylhexane	3.71	1.98	1.88	**4**
1-Methyl-2-pyrrolidinone	-0.38	-5.88	6.50	**5**
4-methyl-2-pentanone (MIBK)	1.31	-2.32	3.56	**4**
α-Methylstyrene	3.48	-0.74	4.46	**4**
Naphthalene	3.30	-1.66	5.05	**4**
Nonanal	3.27	-1.69	4.79	**4**
Nonane	5.65	2.22	3.51	**4**
Octane	5.18	2.10	3.06	**4**
Octanal	2.78	-1.81	4.46	**4**
n-Pentadecane	7.71	2.96	5.00	**4**
Pentanal (valeraldehyde)	1.31	-2.18	3.53	**4**
Pentane	3.39	1.73	1.68	**4**
a-Phellandrene	4.62	1.11	3.52	**4**
Phenol	1.46	-4.63	6.33	**5**
4-Phenylcyclohexene	4.59	-0.40	5.00	**4**
α-pinene	4.44	0.65	3.36	**4**
β-pinene	4.16	0.83	3.34	**4**
2-Propanol (isopropanol)	0.05	-3.50	3.53	**4**
2-Propanone (acetone)	-0.24	-2.68	2.60	**4**
n-Propylbenzene	3.69	-0.36	4.06	**4**
Styrene	2.95	-0.94	3.90	**4**
D4 Siloxane	6.74	0.56	6.06	**4**
Terpineols	3.28	-3.18	6.58	**5**
Tetrachloroethane	2.39	-1.20	4.21	**4**
Tetrachloroethene	3.40	-0.16	3.54	**4**
n-Tetradecane	7.20	2.84	4.63	**4**
Tetrahydrofuran	0.46	-2.45	3.00	**4**
TMPD-DIB	4.91	-3.41	8.32	**2**
TMPB-MIB	3.00	-5.47	8.47	**5**
Toluene	2.73	-0.61	3.30	**4**
1,2,4-Trichlorobenzene	4.02	-1.04	5.26	**4**
1,1,1-Trichloroethane (Methyl chloroform)	2.49	-0.75	2.64	**4**
Trichloroethene (Trichloroethylene)	2.42	-0.02	2.82	**4**
Trichlorofluoromethane	2.53	0.33	1.93	**4**
Trichlorotrifluoroethane	3.16	1.05	1.83	**4**
1,2,4-Trimethylbenzene	3.63	-0.52	4.23	**4**
1,2,3-Trimethylbenzene	3.66	-0.52	4.41	**4**

Compound	log (K_{ow})	log (K_{aw})	log (K_{oa})	Chemical Property Group
1,3,5-Trimethylbenzene	3.42	-0.52	3.87	**4**
Trimethylcyclohexenone	1.70	-2.56	5.27	**4**
2,2,5-Trimethylhexane	4.58	2.22	2.58	**4**
2,2,4-Trimethylpentane	4.09	2.10	2.00	**4**
n-Undecane	5.74	2.47	3.84	**4**
o-xylene	3.16	-0.56	3.83	**4**
mp-xylene	3.16	-0.56	3.83	**4**
BDE-153 (hexa BDE)	8.40	-4.40	12.80	**3**
Cyfluthrin	6.70	-6.80	13.50	**3**
Cypermethrin	6.60	-6.80	13.40	**3**
BDE-99 (penta BDE)	7.60	-3.90	11.50	**1**
Di(2-ethylhexyl) phthalate (DEHP)	7.70	-5.00	12.70	**3**
2,3,7,8-Tetrachlorodibenzo-p-dioxin	6.50	-3.80	10.30	**1**
Stearic acid	8.00	-3.30	11.30	**1**
Permethrin	7.20	-4.10	11.30	**1**
Perchloropentacyclodecane (mirex)	9.80	-1.00	10.80	**1**
BDE-47 (tetra BDE)	6.90	-3.40	10.30	**1**
Bisphenol A	4.10	-8.30	12.40	**3**
Benzo[a]pyrene	6.30	-3.90	10.20	**1**
Linoleic acid	6.90	-4.60	11.50	**3**
Piperonyl butoxide	4.80	-7.60	12.40	**3**
Butylbenzyl phthalate (BBzP)	4.90	-6.30	11.20	**3**
Di(2-ethylhexyl) adipate (DEHA)	8.00	-3.00	11.00	**1**
PCB 153	8.00	-2.70	10.70	**1**
p,p-DDT	6.80	-3.20	10.00	**1**
Mirex	0.10	-9.20	9.30	**5**
Triphenylphosphate	7.10	-2.70	9.80	**1**
Triclosan	5.50	-5.30	10.80	**3**
Dibutyl phthalate (DBP)	4.60	-4.90	9.50	**2**
Diazinon	4.70	-4.70	9.40	**2**
Chlorpyrifos	6.20	-3.20	9.40	**2**
Chlordane	6.70	-2.10	8.80	**1**
PCB 52	6.50	-2.30	8.80	**1**
Pyrene	5.10	-2.70	7.80	**2**

Compound	log (K_{ow})	log (K_{aw})	log (K_{oa})	Chemical Property Group
Galaxolide	4.80	-3.80	8.60	**2**
Pentachlorophenol (PCP)	4.90	-4.30	9.20	**2**
4-Nonylphenol	5.90	-3.50	9.40	**2**
EtFOSE	7.50	-0.90	8.40	**2**
Propoxur	2.20	-5.60	7.80	**5**
Butylated hydroxytoluene (BHT)	4.70	-3.80	8.50	**2**
Phenanthrene	4.50	-2.80	7.30	**5**
Methyl parathion	3.90	-4.30	8.20	**5**
MeFOSE	7.00	-1.10	8.10	**2**
Tris(chloropropyl) phosphate	5.90	-1.60	7.50	**2**
Texanol 2	2.40	-4.80	7.20	**5**
Geosmin	3.40	-3.60	7.00	**5**
Nicotine	2.00	-5.80	7.80	**5**
Caryophyllene	6.90	1.00	5.90	**4**
Pinonaldehyde	1.00	-5.20	6.20	**5**

CHAPTER 3. The role of ventilation and filtration in controlling exposures in commercial buildings

ABSTRACT

A wide range of air pollutants including volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), criteria air pollutants and radon are present in commercial buildings. We evaluate the effect of ventilation on indoor pollutant concentrations in commercial buildings using models and systematic evaluations of available studies. To study the impact of ventilation on ozone, radon, NO_2, and CO, we use a simple well-mixed room mass-balance model, along with data in the current literature. We used the Bennett and Furtaw (2004), fugacity-based model to simulate the impact of ventilation on indoor concentrations of VOCs and SVOCs. We find that minimum ventilation requirements in most commercial buildings should not be based on the requirements to limit exposures to ozone, NO_2, CO, and particles from outdoor air because outdoor air is the primary source of these pollutants. Data are too limited for conclusions about the importance of radon for minimum ventilation standards. However, in California, which is our focus area, radon is likely of second-order concern because radon levels are generally low. Ventilation is most effective at controlling indoor exposures to VOCs emitted from indoor sources that have low octanol-air partitioning coefficients [$\log(K_{oa})<9$]. With current ventilation and filtration system practices, increased ventilation is not as effective as filtration in controlling indoor concentrations of particulate matter from indoor sources or in controlling concentrations of SVOCs with high octanol-air partitioning coefficients that are attached to particulate matter. For these pollutants, removal by filtration and deposition usually dominates among all removal processes. The results of this work show that minimum ventilation requirements in most commercial buildings should be structured around controlling exposures to VOCs with $\log (K_{oa}) <9$, which have indoor sources and pose risks to human health and/or odor concerns.

INTRODUCTION

Building ventilation (the supply of outdoor air indoors) is employed to remove carbon dioxide exhaled by occupants and to limit the indoor air concentrations of pollutants emitted from indoor pollutant sources. However, the rates of ventilation are only one of several factors that affect the indoor pollutant concentrations. Other key factors are the strengths of the indoor pollutant sources, the rates of pollutant removal by air filtration systems, deposition on surfaces, chemical reactions, and the outdoor air pollutant concentration brought indoors by ventilation.

The minimum rates of ventilation for buildings are specified in standards. Historically, for commercial buildings these minimum rates were set to maintain 80% satisfaction with perceived air quality for occupants, or for visitors to a building at the time of entry (Yaglou et al. 1936; Berg-Munch et al. 1986). The need for sufficient ventilation to control indoor pollutants from unvented combustion sources, to maintain sufficient oxygen, to prevent dangerous levels of carbon dioxide (a human bioeffluent), and to control indoor humidity has also long been recognized, but maintaining 80% satisfaction with perceived air quality is the factor that set the limits on minimum ventilation rates.

The American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) provides a minimum ventilation standard for commercial buildings with two options (ASHRAE 2010). In the ventilation rate procedure, minimum ventilation rates are specified in tables for various types of spaces and air quality is assumed acceptable when this amount of ventilation is provided, even though other factors have a strong impact on indoor air quality. ASHRAE's guidance also provides an optional performance-based indoor air quality attainment procedure, which seeks to maintain indoor pollutant levels for contaminants of concern (identified by the user) below levels specified by a cognizant authority (selected by the user). In practice, the Indoor Air Quality Procedure has been of interest to those seeking to save energy by reducing ventilation system operation. The California Title 24 Standard also specifies minimum ventilation rates (California Energy Commission 2008) but includes no procedure analogous to ASHRAE's Indoor Air Quality Procedure. ASHRAE (2010) provides a list of contaminants and health guidelines in Tables B1, B2, and B3 of its Appendix B. However, there is little insight in the current literature that identifies indoor air pollutants whose concentrations are actually sensitive to variations in ventilation rates, and for which health impacts improve with increased ventilation.

In this chapter we start by identifying a set of contaminants measured in commercial buildings in the United States. We review literature and employ models to determine which of these contaminants have indoor concentrations that are substantially affected by ventilation rates, taking account of the location of source of the contaminant (indoors or outdoors), and the rates of pollutant removal by ventilation, filtration and deposition on surfaces. The goal is to first identify the key pollutants of interest for the setting of minimum ventilation rate standards in commercial buildings.

We use modeling and rely on available literature to draw conclusions about the impact of ventilation on pollutant concentrations indoors. For a set of criteria air pollutants and radon, we used a literature review to identify the sources and a simple mass balance model to predict the impact of ventilation rates on indoor concentrations. For volatile organic compounds (VOCs) and semi-volatile organic compounds (SVOCs), we analyze the impact of ventilation on indoor air concentrations as a function of basic chemical properties. Mass-balance models are used to assess the dependence of pollutant removal and occupant exposures on pollutant vapor pressure and the associated extent to which the pollutant resides in air, on airborne particles, and on indoor surfaces. We assume, based on prior research (Diamond et al. 2000), that indoor surfaces are coated with organic films and that an octanol-air partitioning coefficient K_{oa} can be used to characterize the relative amounts of VOCs in air, on particles, and on indoor surfaces. While there is no precise boundary between VOCs and SVOCs, in this document we refer to VOCs as compounds whose K_{oa}'s are ≤ 10, and SVOCs as compounds with $K_{oa} > 10$.

In order to understand the models presented below one needs a basic understanding of commercial building ventilation systems. Commercial buildings in the U.S. normally supply a mixture of outdoor air and re-circulated indoor air to the occupied spaces. The mixture normally passes through particle filters before entering these spaces. The particle filters are replaced every few months, thus, pollutants captured by the filters are removed from the building. To meet minimum ventilation requirements, some commercial buildings provide a fixed rate of mechanical ventilation that is typically 10% to 30% of the total amount of air supply to the

spaces (Turk et al. 1989), the rest of the supply is comprised of re-circulated air. Some commercial buildings temporarily increase the rate of mechanically supplied ventilation well above the minimum rate to reduce the need for air conditioning when the outdoor air is a source of free space cooling. Because ventilation standards specify minimum ventilation rates, the subsequent analyses apply to periods of minimum mechanical ventilation.

Uncontrolled air entry into buildings by leakage through the building envelope, called infiltration, can increase ventilation rates, and the air that enters buildings via air leakage does not pass through particle filters. The usual design intent for commercial buildings is to maintain the building slightly pressurized to eliminate infiltration, but infiltration rates remain substantial in many buildings (Price et al. 2007). In future energy efficient buildings, tighter envelopes and less infiltration are anticipated.

The minimum ventilation rates in standards are normally specified as a minimum outdoor airflow per person or per unit floor area, or a combination of these two parameters. However, for the mass balance modeling here we use ventilation rates per unit indoor air volume, often called air exchange rates or air changes per hour (ACH_i) with units of h^{-1}, because other parameters in the model are also typically normalized by indoor volumes. Available data on minimum ventilation rates in commercial buildings, are limited. Measured minimum ventilation rates are often on the order of 1 h^{-1} or less, with 0.4 to 1.6 h^{-1}encompassing most minimum ventilation rates (Turk et al. 1989; SMCB 2010; Persily and Grot 1985; Lagas Applied Technology 1995). The volume-normalized total rate of air supply to a space (filtered outdoor air plus filtered re-circulated indoor air) is another key parameter in our model. We use the variable name ACH_r for the filtered re-circulated indoor air flow rate.

METHODS

Selecting pollutants of concern

To select pollutants of potential concern, we reviewed literature on indoor air pollutant concentrations (see Table 2-3 for list of studies reviewed) in commercial buildings and compared the reported concentrations to guideline concentrations for acute and chronic health effects. We restricted our review to studies that were conducted in buildings in the United States. We also compared concentrations to odor thresholds and thresholds for sensory (irritation) effects. For pollutants with concentrations approaching the guidelines and thresholds, literature was reviewed to determine the primary location of the sources (indoors or outdoor air) and to determine what is known about the impact of ventilation rates on indoor concentrations. For details on the literature review refer to Chapter 2.

The resulting pollutants of concern in commercial buildings include a suite of VOCs, semi-volatile organic compounds (SVOCs), particulate biological contaminants, other particles, radon, heavy metals (mercury, lead, nickel, chromium, etc.) primarily in dust, and numerous inorganic gases (CO_2, CO, nitrogen oxides, sulfur oxides, O_3, radon).

In general, the sources of the inorganic gaseous pollutants (e.g., carbon monoxide, nitrogen dioxide, ozone) are small in most commercial buildings other than restaurants, with outdoor air as the primary source. The source of CO and nitrogen oxides is combustion, and without indoor cooking there is little or no unvented combustion in most commercial buildings, thus outdoor air

is usually the dominant source. While copy machines and laser printers can be sources of ozone, the outdoor air is usually the dominant source of ozone (Weschler 2006). The outdoor air and the people within a building are both important sources of carbon dioxide. There is evidence of loss of cognitive function in indoor spaces at levels of carbon dioxide found in typical office spaces (Allen et al. 2016; Satish et al. 2012). When outdoor air is the dominant pollutant source, increased ventilation will not diminish the indoor air pollutant concentration and may increase the indoor concentration. Consequently, minimum ventilation requirements in most commercial buildings will not be effective in altering the risks posed by CO, nitrogen oxides, or ozone.

VOCs, many with known or suspected adverse effects on occupants (EPA, 2011), are emitted by building materials, contents, and occupants, and can be reduced by ventilation as well as by source control and various air- or surface-cleaning strategies. For many VOCs, indoor concentrations in commercial buildings typically exceed outdoor air concentrations (Bennett et al 2011) because of the presence of indoor sources. Some VOCs such as benzene, toluene, ethyl benzene and xylenes (BTEX) also have significant outdoor sources (Edwards et al. 2001; Son et al. 2003). The indoor air research community has determined that increasing ventilation rates in buildings will reduce indoor concentrations of VOCs with strong indoor sources; however, the increase in ventilation can simultaneously increase the concentration of VOCs with strong outdoor sources. For many VOCs, indoor sources usually dominate. For these indoor-generated VOCs, ventilation is needed to reduce the risks of acute health hazards and chronic hazards such as reproductive toxicity and cancer and to maintain VOC concentrations below odor and sensory threshold levels.

Particulate matter (PM) is brought into commercial buildings from outdoor air and also comes from indoor sources such as electronic equipment emissions (Destaillats et al. 2008) and as secondary organic aerosol formation from a number of activities including the chemical reaction of ozone with VOCs emitted during cleaning (Destaillats al. 2006; Weschler et al. 1999). Particles emitted from indoor sources are likely to pose health risks to occupants. Much is known about the health impacts of outdoor air particles but, but with the exception of very high emissions from cook stoves, relatively little is known about the magnitude of the risks from indoor-generated particles beyond those containing allergens or in environmental tobacco smoke, which is now absent from most commercial buildings. Empirical data demonstrate that particle concentrations in commercial buildings with filtration and without indoor cooking or dentistry are generally lower than outdoor particle concentrations (Burton et al. 2000; Bennett et al. 2011). Per unit of particle removal, the typical cost of energy use associated with ventilation (Benne et al. 2009) will be higher than the cost of filtration (Fisk et al. 2002). Additionally, ventilation only helps control indoor concentrations of indoor-generated particles and may increase indoor concentrations of particles from outdoor air.

At this time, little is known about the air concentrations of SVOCs in commercial buildings. Apart from some widely studied classes of SVOCs such as flame retardants, phthalates and some pesticides, very few epidemiological studies exist on this class of predominantly bio-accumulating compounds. Evidence in the current literature suggests the major exposure pathways of many indoor SVOCs are non-dietary ingestion and dermal uptake, both active contact-driven and passive uptake from air (Weschler et al. 2008). Given the insensitivity of SVOCs concentrations to ventilation, we expect that ventilation rates will have a small impact on

total exposures. With classes of pollutants that raise some health concerns, for which only limited toxicological data are available, such as SVOCs, an understanding of the fate of pollutants indoors is needed to design studies to evaluate their health effects.

Another pollutant of interest, radon enters indoor environments by infiltration from the soil. It has a half-life of 3.8 days, and decays to produce radon progeny. The radon gas and the progeny can be removed from indoor environments by ventilation. However, the progeny have a high diffusivity, which causes them to attach to particles in the indoor environment. The PM is then removed by filtration. However, the largest removal process for radon progeny is radioactive decay since the progeny have a very short half-life of 3-4 minutes. Ventilation is expected to be the most dominant removal mechanism for radon gas. Our analysis is limited in that we do not account for radon progeny, but radon concentration is considered a good predictor of the health risk from radon progeny. Radon concentrations tend to be lower in California than in many other states (Cohen et al. 1984) and concentrations in commercial buildings are generally lower than concentrations in homes.

Carbon monoxide is a by-product of combustion, which can be found in indoor environments. The combustion processes that occur most commonly in indoor environments are cooking and heating. Most heating systems effectively vent combustion products to outdoors. Many commercial buildings other than restaurants have insignificant combustion-based cooking. Range hoods are employed in restaurants to limit the transport of CO and other cooking-based pollutants indoors, but these systems are imperfect and, in some buildings, a significant amount of CO may enter the indoor air leading to increases in indoor CO levels. Carbon monoxide is also classified a criteria air pollutant, on-road vehicles are strong outdoor sources of carbon monoxide. Levels of CO detected in commercial buildings (Apte et al. 2002) in the U.S. have not been found to exceed any health regulation, neither chronic nor acute.

We also assessed the impact of ventilation on nitrogen dioxide, which is also classified as a criteria pollutant by the U.S. EPA. Indoor sources of NO_2 are the same combustion processes that can be an indoor source of CO. Outdoor air can also serve as a significant source of NO_2 in the indoor environment and, given the general absence of indoor sources, outdoor air is the dominant source of NO_2 in most commercial buildings (Weschler et al. 1994). The primary removal mechanisms under consideration are ventilation, surface interaction and reactions in the indoor air.

The following sections elaborate on the mass-balance models used and the different scenarios modeled, which include varying the ventilation rate and filtration efficiencies.

Mass balance models

We used two different mass balance models (both representing the commercial buildings as a single well-mixed zone) to evaluate a range of pollutants and their behavior with respect to ventilation, chemical partitioning, transport and removal. The first model is a simple well-mixed mass-balance model, that we used to assess the impact of ventilation on ozone, radon, CO and NO_2. With this model we assume insignificant loss of these gaseous pollutants from air as it passes through particle filters, an assumption that may not be fully valid for ozone. The second model is a fugacity-based mass-balance model we used to evaluate the impact of ventilation on

VOCs and SVOCs and particles. Since limited data are available about the effect of ventilation on most pollutants in these classes of compounds, the fugacity-based model allows us to explore the effect of ventilation, based on the chemical properties of these compounds. The individual VOCs and SVOCs obtained from the review in Chapter 2 were modeled. The VOCs and SVOCs span a large range in the chemical property space. Since we grouped individual VOCs and SVOCs based on chemical properties, which affect their transport in the indoor environment, we present the results for each group of VOCs and SVOCs based on the results in Chapter 2.

The fugacity-based mass-balance model for VOCs and SVOCs also includes particles that enter into the building from the outdoors, of which some are filtered and removed from the indoor air. A schematic of the models with basic parameters are shown below. The models are also explained in detail in Appendix B. In the paragraphs below we first introduce the models and then provide the analysis used to set values for parameters used in the model.

Model 1: Well-mixed room model

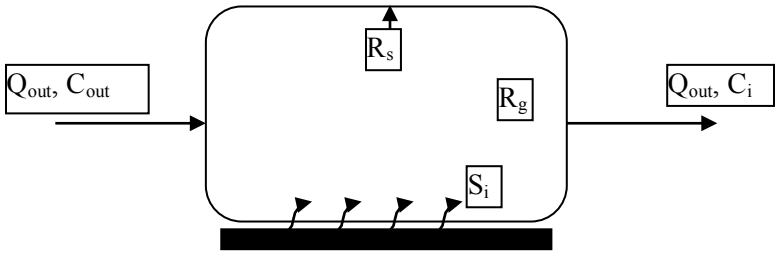

Figure 3-1: Schematic of the simple well-mixed room box model showing gains and losses.

The gains and losses and key parameters for our well-mixed room mass-balance model are shown in Figure 3-1. The differential equation describing this mass balance scheme is the following:

$$V_i \times \frac{dC_i}{dt} = C_{out} \times Q_{out} - C_i \times Q_{out} - R_s - R_g + S_i \quad (3\text{-}1)$$

Where,

V_i = volume of room (m^3)

C_i= concentration of pollutant in the room ($\mu g/m^3$)

C_{out} = concentration in inlet air ($\mu g/m^3$)

Q_i=Q_{out} flow rate of air, into and out of the room (m^3/h)

R_s = removal of pollutant by surface reactions ($\mu g/h$) = $C_i \times k_d \times V_i$

k_d = deposition rate constant (h^{-1}) = $v_d \times \frac{A_i}{V_i}$

v_d = deposition velocity (m/h)

A_i = surface area of the room (m^2)

R_g = removal of pollutant by gas-phase reactions or radioactive decay (μg/h)

k_r = Rate constant for removal by gas-phase reactions (h^{-1}), $= \frac{R_g}{M_g}$

S_i = indoor pollutant source strength (μg/h)

M_g = Mass of pollutant in room air (μg) = $C_i \times V_i$

The applicable first order rate constants are all calculated from data available in the literature for ozone, radon, NO_2 and CO. We present the first order rate constants for each pollutant in the results and discussion section. We also take into consideration whether the sources are predominantly indoors or outdoors.

Model 2: Fugacity-based mass balance model

We adapted the Bennett-Furtaw (2004) fugacity-based mass balance model for this study to assess the effectiveness of removal by ventilation of VOCs and SVOCs. The Bennett and Furtaw (2004) model accounts for indoor sources and transfers of chemicals from outdoor sources and for the partitioning of chemicals among the major indoor media--air, dust, and surfaces (carpets, vinyl floors, walls, and ceilings). In evaluating the performance of their model, Bennett and Furtaw (2004) found good comparison of their results with measurements of chlorpyrifos in air and carpets from an independent study of an indoor application of those chemicals as pesticides in a test house. The model has not been fully evaluated for suitability in modeling mass-balance in commercial buildings, but it is a useful tool to evaluate pollutants in indoor environments.

We provide the relevant equations and inputs used in the model, in Appendix B. We use parameters relevant to commercial buildings where data are available. We use an inventory-based approach with terms that account for the integral of contaminant mass flows or mass storage over time. We formulated the model to evaluate dynamic particle flows and then evaluate chemical flows simultaneously, while accounting for partitioning of chemicals to particles. The mass of chemical pollutant and particles in the compartments are treated as state variables. We assume a constant concentration of particles in outdoor air that enters the indoor environment through ventilation. The indoor concentration of particles is solved for using a mass balance that accounts for filtration of PM as they enter the indoor environments, deposition to surfaces and resuspension from surfaces. The model does not include an indoor source of particles and thus can only be applied to commercial buildings with a negligible indoor particle source.

We use two size bins for particles, instead of the six size bins used by Bennett and Furtaw (2004). The size bins are 10 μm to 2.5 μm and < 2.5 μm, and the assumed mass of outdoor air particles in each bin is provided in the Appendix. Current literature provides data only for these two size bins in commercial buildings, with very limited data on particle concentrations in other size bins. For the filtration system, we assume removal efficiencies equivalent to MERV 6, MERV 8 and MERV 13 filters. As filters are used their efficiency increases. Many existing buildings use lower efficiency filters; however, anecdotally there is a reported trend toward use of more efficient filters. The assumed deposition rate constants for particles in these two size ranges were 0.65 h^{-1} and 0.15 h^{-1} respectively (Siegel et al. 2013). The overall mass balance scheme is shown below in Figure 3-2. VOC removal by homogeneous reactions is assumed negligible. Although air entry by leakage is often significant, indoor sources usually dominate for the SVOCs that attach to particles. The time to equilibrium for gas-to particle partitioning of

SVOCs is relatively quick, on the order of minutes to an hour for most SVOCs. The time to equilibrium is also dependent on particle sizes, with smaller particles achieving equilibrium more quickly compared to larger particles (Weschler and Nazaroff 2008).

To summarize, while modeling the flow of VOCs and SVOCs, we incorporate particulate matter flows with only an outdoor particle source. As a separate exercise, we modeled particles from indoor sources only, neglecting the phase partitioning of particles with VOCs and SVOCs, solely to evaluate how indoor-generated PM concentrations are affected by changes in ventilation rates and filtration.

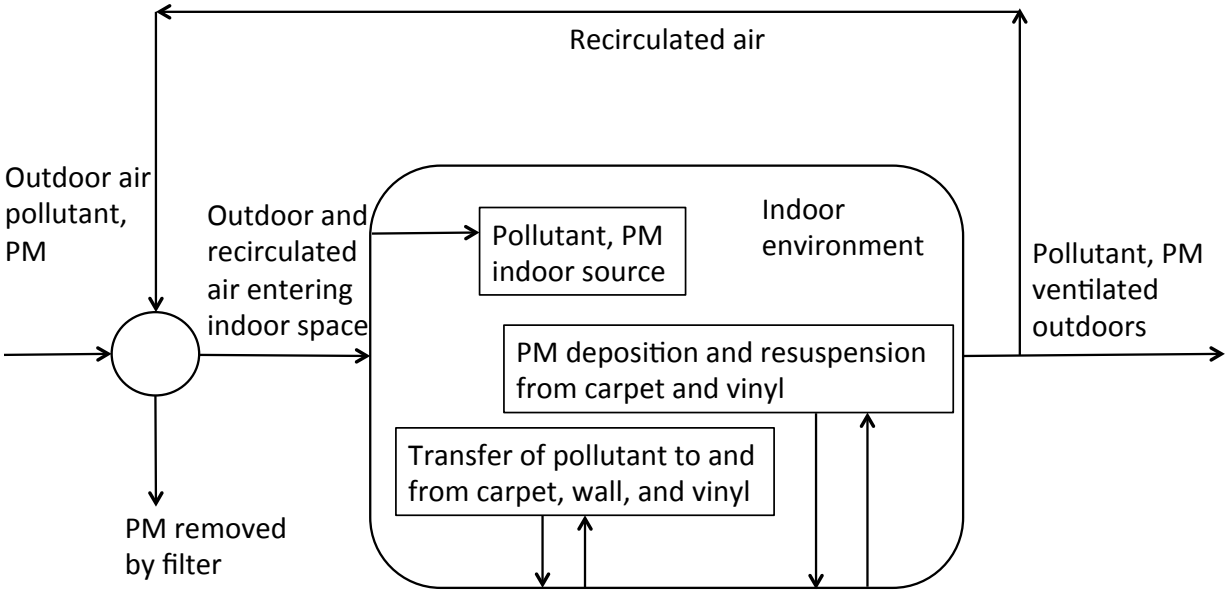

Figure 3-2: The schematic elements of the fugacity-based indoor mass balance model used to describe the movement and partitioning of indoor and outdoor pollutants in commercial buildings

In applying the schematic of Figure 3-2, we developed and solved the following governing differential equation describing the pollutant transport among major indoor compartments such as air, walls, and floors. The PM mass balance is shown in Equation 3-2, and the pollutant mass balance is shown in Equation 3-3. The transfer factors, and individual mass balance equations for chemical partitioning to and from, carpet, vinyl, walls and particles in air are described in Appendix B.

$$\frac{dM_{pm-a}}{dt} = ACH \times M_{pm-oa} + T_{r-va}M_{pm-v} + T_{r-ca}M_{pm-c} - ACH \times M_{pm-a} - T_d\, M_{pm-a} \quad (3\text{-}2)$$

$$\frac{dM_a}{dt} = ACH \times M_{out} + T_{va}M_v + T_{wa}M_w + T_{ca}M_c + M_{indoor-source} - ACH \times M_a - T_{av}M_a - T_{aw}M_a - T_{ac}M_a \quad (3\text{-}3)$$

where,
M_{pm-a} = mass of PM in room air (µg)

ACH = total ventilation rate (h^{-1})
M_{pm-oa} = mass of PM entering room from outdoors (µg)
T_{r-v} = resuspension rate of PM from vinyl (h^{-1})
M_{pm-v} = mass of PM on vinyl (µg)
T_{r-c} = resuspension rate of PM from carpet (h^{-1})
M_{pm-c} = mass of PM on carpet (µg)
T_d = deposition rate of PM onto carpet and vinyl (h^{-1})
M_a = mass of pollutant in room air (µg)
M_{out} = mass of pollutant entering room from outdoors (µg)
T_{va} = transfer factor of pollutant from vinyl to air (h^{-1})
M_v = mass of pollutant in vinyl (µg)
T_{wa} = transfer factor of pollutant from wall to air (h^{-1})
M_w = mass of pollutant in wall (µg)
T_{ca} = transfer factor of pollutant from carpet to air (h^{-1})
M_c = mass of pollutant in wall (µg)
$M_{indoor-source}$ = Indoor source of pollutant (µg/h)
T_{av} = transfer factor of pollutant from air to vinyl (h^{-1})
T_{aw} = transfer factor of pollutant from air to wall (h^{-1})
T_{ac} = transfer factor of pollutant from air to carpet (h^{-1})

We use the ratio defined in the following equation to quantify the effectiveness of removal of the pollutant by ventilation. In this equation all the terms are inventories, i.e., integrals of mass flows over time, as described above, for a full year of building operation. We start with clean rooms initially, and let the model run for 1 year, which is the time the system takes to reach a quasi steady state

% Total mass removed by ventilation:

$$\%\, V_{rem} = 100 \times \frac{M_{out}}{M_c + M_w + M_v + M_{filter} + M_{out}} \quad (3\text{-}4)$$

M_{filter} = Total mass of chemical attached to particles, removed by the filter (µg)
M_{out} = Total mass of chemical removed by air leaving the building (µg)

Building parameters
To evaluate the effect of ventilation on indoor concentrations of VOCs and SVOCs, we ran the model for ~150 pollutants found in commercial buildings (Table A1). The models were run for 4 types of buildings--offices, schools, retail and grocery stores. Data from over 200 individual buildings, comprising offices, schools, retail stores and grocery stores were aggregated. We used these data to find a representative building of each type. The following parameters were used: air change rate (ACH_i), area (A_i), indoor and outdoor concentrations of $PM_{2.5}$ and PM_{10}. The data were used to identify a typical building for each representative building type. As explained below, we then assigned a building evaluation score and picked the following parameters for a building, which performed close to median for the weighted score.

The parameters were initially ranked individually for each building, the individual rankings were then weighted and combined to find the most representative building for each class. This approach to find an average building is used, rather than finding the median of each parameter independently in all the buildings, to construct a hypothetical representative building (Table B1).

The following formula was used to combine the rankings for the various parameters.

$$\text{Score} = 0.05 \times R_{\text{Area}} + 0.15 \times R_{\text{ACH}} + 0.20 \times R_{\text{PM2.5in}} + 0.20 \times R_{\text{PM2.5out}} + 0.20 \times R_{\text{PM10in}} + 0.20 \times R_{\text{PM10out}} \quad (3\text{-}5)$$

The R value for each parameter was calculated as follows

$$R = \begin{cases} \dfrac{\text{Rank}}{\text{Median rank}}, & \text{if Rank} \leq \text{Median Rank} \\ \dfrac{\text{Median rank}}{\text{Rank}}, & \text{if Rank} > \text{Median Rank} \end{cases}$$

The individual score for each building was calculated and the parameters from buildings that are closest to the median in score are used. The following parameters were obtained for each building type: ACH_i, ACH_r, Area, Height, PM_{10} outdoor concentrations, $PM_{2.5}$ outdoor concentrations

The models were run under the conditions listed in Table 3-1, with varying filtration efficiencies and ventilation rates

Table 3-1: Model Scenarios

Scenario	Filter efficiency (η)	VR	VOC/PM	VOC/PM
S1	MERV 6	VR	Outdoor/Outdoor	Indoor/Outdoor
S2		VR/2	Outdoor/Outdoor	Indoor/Outdoor
S3		VRx2	Outdoor/Outdoor	Indoor/Outdoor
S4	MERV 8	VR	Outdoor/Outdoor	Indoor/Outdoor
S5		VR/2	Outdoor/Outdoor	Indoor/Outdoor
S6		VRx2	Outdoor/Outdoor	Indoor/Outdoor
S7	MERV 13	VR	Outdoor/Outdoor	Indoor/Outdoor
S8		VR/2	Outdoor/Outdoor	Indoor/Outdoor
S9		VRx2	Outdoor/Outdoor	Indoor/Outdoor

The concentrations of pollutants (C_{air}, $\mu g/m^3$) in the room were also evaluated to assess the reduction in exposures at various ventilation rates and source locations. For particulate matter, we use the following equations to assess the effectiveness of removal by ventilation and filtration. The numerators of these equations equal the total mass of particles removed by ventilation and filtration, respectively, for the simulation period. The denominator, which is the same for both equations, is the total removal of particles from the space by all processes.

Particulate mass removed by ventilation, fraction:

$$PM_v = \frac{M_{pm-out}}{M_{pm-out}+M_{pm-filter}+M_{pm-d}} \quad (3\text{-}6)$$

Particulate mass removed by filter, fraction:

$$PM_f = 100 \times \frac{M_{pm-filter}}{M_{pm-out}+M_{pm-filter}+M_{pm-d}} \quad (3\text{-}7)$$

Where,

M_{pm-out}= Total mass of PM removed by air leaving the room (μg)

$M_{pm-filter}$ = Total mass of PM removed by filtration (μg)

M_{pm-d}= Total mass of PM which settles on carpet and vinyl (μg)

Exposure metrics

We employed the dimensionless exposure metric intake fraction (Bennett et al. 2002) using the following equations to assess the effectiveness of ventilation in reducing exposure to a pollutant from an indoor source.

The normalized concentration was multiplied by breathing rates to obtain a dimensionless exposure metric for an individual (individual intake fraction, iF):

$$iF = C_{nor} \times BR \quad (3\text{-}8)$$

Where,

BR = Average daily breathing rate of an adult (m^3/h)

C_{nor} = Concentration of pollutant in air normalized to source strength (h/m^3)

Normalizing the concentration to source strength

$$C_{nor} = \frac{C_T}{EF} \quad (3\text{-}9)$$

Where,

C_T = Total pollutant concentration in indoor air, under quasi-steady state conditions (μg/m^3)

EF = Emission rate of pollutant from the source (μg/h)

In this situation, the intake fraction is the fraction of pollutant emitted from the source that is inhaled. The intake fraction is proportional to the time-averaged pollutant concentration in the indoor air.

When outdoor air is the pollutant source, we used the following dimensionless exposure metric, concentration ratio. It is important to note that concentration ratio is a metric of exposure rather than of intake in contrast to intake fraction.

$$CR = \frac{C_T}{C_{out}} \quad (3\text{-}10)$$

Where,

CR = Concentration ratio, dimensionless

C_{out} = Total pollutant concentration in outdoor air (μg/m^3)

We use the similarly calculated dimensionless exposure metric, indoor proportion of outdoor particles (Riley et al. 2002) when outdoor air is the primary source for PM

$$\mathrm{iPOP} = \frac{C_T}{C_{out}} \tag{3-11}$$

where,
iPOP = Indoor proportion of outdoor particles

Sensitivity analysis
We carried out a detailed sensitivity analysis of the fugacity model to provide an approximate measure of the importance of each input parameter with respect variability and/or uncertainty. Out approach is based on methods described by Morgan and Henrion (1992), who propose a factorial design where the response of the model outcome is compared to stepwise small changes in each of the model inputs. For this we ran the models using fixed values of the primary parameters of importance in this study: VP, K_{oa}, outdoor air supply fraction, and total ventilation rates. All other input parameters were varied and the sensitivity of the output to the input was assessed. We assumed that these are inputs with the most sensitivity but needed to assess the importance of all parameters we considered of secondary sensitivity. Each of the other secondary parameters listed in Table B2 was then subject to a 1% change, sequentially. The maximum change in output (fraction of pollutant removed by ventilation), with respect to a change in the input was tallied. With this process we found the output metric was not notably sensitive to variations of any of the input parameters considered of secondary importance. In addition, to evaluate the uncertainty importance of each input, we calculated the normalized-standard-deviation-weighted sensitivity ratio. This sensitivity metric is calculated as follows. After we evaluate the sensitivity of the parameter, x, by subjecting x to a 1% change and observing the change in y we use the following formula to assess the uncertainty importance:

$$\mathrm{SVF} = \left(\frac{\frac{\Delta y}{y}}{\frac{\Delta x}{x}}\right) \times \mathrm{CV} \tag{3-11}$$

where,
SVF = sensitivity variation factor
CV = coefficient of variation of the parameter x, which is the standard deviation divided by the mean value, and reflects its normalized uncertainty (variability).

RESULTS AND DISCUSSION

In the following section we present the results for two categories of substances. The first section details the results for chemicals whose indoor concentrations can be modeled by using a simple indoor air mass balance model. We present the results for ozone, nitrogen dioxide, carbon monoxide, and radon in the first section. The second section details the results for the compounds which are modeled using a fugacity model. We present the results for PM, VOCs and SVOCs in the second section.

Results of indoor air modeling for substances with no surface partitioning

The simple well-mixed room model is used to model the pollutant mass balance for the following compounds: ozone, carbon monoxide, nitrogen dioxide and radon. We present the results for each compound separately since they have very different parameters that determine the transport and concentration of chemicals indoors.

Ozone

Even though ozone reacts with some airborne pollutants such as with d-limonene, nitric oxide and hydrogen sulfide, the concentrations of the reactants indoors may not be high enough to cause significant ozone removal (Weschler et al. 2000); consequently, ozone removal from reactions on surfaces dominate relative to reactions within air. From Table 3-2 we conclude that surface reactions will usually be a larger removal mechanism of ozone indoors than ventilation. Ventilation can still significantly impact the concentration. Because the outdoor air is normally the source of ozone, indoor ozone concentrations will be higher at a higher ventilation rate. Ozone might also be unintentionally removed by reactions with contaminants on particle filters (Destaillats et al. 2011) and the simple well-mixed room model does not account for this removal method. From Figure 3-3, we can see that the indoor ozone concentrations remain well below outdoor concentrations at low ventilation rates, and approach anywhere between 35%-80% of outdoor concentrations when the ventilation rates are high. Under conditions of minimum ventilation, with the ventilation rate typically in the range of 0.4 to 1.6 h^{-1}, indoor ozone concentrations will remain well below outdoor concentrations.

Table 3-2: Ozone first order deposition rate constants

Ozone		Source
ACH (A_i)	0.4 - 1.6 h^{-1}	Turk et al. (1989),SMCB (2010), Persily and Grot (1985), Lagas Applied Technology (1995), Weschler et al. (2000)
v_d	0.015 - 0.075 cm/s	
A_i/V_i	3 - 4 m^{-1}	
$k_d = v_d \times A_i/V_i$ (h^{-1})	1.6 - 8.1, 2.2 - 10.8	

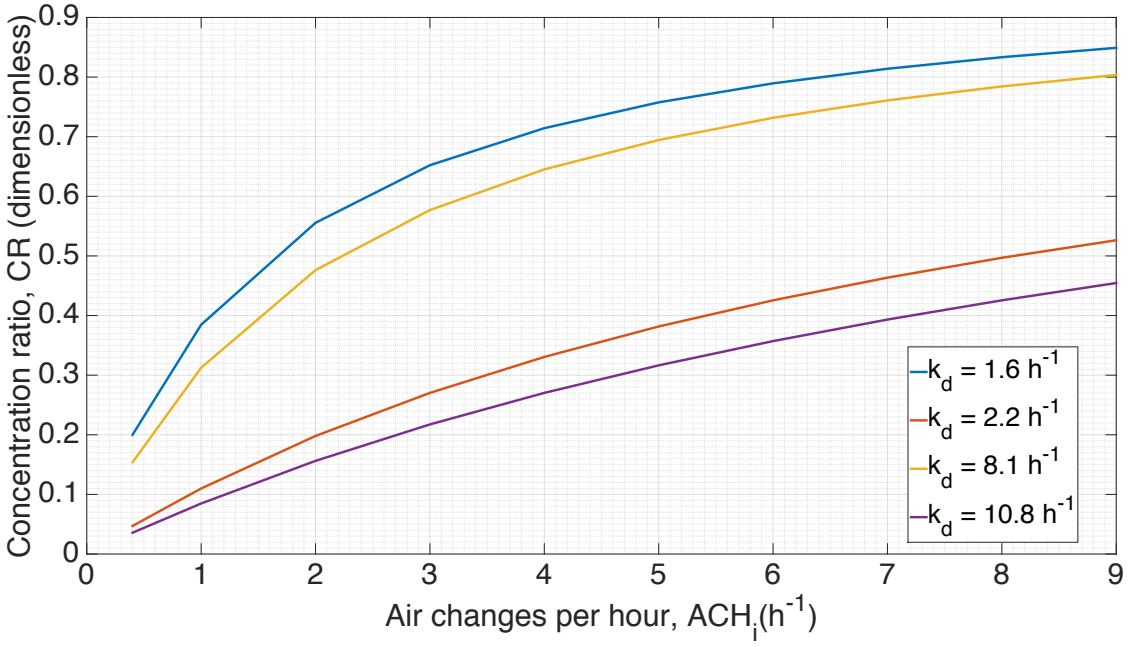

Figure 3-3: Plot of ozone indoor-outdoor concentration ratios vs. varying air changes per hour (ACH_i), at different ozone deposition velocities

Because indoor ozone concentrations will generally increase, not decrease, with ventilation rate, and because the impact of ventilation rate on ozone is reduced by depositional losses, other pollutants will determine the minimum amount of ventilation needed in commercial buildings to protect human health from O_3 exposures. The risks associated with increases in indoor O_3 concentration with ventilation rate could be a factor that places an upper limit on minimum suggested rates of ventilation.

Carbon monoxide

CO has indoor and outdoor sources and it is essentially an inert gas (Spengler et al. 2000), so R_s and R_g drop out of Equation 3-1. Ventilation is the dominant removal method for CO, since all other removal mechanisms do not play a measureable role in CO removal. However, when indoor CO sources are absent, outdoor air is the CO source so the primary role of ventilation is to cause a time lag between the outdoor and indoor CO concentration. Where indoor CO sources are absent, which will be the case for most commercial buildings, minimum ventilation recommendations will not be determined by the need to control indoor CO.

Nitrogen dioxide

The primary removal mechanisms reported for NO_2 are ventilation and surface interaction (see Table 3-3). The resulting mass balance causes S_i to drop out of Equation 3-1. Table 3-3 provides an estimate of the first order deposition removal rate for NO_2.

Table 3-3: NO_2 first order rate constants

NO_2 rate constants			Source
ACH	0.4 - 1.6	h^{-1}	Weschler et al. (2000)
k_d	~0.8	h^{-1}	Nazaroff et al. (1992)

The deposition velocities available in the literature were used to calculate the first order rate constant for surface reactions. The first order interaction term indicates that surface reactions compete with ventilation as a dominant removal mechanism. Because outdoor air is normally the dominant NO_2 source, increased ventilation will increase indoor NO_2 concentrations, but the impact of ventilation is dampened by the removal of NO_2 by surfaces. From Figure 3-4, we can see that indoor NO_2 concentrations are substantially lower than outdoor concentration at the minimum ventilation rates typically found in commercial buildings. At very high ventilation rates the indoor concentration approaches the outdoor concentration. In commercial buildings, which typically lack significant indoor sources of NO_2, other pollutants will determine the minimum amount of ventilation needed for health. The risks associated with increases in indoor NO_2 concentration with ventilation rate could be a factor that places an upper limit on minimum suggested rates of ventilation.

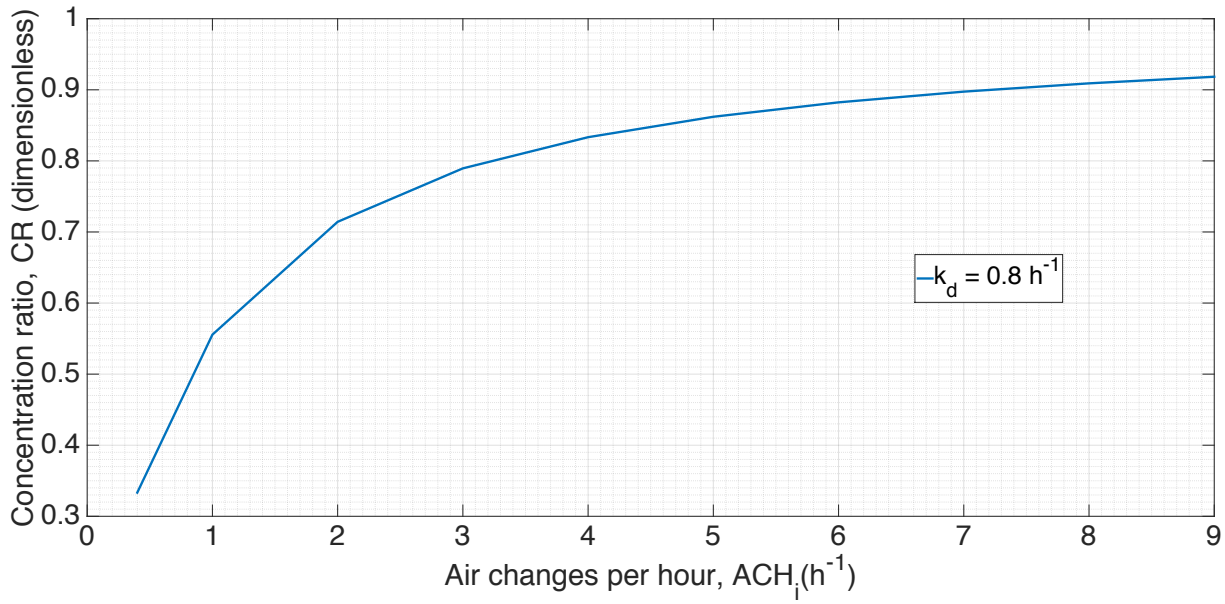

Figure 3-4: Plot of NO_2 indoor-outdoor concentration ratios vs. varying air changes per hour (ACH_i) for a building with no indoor nitrogen dioxide source.

Radon

Radon enters indoor air primarily by infiltration from the soil below structures and outdoor air is also a source. Equation 3-1 applies for radon, with R_s set equal to zero and R_g equal to the loss rate of radon by radioactive decay, which is 0.005 h^{-1}. Based on the parameters for the mass balance model (see Table 3-4), ventilation is the dominant removal mechanism for radon indoors. We are unable to predict how radon concentrations in commercial buildings vary with ventilation rates because of an absence of data on radon source strengths in commercial

buildings, particularly commercial buildings in California. In homes, the source strength variations can completely dominate removal by ventilation, high rates of radon entry into a building from the soil can lead to high indoor concentrations even at typical ventilation rates (Nazaroff et al. 1983). However, as mentioned earlier, radon levels in California are generally lower than in the rest of the nation. To the extent that outdoor air contributes radon indoors, the indoor exposures from outdoors will be largely unaffected by ventilation rates.

Table 3-4: Radon first order rate constants

Radon			Source
C_o	400	pCi/m^3	
Ventilation rate	0.4 - 1.6	h^{-1}	Weschler et al. (2000)
S_i	50 – 750	$(pCi/m^3\text{-}h)$	
Decay rate	0.005	h^{-1}	

Results of mass balance model – Particles

We performed separate modeling for particles from outdoor and indoor sources only. The modeling was conducted to evaluate iF and iPOP, which are used to assess how exposure to PM is affected by ventilation and filtration in commercial buildings.

The resulting estimates of how PM is partitioned between filtration, ventilation, deposition to surfaces and cleaning for varying ventilation rates and filtration efficiencies is shown in Figure 3-5 for outdoor-generated PM and in Figure 3-6 for indoor-generated PM. The total particle mass removed is obtained by a simple sum of the masses of particles in each size class. Filtration is the dominant removal mechanism for outdoor-air particles (Figure 3-5), indicating that, with the filter efficiencies and air recirculation rates assumed, changing ventilation rates have a small impact on indoor concentrations of PM from outdoor sources. See Table B2 in Appendix B for model parameter specifications, such as deposition and resuspension velocities, filtration efficiencies. Filtration is the dominant removal mechanism for outdoor-generated particles across the scenarios in all types of buildings. For indoor generated PM, filtration is a less dominant removal pathway compared to PM from outdoor air since PM from outdoor air is filtered before it enters the indoor space. This trend is seen in all building types, offices, retail, grocery and schools. In scenarios with low filtration efficiency (MERV 6 filter in S1, S2, S3) in office buildings ventilation is the dominant loss mechanism removing 35–45% of PM. Filtration only removes 29–42% of the PM in these scenarios. However, as the filtration efficiency increases, filtration becomes the dominant removal pathway removing 60 – 75% of PM mass. In comparison ventilation removes about 15 – 40% of PM mass.

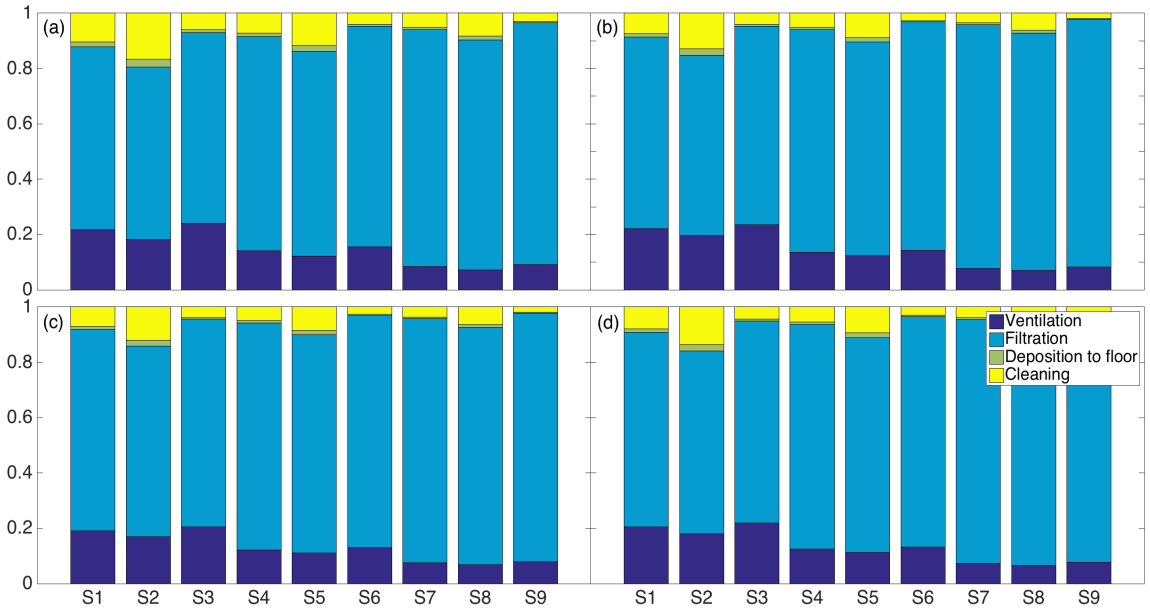

Figure 3-5: Removal pathways of outdoor generated PM_{10} and $PM_{2.5}$ combined, in (a) Office buildings (b) Retail stores (c) Grocery Stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis)

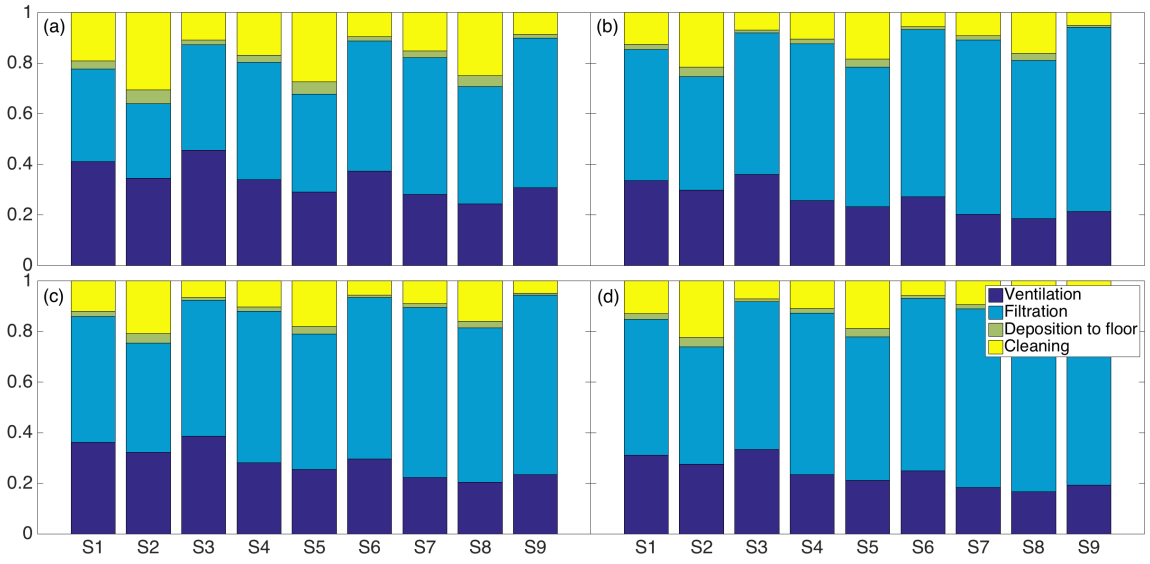

Figure 3-6: Removal pathways of indoor generated PM_{10} and $PM_{2.5}$ combined, in (a) Office buildings (b) Retail stores (c) Grocery Stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis)

For outdoor-generated PM even in scenarios with low filtration efficiency (MERV 6 in S1, S2, S3), filtration is the dominant removal pathway contributing some 62–70% removal of PM from offices, retail stores, grocery stores and schools. Analogously, ventilation from indoor to outdoor air removes about 17–23% of the outdoor-generated PM mass enters the ventilation system from outdoors. With a moderate efficiency filter (MERV 8 in S4, S5, S6) filtration removes as much as 73–84% of the entering PM and ventilation from indoors to outdoors removes 11-16% of PM.

As ventilation rates (VRs) are decreased (S2, S5, S8) the removal by deposition and cleaning increases. Since the residence time of PM in the indoor space increases contributing to more mass of PM being deposited on surfaces and subsequently removed by cleaning. In these scenarios, the PM loads reside longer in the indoor environment increasing the potential for removal by these pathways. As the VRs are increased (S3, S6, S9), the removal percentage of PM by ventilation slightly increases. In addition, we note that improving filtration efficiency (MERV 8 in S4, S5, S6 and MERV 13 in S7, S8, S9) has more impact on PM removal compared to other scenarios. We also see from Figure 3-5 (indoor PM originating from outdoors) and Figure 3-6 (indoor-generated PM) in Scenarios S7, S8 and S9, which have a high efficiency filter (MERV 13), changes in VRs do not have a high impact on the amount of PM removed by various pathways as filtration is the dominant removal pathway. The percentage contributions of other removal pathways (cleaning and deposition to indoor surfaces) for PM of indoor and outdoor origins are similar. Ventilation is modestly effective at removing indoor-generated particles at the low fractions of outdoor makeup air encountered during periods of minimum building VRs. The larger particles (PM_{10}) are most effectively removed by filtration and deposition and smaller particles ($PM_{2.5}$) are removed effectively by filtration and ventilation.

Next, we analyze the effect of changing ventilation and filtration on the exposure metrics iPOP for PM of outdoor origin and iF for PM of indoor origin. The iPOP for PM calculated in all the scenarios is shown in Figure 3-7. The iPOP decreases when filtration efficiency is improved. The iPOP ranges from 0.5 to 0.6 at low filter efficiency, 0.3 to 0.4 at medium filter efficiency and 0.15 to 0.2 at high filter efficiency. We also see that the iPOP values are similar in the various building types; and that iPOP increases as the VR increases indicating that for particles with outdoor sources, bringing in more outdoor air increases occupant exposure (Figure 3-7). However the changes associated with iPOP as the VR changes are smaller compared to the changes associated with improved filtration efficiency. This result is well aligned with previous work by Fisk et al. (2002), where filtration was identified as an action leading to significant and low-cost reductions in particulate matter concentrations. If we assume no particle filtration or very low efficiency filtration, increased ventilation will remove indoor-generated particles from the indoor air but bring in outdoor-air particles. For a building with moderate (MERV8 in S4, S5, S6) or high efficiency particle filtration (MERV13 in S7, S8, S9), VR will have a small impact on indoor concentrations of particles because particle removal by filtration dominates relative to particle removal by ventilation.

Figure 3-8 shows how iF for indoor-generated particles varies with VR. For indoor-generated particles, iF is proportional to average indoor air concentration. This figure shows that ventilation rate has a modest impact on iF when we use assumed values of air filter efficiencies and air recirculation rates. The iF is seen to decrease when filtration efficiency increases. The iF shows a larger variation between different types of buildings, ranging between 800 to 1000 ppm in offices, 80 to 100 ppm in retail stores, 400 to 3000 ppm in grocery stores and 5 to 8 ppm in school buildings for PM_{10}. For $PM_{2.5}$, iF ranges between 100 to 300 ppm in offices, 100 to 300 ppm in retail stores, 5000 to 10000 ppm in grocery stores and 80 to 100 ppm in school buildings. The iF does not vary significantly when only VR is changed while keeping filtration efficiency constant. However, iF changes significantly when filter efficiency is varied. As particle removal decreases with diminished VR, the filtration system compensates by removing more particles.

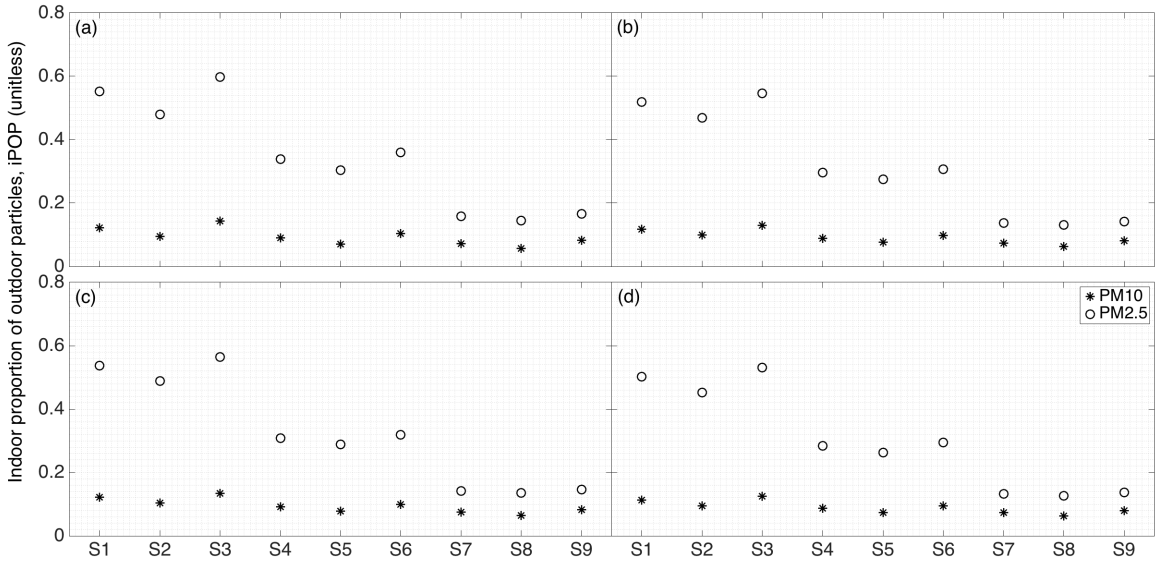

Figure 3-7: iPOP for PM_{10} and $PM_{2.5}$ of outdoor origin, in (a) Office buildings (b) Retail stores (c) Grocery stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis)

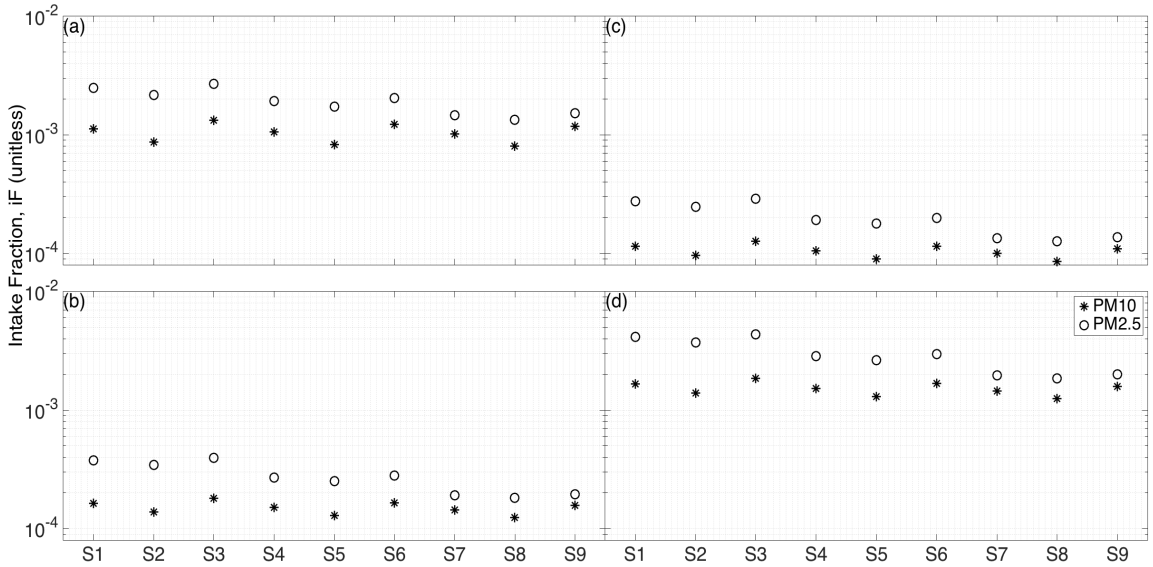

Figure 3-8: iF for PM_{10} and $PM_{2.5}$ of indoor origin, in (a) Office buildings (b) Retail stores (c) Grocery stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis)

Results of fugacity based mass balance model – VOCs and SVOCs

The batch of model parameterizations described in the Methods section provided the tools for evaluating the behavior of VOCs and SVOCs found in commercial buildings. Separate model runs applied to indoor and outdoor sources of VOCs and SVOCs--each with an outdoor PM source. Because of the preferential partitioning of SVOCs on PM, the results from modeling the PM flows were needed to evaluate how SVOC concentrations vary. For modeling VOCs and SVOCs, only PM from outdoor air were included in the model.

We also varied the total ventilation rate and the filtration efficiencies (η) independently. The combinations of parameters used in the model evaluation process are listed in Appendix B, and the various modeling scenarios are listed in in Table 3-1. The models were run to simulate 1 year of building operation. This produces results that provide insight into how the system behaves in quasi-equilibrium that is effectively at steady state. Each VOC and SVOC was assumed to exist in air both in the gas and particle-bound phases, the total air concentration in both phases is used to calculate the removal by all pathways. For particles and SVOCs that sorb to particles, the fraction of outdoor air in the supply airstream, which contains a mixture of outdoor air and re-circulated indoor air, becomes a critical parameter. At typical outdoor air conditions, the fraction of outdoor make-up air is typically between 10 – 40% of total air intake. In the model we maintain the fraction of re-circulated air constant across all scenarios at ~70% for all the buildings.

For VOCs and SVOCs originating from outdoor air Figures 3-9 and 3-11 show the model estimates of VOC and SVOC removal due to ventilation. For VOCs and SVOCs with an outdoor source only, ventilation is seen to completely dominate the removal for highly volatile organic compounds (Groups 3, 4 and 5) although ventilation is also the pollutant source (Figure 3-9). As the K_{oa} increases and VP decreases, going from Group 5 to 1, we see that ventilation provides a smaller fraction of pollutant removal. This is the case because, as K_{oa} increases, more of the pollutant mass is attached to particles, which are removed by filtration. As the outdoor air in the supply airstream increases, a larger portion of the high-K_{oa} pollutants are removed by ventilation.

For VOCs and SVOCs that have only an indoor source, Figure 3-11 shows the predicted percent of VOC and SVOC removal by ventilation as a function of the fraction of outdoor air in the supply airstream. As expected, when only indoor VOC sources are present the percent of the pollutant removed by ventilation increases with amount of outdoor air in the supply airstream. With a low value of K_{oa} (Group 3, 4 and 5) indicating that little of the airborne VOC is attached to particles, nearly all pollutant removal is by ventilation. When VR is low and the K_{oa} (Group 1 and 2) is high (implying that most of the airborne SVOC is attached to particles), less than half of pollutant removal will occur by ventilation. However, for SVOCs with a moderate to high K_{oa}, the impact of ventilation rate on airborne concentrations is not readily determined, because as SVOC removal by ventilation diminishes, SVOC removal by filtration increases—making the overall removal dependent on the variable parameters of the filtration system. For the high K_{oa} compounds, other pathways such as uptake by and deposition to surfaces (carpets, walls, vinyl), accounted for less than 10% of all removal processes.

Figures 3-10 and 3-12 show the estimated impact of the amount of outdoor makeup air on exposure metrics of VOCs and SVOCs, for outdoor-air- and indoor-air-originating sources, respectively. Regardless of the location of the source, for SVOCs with a high K_{oa} (Groups 1 and 2) the amount of outdoor makeup air has little impact on exposure as indicated by the intake fraction and concentration ratio. For these high-K_{oa} pollutants, filtration is the dominant pollutant removal mechanism because most of the chemical is attached to particles. Where there are only outdoor sources, there is an increase in the indoor-to-outdoor concentration ratio as the fraction of outdoor air entering increases (Figure 3-10). In general the concentration ratios are seen to increase as the amount of outdoor air entering the building increases. For K_{oa} <9 (Groups 3, 4,

and 5), increases in VR have little impact on the indoor-to-outdoor concentration ratio, and hence little impact on exposures. With values of K_{oa} in the range 9 to 11, the outdoor air fraction has a significant impact on the concentration ratio. However, most of the benefit of increased outdoor air makeup fraction occurs as the outdoor air fraction increases from 0.05 to 0.1, and measured outdoor-air makeup fractions are usually greater than 0.1. For K_{oa} >11 (Groups 1 and 2), we see an increase in the concentration ratio. Since most of the SVOCs are attached to particles, they are removed by filtration when entering the building through the filters, or when the air is re-circulated. The concentration ratios approach ~0.4 because, filtration removes approximately 60% of the particles and consequently removes 60% of the SVOCs entering from outdoors.

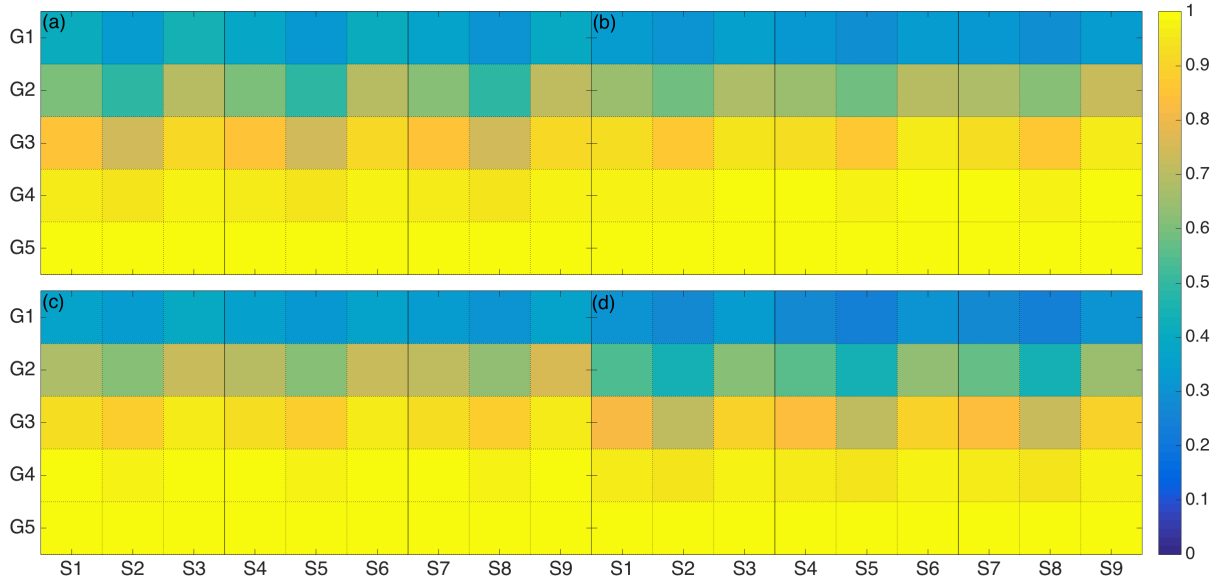

Figure 3-9: Fraction of volatile and semivolatile organic pollutants of outdoor origin, removed by ventilation to outdoor air in (a) Office buildings (b) Retail stores (c) Grocery stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis) and Table 2-2B for details on chemicals in Groups G1, G2, G3, G4 and G5 (y-axis)

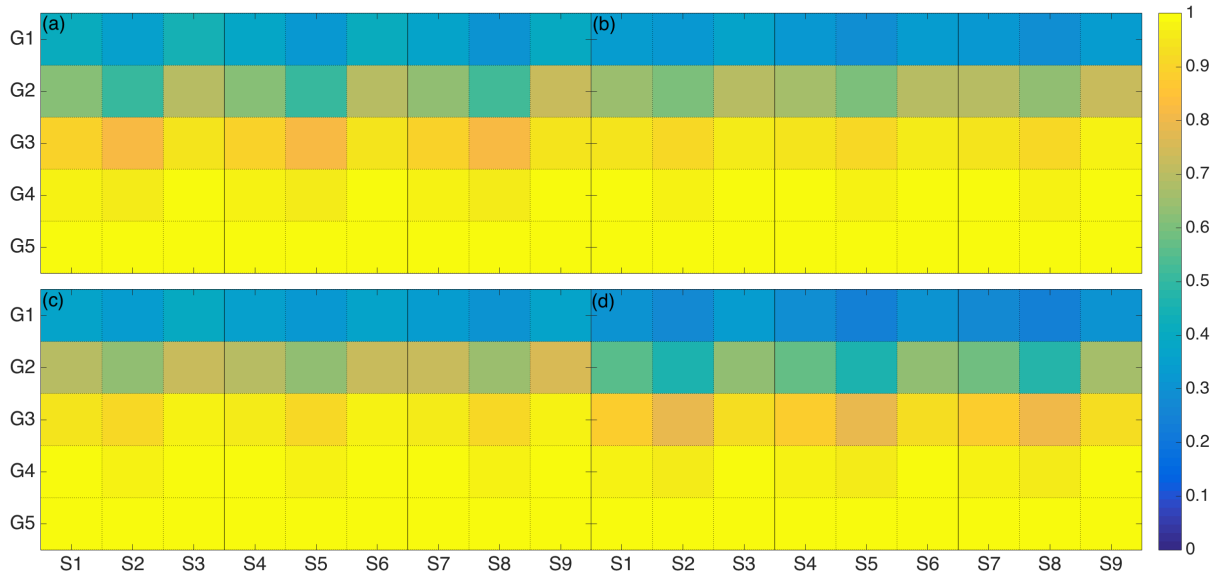

Figure 3-10: Concentration ratio (CR) for volatile and semivolatile organic pollutants of outdoor origin, in (a) Office buildings (b) Retail stores (c) Grocery stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis) and Table 2-2B for details on chemicals in Groups G1, G2, G3, G4 and G5 (y-axis)

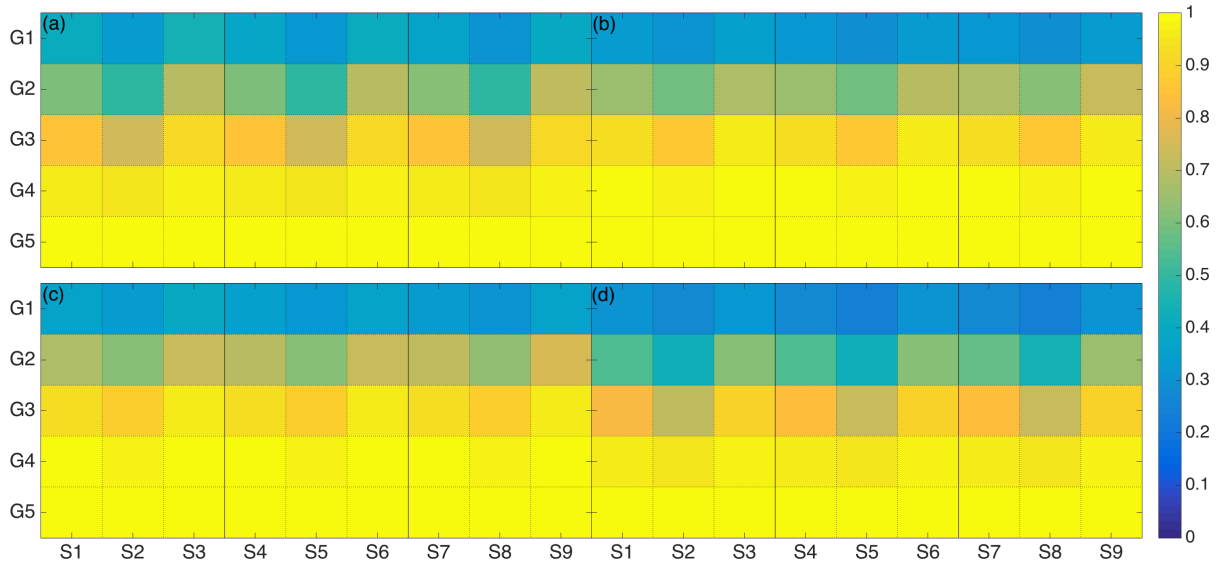

Figure 3-11: Fraction of volatile and semivolatile organic pollutants of indoor origin, removed by ventilation to outdoor air, in (a) Office buildings (b) Retail stores (c) Grocery stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis) and Table 2-2B for details on chemicals in Groups G1, G2, G3, G4, and G5 (y-axis)

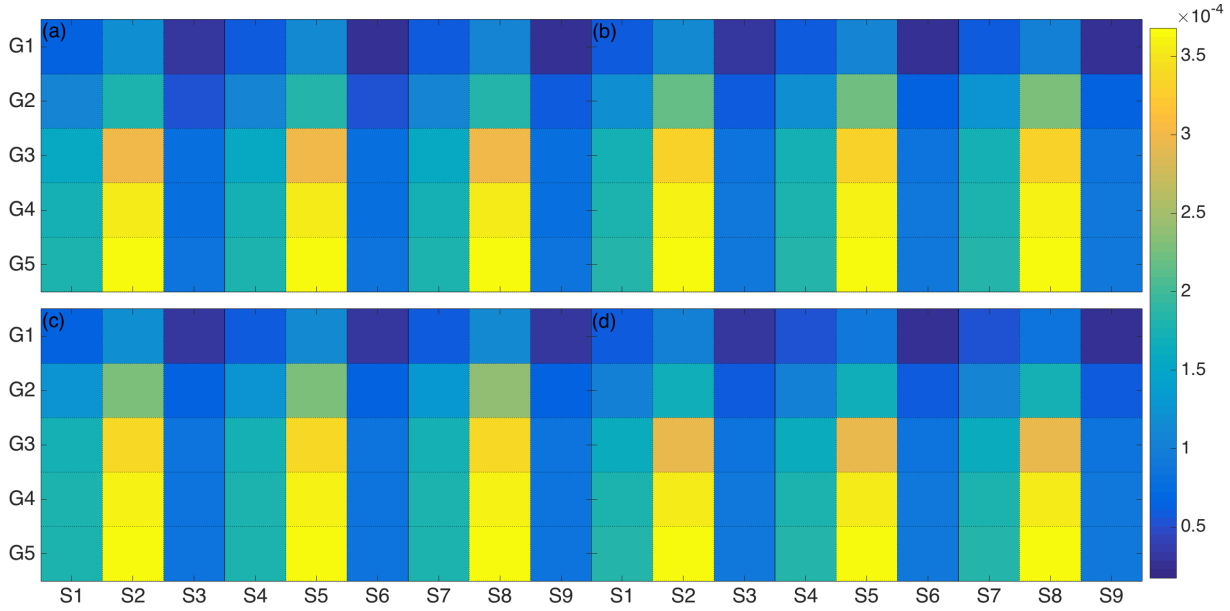

Figure 3-12: Intake fraction (iF) for volatile and semivolatile organic pollutants of indoor origin, in (a) Office buildings (b) Retail stores (c) Grocery stores and (d) School buildings. Refer to Table 3-1 for details on model scenarios (x-axis) and Table 2-2B for details on chemicals in Groups G1, G2, G3, G4, and G5 (y-axis)

For pollutants from indoor sources that have a low K_{oa}, the intake fraction drops rapidly as the fraction of outdoor make-up air increases to approximately 0.4 (Figure 3-12). As the value of K_{oa} increases, indicating that more of the SVOC is attached to particles, the intake fraction is less impacted by fraction of make-up outdoor air.

For high K_{oa} pollutants (Group 1), e.g., $\log(K_{oa}) \geq 12$, as the outdoor air fraction becomes high, the total SVOC removal rate increases because venting air to outdoors (which increases with outdoor air make-up fraction) removes all SVOCs in the vented airstream while the filter removes SVOCs from recirculated indoor air with less than 100% efficiency. Consequently, as the outdoor air fraction becomes high, there is only a slight decrease in the intake fraction. For these high-K_{oa} pollutants, as the removal rate by ventilation decreases (decreased outdoor air make-up fraction), there is a compensating increase in removal of the pollutant by filtration and there is no substantial reduction in exposure with the increase in outdoor air fraction.

Table 3-5: Summary of key results

Pollutant	Group	Source location	Exposure decreased by increased ventilation?	Exposure decreased by filtration?
$\log (K_{oa}) < 9$	3,4,5	Indoors	Yes	No
$9 <= \log (K_{oa}) < 11$	1,2	Indoors	Modest impact	Modest impact
$\log (K_{oa}) > 11$	1	Indoors	No	Yes
$\log (K_{oa}) < 9$	3,4,5	Outdoors	No	No
$9 <= \log (K_{oa}) < 12$	1,2	Outdoors	No	Modest impact
$\log (K_{oa}) > 12$	1	Outdoors	No	Yes

Sensitivity analysis

The sensitivity analysis gave insight to evaluate parameters that are most responsible for driving the values of iF and iPOP up or down and to identify uncertainty importance. The results of the sensitivity analysis are detailed in the Appendix B in Figure B2. To summarize, we examined the differential change in percentage removal of pollutant by ventilation, which subjecting each input to an incremental change. The sensitivity variability factor was calculated for each input parameter. We found that the output was most sensitive to changes in the boundary layer thickness. The output was also sensitive to other parameters such as outdoor concentrations of PM, fraction of organic carbon in PM, and thickness of carpet.

DISCUSSION

In this section, we will review and evaluate results for (a) pollutants that do not partition to surfaces such that they remain entirely in the air phase (O_3, CO, NO_2, and radon) (b) particulate matter and (c) pollutants such as VOCs and SVOCs that partition both to surfaces and particles such that the impact of ventilation on exposure requires a more detailed model evaluation across a range of system parameters.

Model evaluation results

For pollutants that are do not partition to indoor surfaces and remain entirely in the air phase, the model-based analysis here reveals that the impact of ventilation depends on the pollutant source. Increased ventilation rates will not reduce exposures to CO or NO_2 in most commercial buildings, because there are normally no significant indoor sources. The role of ventilation in reducing exposures to radon is uncertain because of the absence of quality data on indoor source strengths in commercial buildings. For California, radon concentrations are generally low. Increased ventilation will increase indoor concentration of outdoor air particles, but the impact is modest if the outdoor air enters through a filtration system with a moderate to high filter efficiency. Increased ventilation will decrease indoor concentrations of indoor generated particles, but again the impact will be modest if the building has a filtration system with a moderate to high efficiency and an air recirculation rate of at least a couple indoor air volumes per hour.

Thus, CO, CO_2, radon, and particles are not contaminants that necessarily drive the selection of minimum ventilation rates in most commercial buildings in California. However, there are exceptions. In buildings with significant indoor combustion sources such as from cooking, CO and NO_2, and possibly particulate matter, may be key pollutants for which ventilation can mitigate exposure. There are also buildings such as gyms, salons, and dental offices, which could have atypical pollutant sources, compared to the rest of the commercial buildings. Also, if a building has no particle filtration or low efficiency filters (such as a filter with a MERV 6 rating), ventilation standards may need to consider indoor-generated particles.

For VOCs and SVOCs, the importance of ventilation rate depends on the location of the source and the K_{oa} value of the substance of interest. If outdoor air is the dominant source, increased ventilation will not reduce exposures and may increase exposures. If indoor sources dominate, which is common for many VOCs and SVOCs, increased ventilation will be very helpful in controlling exposures when the K_{oa} is low (e.g., $\log(K_{oa}) < 9$) and not helpful if the K_{oa} is large

(e.g., $\log(K_{oa}) > 12$). With intermediate values of K_{oa}, ventilation is moderately effective in reducing exposures. If food and dermal contact is an important source of exposure, which is likely for many of the key SVOCs, the impact of ventilation rate on total exposures will be substantially smaller than indicated by our analyses.

Comparison to measured data

Very few studies report the impact of ventilation rate in commercial buildings on airborne VOC concentrations. Hotchi et al. (2006) measured VOC concentrations in a big box retail store. An average 50% increase in concentrations of VOCs was seen when some air handling units in the building were turned off for load handling. Menzies et al. (1996) carried out a controlled double blind study in office buildings, where lower VOC concentrations were measured when ventilation rates were increased. Menzies et al. (1996) reported that greater ventilation in office buildings, led to higher indoor concentrations of NO_2 and particulate matter, pollutants which have dominant outdoor sources. Zuraimi et al. (2006) reported that shutting down the ventilation system caused an increase in VOC levels in office buildings in Singapore. Also, Hodgson et al. (2004), report that concentrations of pollutants with indoor sources decreased with increased ventilation in studies carried out in a call center in the US.

Limitations

Currently our model limits the exit pathways of the SVOCs from indoor air, since we do not yet include cleaning and surface reactions as removal processes. Flow of consumables out of the buildings, such as clothes, trash, and other products will also alter the SVOC load indoors. Additionally, humans may serve as SVOC sources/sinks and contribute to redistribution of SVOCs from their actual source location. We expect that human activities in commercial buildings, such as periodic cleaning could alter the flux of chemicals from the room air into surfaces such as carpet, wall, and vinyl. Thus for SVOCs, the modeled results provide an upper bound on how well ventilation can perform in pollutant removal. We have not modeled chemical reaction pathways, which could be a significant removal mechanism for some VOCs and SVOCs such as terpenes and phthalates (Weschler et al. 2000).

For particles, we have assumed removal by a filter system with fixed particle removal efficiency relative to current typical practice. Filter efficiencies will generally increase during filter use and we have not accounted yet for this temporal variability of efficiencies. Our modeling has also assumed that air is recirculated through filters at a rate typical of existing commercial buildings. In future energy efficient buildings, air recirculation rates, and thus particle filtration rates, may be reduced and result in indoor concentrations of indoor-generated particles and SVOCs more highly affected by the rate of ventilation. Some of our results are based on the assumption that minimum outdoor-air make-up fractions are typically in the range of 0.1 to 0.4 and total air supply rates are approximately four indoor air volumes per hour. This is clearly the design intent for many buildings, but measured data are limited. Also, the total air supply rates per unit indoor air volume may be significantly smaller in buildings that have higher ceilings, such as some retail buildings and large assembly rooms. Many commercial buildings have different schedules for air handler operation and outdoor air ventilation during nights and weekends however, we assume that the building is ventilated continuously at the same rate over the course of the annual modeling.

We do not account for SVOC and particle entry through building infiltration—that is transfer through small openings in the building envelope. Some types of commercial buildings such as small retail buildings can also have other direct air exchange with the outdoors through doors that are kept open. For the modeling of how ventilation rates affect SVOC exposures, we have also neglected indoor sources of particles that contain bound SVOC. For most of the pollutants considered, there are limited data available in the literature to validate the model predictions. For SVOCs, the fugacity model is the best available tool; however, very limited data are available for in-depth model performance evaluations.

There may be numerous unidentified pollutants in indoor air, and for some identified pollutants we have no established health thresholds. This paper has only considered pollutants reported in previous studies. Because this is an exposure study, we have only assessed the effects of pollutants individually, exposures to mixtures of pollutants have not been explicitly assessed.

Key pollutants for minimum ventilation standards in commercial buildings
Table 3-5 lists VOCs and SVOCs measured in commercial buildings, at concentrations exceeding or approaching guidelines or odor/sensory thresholds, plus the common phthalates and flame retardants, and other SVOCs of concern for health. This table also provides an estimate of K_{oa} values for these substances. The final column of the table indicates whether ventilation rate is predicted to have a large, intermediate, or small impact on exposures when the sources are indoors. The table also includes particles and the inorganic pollutants discussed in this chapter, except for radon. If a large impact is indicated as a result of sources being typically indoors and indoor concentrations approaching levels of concern, the potential for reducing pollutant exposure should be a priority for establishing minimum ventilation standards. When a small impact is indicated or the dominant source is from outdoor air, the pollutant need not be considered for the establishment of standards, except when our key assumptions--such as the absence of unvented indoor combustion--are invalid. When an intermediate impact is indicated, further analyses may be needed before determining whether the pollutant is an important consideration for ventilation standards.

CONCLUSIONS

This chapter provides an evaluation of how effectively ventilation can remove compounds from indoor environments and alter occupant exposures. We found ventilation to be the dominant removal mechanism for radon and CO. It was found to contribute significantly to O_3 and NO_2 removal when the predominant sources are indoors. However, since the dominant source of O_3, CO and NO_2 in most commercial buildings is outdoors, increased ventilation rates either increase indoor exposures or have only a small impact on indoor exposures. Thus, minimum ventilation requirements in such buildings need not be based on the need to control exposures to O_3, CO and NO_2. For radon, we have minimal data from commercial buildings in California. If the outdoor air is the dominant source of radon, ventilation rates will not impact exposures.

For particles from outdoor air, exposures increase as ventilation is increased. For particles with indoor sources, increasing ventilation decreases exposures, but the impact is modest if the building has a good filtration system. In most commercial buildings, outdoor air is the dominant source of particles, although there may be exceptions such as restaurants and other buildings

with indoor combustion. In general, large ventilation rates will be a poor strategy for controlling indoor concentrations of particles in commercial buildings and efficient filtration is expected to control particulate concentrations and be cost-effective. So, minimum ventilation requirements will not, in general, be based on the need to control exposures to particles.

The chemical properties of VOCs and SVOCs have important impacts on the modeled effects of ventilation rates on exposures to these compounds. For VOCs with $\log(K_{oa}) < 9$, when the outdoor air is the dominant source, increased ventilation rates will increase exposures, however the increases are not high. With an indoor source and $\log(K_{oa}) < 9$, increased ventilation can significantly reduce exposures, thus, the need to control exposures to these compounds may determine minimum ventilation requirements in most commercial buildings. Some examples of pollutants with $\log(K_{oa}) < 9$ include low molecular weight aldehydes such as formaldehyde and acetaldehyde and aromatics such as toluene. Many of these pollutants are VOCs with predominantly indoor sources, which makes ventilation an important removal pathway.

For compounds with $\log(K_{oa})$ between 9 and 12, with an outdoor source, increases in ventilation causes significant increase in exposures. Similarly, when there is only an indoor source, increase in ventilation causes a reduction in exposures. The effect of ventilation on exposures is not as significant as for compounds with $\log(K_{oa}) < 9$. The model likely overestimates the effect of ventilation on these compounds with $\log(K_{oa})$ between 9 and 12 because removal by processes other than ventilation is not yet incorporated in the models.

For indoor-generated SVOCs that have $\log(K_{oa}) > 11$, increases in the outdoor air fraction above 0.3 have a relatively small impact on exposures, with filtration a larger source of contaminant removal than ventilation. For SVOCs with large values of $\log(K_{oa})$ (> 12) and an indoor source, increased ventilation is predicted to have a small impact on exposures. Examples of some compounds with $\log(K_{oa}) > 11$ are brominated flame retardants, heavier phthalates like diethyl–hexyl phthalate, pesticides like permethrin, and dioxins compounds. There is limited empirical data to validate the model predictions. There are also substantial differences among the commercial building stock in characteristics and operations not captured in our modeling. The limited available data and modeling results are consistent with our predictions of the impacts of ventilation on indoor pollutant exposures.

CHAPTER 4. Dermal exposure to semi-volatile compounds in indoor environments

ABSTRACT

In modern indoor environments, we are exposed to a broad suite of semi-volatile organic compounds. SVOCs, such as phthalates and flame retardants, are ubiquitous and present in various microenvironments such as residences and commercial buildings. We explore the role of air in passive dermal uptake of chemicals. To test the role of passive uptake in adults, we evaluated skin loading of SVOCs by collecting wipe samples from adults. We collect wipe samples from foreheads since they are expected to provide a reliable estimate of passive air to skin transfer. We collect three sequential wipe samples from the foreheads of adults along with one hand wipe sample. The hand wipe sample is collected to provide a contrast between passive air to skin transfer and contact driver transfer. The wipe samples are analyzed for a suite of SVOCs and squalene, which is a skin surface lipid. For a limited sample size of 2 subjects, we also analyze the sapienic acid concentrations. The data on squalene is used to infer the depth of sampling associated with each skin wipe sample. In agreement with common knowledge on skin wipes, we are able to quantify that the first skin wipe removes chemicals from about 1 μm of the skin surface. Each subsequent wipe removes chemicals from about 0.5 μm of the skin surface. We also model the concentration profile of select pollutants in the stratum corneum. The modeled concentrations are compared to measured concentrations to assess the fit. We use the information on model fit to develop a metric called 'equivalent time of exposure'. The ETE is calculated by combining the information across the same wipe sample for the subjects. This metric is used to assess the usefulness of skin as a passive sampler for recent exposures. Overall the results of sampling indicate that a wide suite of SVOCs is present in indoor environments. This drives the need for better understanding of the exposure pathways, such as passive dermal uptake from air. The passive dermal uptake is evaluated using forehead wipe samples. In addition modeling the transfer of SVOCs in the stratum corneum shows that the skin membrane can be a useful indicator of recent exposures to chemicals.

INTRODUCTION

Humans spend a significant proportion of their daily schedule inside of buildings (Klepeis et al. 2001). In modern buildings there are concerns about human exposure to a broad range of semi-volatile organic compounds (SVOCs) that are transferred from building materials, furniture, consumer products and personal-care products to indoor surfaces and indoor dust. This chapter focuses on methods to better understand the dermal uptake of SVOCs from indoor environments. Both human subject skin surface wipes and theoretical models provide the tools for assessing residential dermal uptake. To set the framework for the assessment of dermal uptake of SVOCs, we begin with a literature-summary-based background discussion of the fate of SVOCs in and among the air and surfaces of the indoor environment. This establishes what media are relevant for indoor dermal contact and uptake assessment. The background summary also addresses what is known about both passive and active dermal uptake indoors and to what extent this knowledge comes from models and measurements.

Based on insights from the background material, we propose methods to evaluate how to make optimum use of skin wipes as a sampling method for dermal exposure. We describe the approach we used to collect hand wipe samples that can be used to measure the levels of chemicals of

interest along with the skin lipid squalene. We propose an approach for estimating the sampling depth of a skin wipe, by taking sequential wipe samples. Some limited data (2 subjects) on oxidation products also provides useful information on depth of chemical removal by skin wipes. Since there are no known analytical standards for skin oxidation products we do not provide concentration estimates. However, we are able to assess a relative measure of how the oxidation product values vary in sequential wipes. In addition, we also measure sapienic acid levels for a limited sample size of 2 subjects. We study the passive transfer of chemicals to skin from air by collecting sequential skin wipe samples from an area having primarily passive contact (the forehead) and contrast these measurements with those from an area with contact driven transfer of chemical to skin by collecting simultaneous hand wipe samples. We provide the estimates of chemical concentrations both as total $\mu g/cm^2$ and normalized by skin lipid as μg/g-squalene.

To evaluate and compliment the experimental work, we developed a model for estimating the transfer of chemical through skin, based on Fickian diffusion within the stratum corneum. We calculate the diffusion coefficients for the model using the empirical models developed by the United States Environmental Protection Agency (Guy and Potts 1993; USEPA 2007) to study the sensitivity of concentrations in skin at various depths to the diffusion coefficient. Three compounds are selected for skin modeling--di-ethyl phthalate, di-methyl phthalate and di-n-butyl phthalate. We provide estimates of the chemical concentration in air using standard partitioning modeling and from this air concentration estimate the depth profile of concentration of chemical in the upper layers of the stratum corneum. This is compared to estimates of the skin chemical concentration profile obtained using consecutive skin wipe samples. We conduct the analysis to explore the feasibility of using skin wipes to develop a concentration profile of chemicals in the upper layers of the stratum corneum, and subsequently explore the role of skin as a passive personal sampler for recent exposures.

BACKGROUND

In this section, we set the framework for the assessment of dermal uptake of SVOCs based on a literature-based background discussion of the fate of SVOCs in and among the air and surfaces of the indoor environment. We use this review to identity the indoor environmental media relevant for indoor dermal contact and uptake assessment. We also review here what is known about both passive and active dermal uptake indoors and to what extent this knowledge comes from models and measurements.

Indoor Distributions of SVOCs

Numerous classes of SVOCs such as flame retardants, phthalates, and pesticides are found in indoor environments. SVOCs can be found largely on indoor surfaces and in the particle phase, due to their high affinity for organic media compared to the gas phase (Weschler and Nazaroff 2010). Sources include flooring, walls, countertops, furniture surfaces, upholstery, electronic equipment, and personal care products (Xu et al. 2011, Rudel et al. 2010, Wensing et al. 2005). High production volumes of SVOCs and their use in consumer products, has resulted in significant environmental exposures. By modeling the interaction and movement of SVOCs among various indoor surfaces, Weschler and Nazaroff (2008) developed an equilibrium partitioning approach to understand SVOC transport and distribution in the indoor environment. They report that SVOCs tend to be mostly associated with particles in air. As a result, the total concentration in air can exceed the gas-phase concentration estimated based on the saturation

vapor pressure. Strongly sorbing surfaces that have higher boundary-layer mass-transfer resistance for air-to-surface transfer tend to take a long time to come to equilibrium with the indoor air. Once they have taken up SVOCs, these surfaces can continue to act as sources of SVOCs even if the source is physically removed. Even though it does not contain a majority of the indoor inventory of SVOCs, air is typically the medium that controls the distribution of SVOCs in the indoor environment. Current research on the fate and transport of SVOCs in the indoor environment shows that they are present on various indoor surfaces as well as on particulate matter and have long residence times (Weschler and Nazaroff 2010, Shin et al. 2013, Zhang et al. 2009). Indoor environments are capable of delivering pollutants to human occupants at a continuous rate for years (Shin et al. 2013). Direct measurements on the relative importance of the primary exposure pathways (inhalation, dietary and non-dietary ingestion, and direct and indirect dermal uptake) are limited. There is significant uncertainty about the relative contribution of each pathway to cumulative intakes and health impacts.

Once they have taken up SVOCs, these surfaces can continue to act as sources of SVOCs even if the source is physically removed. In air, SVOCs tend to be mostly associated with particles. As a result, total concentration in air can far exceed the concentration estimated based on the saturation vapor pressure. The time taken to achieve equilibrium partitioning is heavily influenced by the unitless octanol-air partitioning concentration coefficient (K_{oa}), which expresses ratio of concentration in octanol, in mol/m^3, divided by concentration in air in mol/m^3 in contact with the octanol. As K_{oa} increases from 10^{-7} to 10^{-14}, the time for SVOCs to reach equilibrium increases from 1 h to >30 years. The thickness of the film acting as a receptor compartment also affects the time to equilibrium, to a much lesser extent. Similarly, K_{oa} affects the rate of sorption onto particles, with decreasing K_{oa} leading to slower rates.

Passive and Active Dermal Uptake

In an effort to better understand exposure to SVOCs, there is a need to study the role of air as the exposure medium and quantify the amount of chemical delivered by air for dermal uptake. Pollutants can transfer from indoor environments to the skin surface through at least two pathways: air-to-skin transfer and contact driven transfer with indoor surfaces. We focus here specifically on the air-to-skin pathway and attempt to contrast it with the contact driven uptake of chemicals. We also study how effectively the air mediated delivery of various chemicals changes as a function of the chemical properties. Diffusivity through air and lipophilicity of chemicals are important parameters that influence the concentrations of chemicals in various media such as air, skin and other indoor surfaces. From the results of Chapter 3 we know that chemicals with a high octanol-air partitioning coefficient (K_{oa}) preferentially partition onto organic films and in this chapter we discuss skin, another organic surface. In this chapter we discuss skin and other organic surfaces. Using a modeling approach, Weschler and Nazaroff (2008) have demonstrated theoretically that volumetric clearance by skin in the indoor environment can be as high as 10 to 20 m^3/h for SVOCs with high octanol-air partition coefficients, higher than typical inhalation intake of 1 m^3/h. But without residential measurements we cannot determine whether this volumetric clearance represents what actually goes into or through skin. Our analysis of the Weschler and Nazaroff (2008) results reveals that this uptake rate can only apply when the air is maintained at relatively constant chemical potential (or fugacity)—a situation that may not apply if the skin and other surface uptake processes deplete SVOC inventory in air. This situation is likely when we consider that the air of

the indoor environment contains very little of the indoor mass of SVOS and mass transfer resistance limits the ability of SVOCs reservoirs in indoor surfaces replenish what is removed from air by occupants. Weschler and Nazaroff (2008) modeled the dermal uptake from air to skin highlighting that dermal uptake of chemical from the gas phase could be an important exposure pathway for SVOCs. A recent study by Weschler et al. (2016), evaluated the direct air to skin to blood transfer of SVOCs for di-ethyl phthalate (DEP) and dn-n-butyl phthalate (DnBP). Volunteer subjects were exposed for 6 hours to elevated concentrations of DEP and DnBP in a room. In one set of experiments, the volunteers were exposed to the pollutants while breathing DEP and DnBP free air from a hood. In another set of experiments they also breathed the room air laden with DEP and DnBP. Urine samples were collected for 54 hours after, and they were analyzed for metabolites of DEP and DnBP. The experiments showed that for both DEP and DnBP dermal uptake from air is a significant exposure pathway, comparable to inhalation uptake. Morrison et al. (2016), conducted similar experiments as Weschler et al. (2016), to study the effect of clothing on dermal uptake of phthalates. They exposed subjects wearing clean and soiled clothing (clothing exposed to pollutants for 9h in room) for 6h in a room with known concentrations of DEP and DnBP. Clean clothing was seen to a protective effect against dermal uptake and soiled clothing was shown to enhance uptake 3-6.5 times compared to the participants in the Weschler et al. (2016) study.

Although several studies employ modeling and experimental approaches to evaluate exposure to SVOCs, none include an empirical measurement of dermal uptake. The US Environmental Protection Agency's Children's Total Exposure to Persistent Pesticides and Other Persistent Organic Pollutants study (USEPA CTEPP) was designed to estimate the total exposure to chemicals in the indoor environment. Surface wipes, food samples, water, air and urine samples were collected in homes and day care centers for nearly 257 children. Modeling estimates explained about 60% of the permethrin metabolite concentrations found in urine. McKone et al. (2007) combined an indoor fugacity model with the CalTOX (McKone et al. 2003) model to estimate the inhalation intake of organophosphate pesticides in the Salinas Valley. Data from the Total Diet Survey (FDA TDS) on pesticide residues was used to estimate the ingestion intake, but the study did not fully assess dermal uptake. Conversely, Zartarian et al. (2000) used a combination of stochastic and mechanistic models to estimate the non-dietary ingestion and dermal uptake of chlorpyrifos, but did not characterize inhalation and dietary intake.

Lorber (2008) estimated the relative contribution of dermal, ingestion and inhalation exposures to total PBDE levels. Their assessment was based only on data available from the extant literature and combined with a pharmacokinetic model. Results showed that the modeled PBDE levels were consistently lower than reported values of the compounds. Gong et al. (2014) built a transdermal uptake model based on Fickian diffusion through the skin membrane. The rate of dermal uptake of several phthalates: di-ethyl phthalate, di-iso-butyl phthalate, di-n-butyl phthalate, butyl benzyl phthalate and di-ethyl-hexyl phthalate were modeled. In the scenarios modeled, the rate of dermal uptake of chemicals was found to be comparable to inhalation uptake. Lorber et al. (2016) employed a combination of a transdermal uptake model with a pharmacokinetic model to predict the rate of uptake of DnBP from air. They compared the model predictions to experimental measurements by Weschler et al. (2016). The model was found to overpredict DnBP metabolite concentrations by 1.1-4.5 times. The authors applied the model to

typical airborne concentrations of DnBP in the United States and concluded that total intake of DnBP from air via inhalation and dermal could account for up to 25% of total exposure.

These relevant but limited assessments reveal the need for a comprehensive model that uses a full range of chemical-fate models, empirical estimate tools and measurements to quantify all three exposure pathways. Also, as highlighted by Kissell (2008), dermal permeation and uptake, has often been underestimated in the exposure-science literature. Most of the studies measuring flux through skin use high concentrations of chemicals in the donor solution placed in a diffusion cell above an excised skin sample. The flux is typically measured using the difference in concentration between the donor and receiver solution. The chemical adsorbed on skin surface and within the skin sample are not accounted for, leading to a potential underestimate of dermal permeation.

Skin as a Mass-Transfer Medium

The skin is the largest organ in the body, and it is comprised of two distinct layers: the epidermis (stratum corneum and viable epidermis) and the dermis. The thickness of the skin is variable throughout the body, it is thinnest in areas such as the forehead, face, forearm, scalp, and back of the hand, which are typically the maximally exposed areas. The dermal permeability of chemicals has been shown to be a function of octanol-water partitioning coefficient and molecular volume. Empirical models (Potts and Guy 1992, 1995; McKone and Howd 1992) and mechanistic approaches (Mitragotri 2003) have been developed and used to derive mathematical functions for describing skin permeation based on the solution to Fick's second law of diffusion and used to estimate the rate of permeation through the epidermis. One output of these models is the permeability coefficient, which is a composite parameter that accounts for resistance to transfer in the lipid and aqueous layers of the skin membrane. The corresponding skin diffusion coefficient can be calculated using the permeability coefficient at steady-state flux conditions. Lian et al. (2008), provide a summary of compounds for which dermal permeability coefficients have been determined using human skin for in-vitro experiments in diffusion cells. It is noteworthy that all compounds that have been studied have relatively low octanol-air partitioning coefficient ($<10^6$) and low molecular weight (<200 g/mol). The skin is a complex membrane and the skin diffusion coefficient is an important parameter that determines the movement of chemical through skin. However, it is also one of the more poorly characterized parameters involved in dermal uptake estimates. Typically, the diffusion coefficient is determined using the permeability coefficient, which is rate of movement of chemical through skin at steady-state conditions. There is a lack of data particularly for direct measurements of skin diffusion coefficient for compounds in the chemical property space into which SVOCs normally fall, as seen in Figure 4-1.

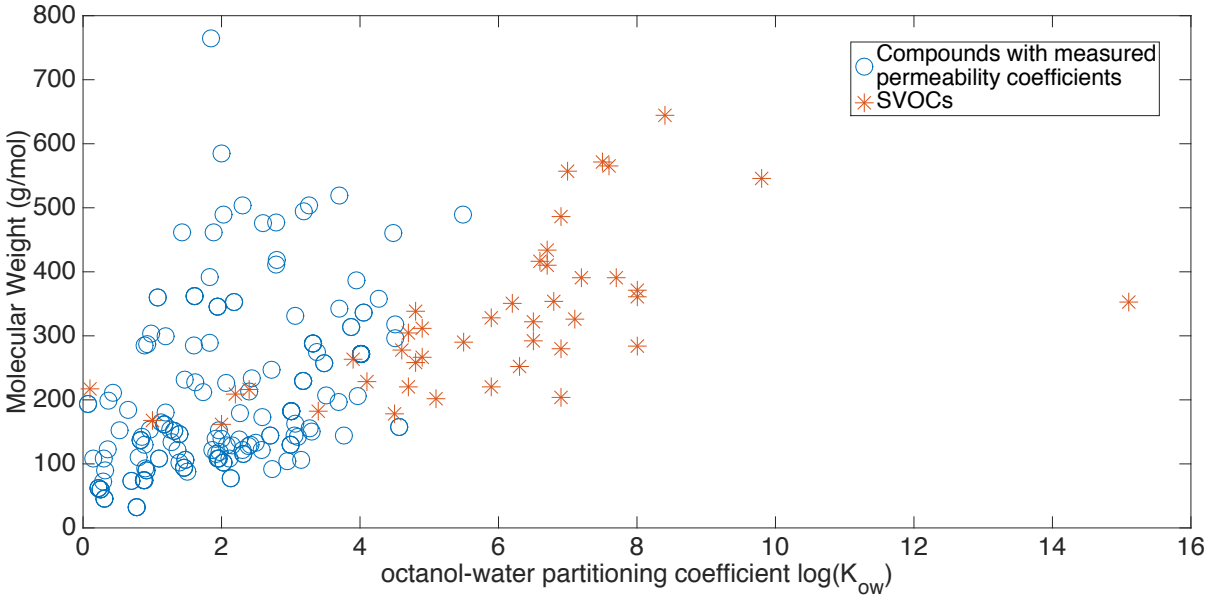

Figure 4-1: Permeability coefficient data availability, adapted from Lian et al. (2008)

The indoor dermal uptake of SVOC involves two processes: 1) loading of chemical on skin and 2) permeation of chemical through skin into the blood perfused tissues of the dermis. The loading of chemical on skin includes two transfer pathways -- contact driven loading of chemicals on skin and passive air-to-skin transfer of chemicals. The combined dermal uptake of chemicals through both passive and active transport remains highly uncertain because of a lack of field measurements. Based on the skin uptake model described below, we hypothesize that contact driven uptake will dominate the loading for children and toddlers and air-to-skin transfer will be the primary loading mechanism for adults. To test the role of passive uptake in adults, we evaluated skin loading of SVOCs by collecting wipe samples from adults. We collect wipe samples from foreheads since they are expected to provide a reliable estimate of passive air to skin transfer. We collect three sequential wipe samples from forehead of adults along with one hand wipe sample. The hand wipe sample is collected to provide a contrast between passive air to skin transfer and contact driver transfer. Wipe sampling is a common method used to analyze dermal exposure. Some studies have collected single wipe samples from a single location, (typically hands) to assess dermal exposure (USEPA CTEPP). There are some studies, which collect more than one wipe sample and at different locations (Gong et al. 2013). Gong et al. (2013) measured phthalate levels in wipe samples. The results showed that phthalate levels vary by sampling location and time of collection of the skin sample.

There are various lipids present on the skin surface, secreted by the epidermal and sebaceous glands. Sebaceous gland secretions comprise of triglycerides, wax esters, free fatty acids, cholesterol esters and squalene. Epidermal secretions are predominantly free fatty acids and cholesterol, these lipids are not as abundant as the sebaceous gland secretions. As described in literature (Nicolaides et al. 1974; Downing et al. 1969; Strauss et al. 1991), the mass composition of the skin lipids is as follows: triglycerides (20-40%), wax esters (23-29%), squalene (12%), free fatty acids (5-40%), and cholesterol and wax esters (1-5%). The skin surface lipids primarily of interest for our research are squalene and sapienic acid. Squalene is the most abundant lipid on the skin surface, it belongs to the class of compounds called tri-terpenes. Squalene is primarily

secreted by the sebaceous gland. A useful marker of epidermal secretions is sapienic acid. This compound is found exclusively in human skin, hence the name sapienic acid, it belongs to the class of unsaturated fatty acids. The current work also attempts to estimate the depth of sampling by skin wipes. To assist in this effort we have looked at the ratios of certain oxidation products of squalene and sapienic acid to their parent compounds. The oxidation of those compounds is discussed in more detail below.

Squalene is a tri-terpene that has three double bonds. This chemical structure makes it highly reactive towards ozone, as established by Wisthaler and Weschler (2010). Squalene can react with ozone to produce many oxidation products, both volatile and non-volatile. While there has been some research on the volatile oxidation products of squalene-ozone reactions, the non-volatile compounds are not as well understood. The ozone molecule attacks the double bonds, leading to the formation of a primary ozonide compound. The primary ozonide compound can break down into other products leading to formation of a variety of ozone reaction compounds (Wisthaler and Weschler 2010; Petrick and Dubowski 2009; Fonshee et al. 2015). This is the first study to evaluate squalene, sapienic acid and ozone oxidation product concentrations in skin wipe samples. Another area of exposure science that has not been well studied is the use of skin as a passive sampler of personal exposures. The skin is subject to a variety of changes due to human activities such as bathing, changing clothes, sleeping and other activities. However, lipids on the skin surface can take up chemicals from air passively and serve as a useful sampler for personal exposures. In addition, although it not documented, we expect that other reactive oxidative species (ROS) can also react with skin lipids altering the indoor chemistry, they reduce the concentrations of ROS and increase concentrations of volatile reaction products in air (Gligorovski and Weschler 2013).

For this research, we focus on phthalates for dermal modeling because they have established health effects and have been shown to permeate through skin (Weschler et al. 2016). Phthalates with higher molecular weights such as diethyl hexyl phthalate are used in vinyl flooring as plasticizer, and lower molecular weight phthalates such as di-ethyl phthalate and di-n-butyl phthalate are used in perfumes, cosmetics and other personal care products (Schettler 2006; Hauser 2005). Phthalates are quickly metabolized to their monoesters, and then subject to phase II metabolism by glucoronidation (Silva et al. 2003; Blount et al. 2000). Higher levels of some phthalate metabolites in people have been linked to personal care product use (Silva et al. 2004; Adibi et al. 2003; Janjua et al. 2007; Duty et al. 2005). It is hypothesized that relative to men, women use more personal care products, which contain phthalates, leading to higher exposures. Studies have not been conducted on permeation through human skin. DBP and other phthalates have been linked to a variety of human toxicology effects, such as reproductive teratogenicity, and decreased sperm quality in men, and thyroid function disruption (Swan et al. 2005; Duty et al. 2003; Meeker et al. 2011). Another study highlighted that greater levels of phthalates in urine are linked to homeostatic model assessment, a measure of insulin resistance (Stahlhut et al. 2007). Models were adjusted for age, race, fat consumption, physical activity levels, serum cotinine serum and urine creatinine. The associations between metabolite levels and insulin resistance were found to be significant. After adjusting for renal and hepatic function, the association was weaker but still significant.

METHODS

In this section, we discuss methods used for skin wipe collection and interpretation the modeling approach used to make estimates of skin permeation. The skin-wipe samples and the modeling analysis provide complimentary tools for assessing the mechanisms, magnitude, and significance of dermal uptake of SVOCs from the indoor environment.

Skin wipe collection and interpretation

Our skin-wipe collection was carried out as a convenience sample to explore what is on the skin of a group of subjects recruited from Northern California. The purpose of the sampling is to get a scoping assessment of what might be found in human skin. This effort was not intended as a probabilistic sample. The premise of the study is that the ubiquitous nature of SVOCs in the indoor environment suggests that we should find SVOCs in the skin of almost all randomly selected subjects. The skin wipe samples also provide the opportunity to explore differences in passive versus active sampling and the extent of permeation below the skin surface.

Study Design – We recruited 13 subjects for our wipe-sampling study. The subjects were all adults 18 years of age or older. They comprised of 7 men and 6 women and represented a convenience sample. The samples were representative of residential exposures. The study team member visited the participants at their residence to collect samples. One study team member (to reduce variability) collected all samples. The participants were required to sign a consent form and their date of birth and gender were provided. Gauze pads (2 cm x 4 cm) were first cleaned with 1:1 mixture of hexane and acetone in a Soxhlet extractor. The gauze pads were air-dried and then wrapped in aluminum foil, which was cleaned prior to use with isopropanol. We collected three consecutive wipe samples from the foreheads, and one wipe sample from the palm. We also collected one blank sample per subject. The wipe protocol is detailed in Appendix C.

Analysis – Each wipe sample was collected using a clean gauze pad and the samples were immediately stored in a cooler for transportation after collection. The wipes were first extracted using a 5 ml, 3:1 mixture of hexane and acetone, and sonicated. After transferring the supernatant we extracted from the wipes again by adding 5 ml of acetone and sonicating. The supernatant was again transferred and we evaporated the sample to 1 ml. The sample was filtered and split using a Hamilton syringe for gas and liquid chromatography analysis. For gas chromatography samples, an internal standard mix was added to the extract before analysis. The operating conditions for the gas chromatography/mass spectrometry (GC-MS) are detailed in the Appendix C, Table C1and Table C2. The list of chemicals analyzed in the GC-MS is provided in Table 4-1. The samples were analyzed for a suite of phthalates, and one skin surface lipid--squalene. The skin surface lipids of primary interest for this study are squalene, sapienic acid. The liquid chromatography LC analysis was conducted only for a limited sample of two subjects. The LC analysis was conducted for sapienic acid and oxidation products of ozone.

The chemicals we analyzed were in the classes of ultraviolet (UV) protection (octocrylene, homosalate), musk (galaxolide), plasticizers (di-methyl phthalate, di-ethyl phthalate, bis-2-ethyl hexyl phthalate, butyl benzyl phthalate, di-n-butyl phthalate), and flame retardants (tris-chloro isopropyl phosphate, tri-n-butyl phosphate). The summary statistics (mean, median, 5^{th} and 95^{th} percentile) of compounds for each wipe sample are given in Table 4-1. We report the concentrations in two metrics: $\mu g/cm^2$ and g/g-squalene. We use $\mu g/cm^2$ as the standard

reporting metric, which enables us to compare the measurements to values reported in literature. There was a decrease in the levels of compounds for most chemicals in the sequential wipe samples. In addition, we performed a one-way analysis of variance (ANOVA) to examine whether the levels are significantly different between forehead wipe 1 (FH-1) and forehead Wipe 3 (FH-3) for the compounds. In Table 4-4 we explore the correlation of chemical concentrations found on the forehead wipe (FH-1) and hand wipe (HW). We calculated the Spearman correlation coefficient for the FH-1 and HW samples, which provides a non-parametric measure of their association. We use the following notations for wipes throughout this chapter (a) Forehead Wipe 1 (FH-1) (b) Forehead Wipe 2 (FH-2) (Forehead Wipe 3 (FH-3) and Hand Wipe (HW-1). The correlation coefficient was also evaluated for the different chemicals to assess whether compounds could be attributed to a common source. We compare the amount of squalene extracted over the given area to standard literature estimates of squalene levels and amounts extracted by wipe samples to estimate the amount of skin surface sampled by a skin wipe. The correlation coefficients for various chemicals in each wipe sample were also calculated.

RESULTS

We obtained GC-MS analysis results for 20 chemicals of interest for environmental health, which included UV protection products, personal care products, plasticizers, and flame retardants. In Table 4-1, we list the completeness of the data for the various chemicals. The following compounds were present in >90% of the samples collected: octocrylene, homosalate, galaxolide, di-methyl phthalate, di-ethyl phthalate, di-n-butyl phthalate, bis-2-ethyl hexyl phthalate, di-octyl terephthalate and squalene. We conducted detailed analyses only for squalene plus the 12 compounds that are measured in more than 50% of the samples.

Table 4-1: Percent completion of data
% complete = Number of samples (>LOQ)/Total number of samples (N)

Number of samples	FH-1, N = 13	FH-2, N = 13	FH-3, N=13	HW-1, N=11
Octocrylene	100	100	100	100
Homosalate	100	100	100	100
Acetyl tributyl citrate	54	54	54	100
Galaxolide	92	92	85	91
Butyl benzyl phthalate	62	46	38	82
Di-methyl phthalate	100	100	100	91
Di-ethyl phthalate	100	100	100	100
Di-n-butyl phthalate	100	100	100	100
Bis (2-ethyl hexyl) adipate	38	15	31	82
Bis (2-ethyl hexyl) phthalate	100	100	100	100
Di-n-octyl phthalate	23	15	15	18
Tri-n-butyl phosphate	38	38	38	27
Tris-1-chloro isopropyl phosphate	92	85	85	100
Tri phenyl phosphate	38	31	31	64
Tris-2-chloroethyl phosphate	15	15	15	9
Tris-1,3-dichloro-2-propyl phosphate	23	15	15	64

Number of samples	FH-1, N = 13	FH-2, N = 13	FH-3, N=13	HW-1, N=11
Tris-2-butoxyethyl phosphate	15	15	15	45
Di-isobutyl phthalate	92	100	100	100
Squalene	100	100	100	100
Dioctyl terephthalate	100	100	100	100

Table 4-3 provides a detailed summary of chemical recovery in the wipe samples. As seen in Table 4-3, we obtain about 40-65% of the chemical in FH-1, removing 50% of the total chemical on an average. The total chemical removed is obtained by summing the total mass of chemicals removed from FH-1, FH-2 and FH-3. In FH-2, we remove about 20-40% of the chemical, removing about 30% on an average. In FH-3, we remove 10-30% of the total chemical and on an average about 20% of the total mass of pollutant removed in all three wipe samples. The ratio of chemical concentrations in FH-1 and HW-1 were also evaluated. Most chemicals exhibit lower concentrations in FH-1 samples compared to HW-1. In analyzing the median ratios of HW-1/FH-1, we find that only di-methyl phthalate is present in higher concentrations in the FH-1 samples compared to HW-1. We present the median ratio for each individual chemical and the range of ratios in the following parenthesis.

Table 4-2A: Summary of concentrations in $\mu g/m^2$

Compound	Concentrations ($\mu g/m^2$)	FH - 1	FH - 2	FH - 3	HW
Octocrylene	Median	33	17	15	94
	Mean	3730	3116	1168	172
	5^{th} percentile	3.1	0.7	0.5	15
	95^{th} percentile	40562	34051	12648	474
Homosalate	Median	54	27	15	75
	Mean	14847	17202	6777	119
	5^{th} percentile	6.9	2.6	1.6	8.8
	95^{th} percentile	163581	189840	74719	607
Acetyl tributyl citrate	Median	52	36	20	52
	Mean	109	57	42	109
	5^{th} percentile	0.7	14	9.1	11
	95^{th} percentile	280	109	105	370
Galaxolide	Median	7.5	2.7	2.0	11
	Mean	25	13	7.3	15
	5^{th} percentile	0.4	0.1	0.0	0.2
	95^{th} percentile	168	97	50	45
Butyl benzyl phthalate	Median	16	9.4	6.4	65
	Mean	16	12	7.2	60
	5^{th} percentile	3.9	1.4	0.8	20
	95^{th} percentile	29	28	14	127
Di-methyl phthalate	Median	1.1	0.9	0.8	1.0
	Mean	1.2	0.8	0.8	1.2
	5^{th} percentile	0.05	0.05	0.04	0.15
	95^{th} percentile	4.1	1.6	1.6	2.1

Compound	Concentrations ($\mu g/m^2$)	FH - 1	FH - 2	FH - 3	HW
Di-ethyl phthalate	Median	154	108	124	207
	Mean	156	124	109	329
	5th percentile	4.5	1.8	1.4	109
	95th percentile	418	342	196	1476
Di-n-butyl phthalate	Median	39	25	20	94
	Mean	40	26	21	99
	5th percentile	17	5.1	5.1	50
	95th percentile	77	60	51	192
Bis (2-ethyl hexyl) phthalate	Median	708	342	259	2072
	Mean	852	342	263	3853
	5th percentile	172	109	79	418
	95th percentile	2357	799	575	15725
Tri-n-butyl phosphate	Median	1.2	0.9	1.0	1.3
	Mean	1.2	0.7	1.1	1.4
	5th percentile	0.1	0.1	0.8	1.0
	95th percentile	2	1.4	1.5	1.8
Tris-1-chloro isopropyl phosphate	Median	3.1	2.3	1.1	11
	Mean	14	7.7	5.5	34
	5th percentile	0.1	0.0	0.0	1.3
	95th percentile	78	49	38	142
Tri phenyl phosphate	Median	4.3	1.8	1.4	4
	Mean	24	19	11	22
	5th percentile	1.6	0.7	0.9	2.9
	95th percentile	107	73	41	130
Di-isobutyl phthalate	Median	49	38	21	92
	Mean	66	39	29	263
	5th percentile	1.3	5.2	3.5	29
	95th percentile	263	139	98	1179
Squalene	Median	226523	125959	78047	14635
	Mean	246076	122697	92531	24837
	5th percentile	89894	55547	15709	4161
	95th percentile	621194	203999	218761	111378
Dioctyl terephthalate	Median	737	256	141	2256
	Mean	1621	825	586	3070
	5th percentile	60	19	15	716
	95th percentile	6702	4433	4062	8124

Table 4-2B: Summary of concentrations in µg/µg-squalene

Compound	Concentrations (µg/µg-squalene)	FH - 1	FH-2	FH-3	HW
Octocrylene	Median	1.7E-04	1.4E-04	1.5E-04	4.2E-03
	Mean	5.8E-03	1.6E-02	5.5E-03	1.7E-02
	5th percentile	7.3E-06	0.0E+00	0.0E+00	1.6E-03
	95th percentile	7.1E-02	2.1E-01	6.7E-02	1.2E-01

Compound	Concentrations (µg/µg-squalene)	FH - 1	FH-2	FH-3	HW
Homosalate	Median	1.8E-04	1.7E-04	2.0E-04	3.3E-03
	Mean	2.2E-02	9.0E-02	3.1E-02	1.8E-02
	5th percentile	2.7E-05	1.8E-05	4.4E-05	6.2E-04
	95th percentile	2.9E-01	1.2E+00	4.0E-01	1.6E-01
Acetyl tributyl citrate	Median	3.0E-04	4.1E-04	7.0E-04	3.5E-03
	Mean	5.2E-04	5.0E-04	5.7E-04	8.0E-03
	5th percentile	7.6E-06	8.1E-05	1.2E-04	7.1E-04
	95th percentile	1.2E-03	1.0E-03	8.8E-04	3.2E-02
Galaxolide	Median	3.4E-05	3.4E-05	1.7E-05	3.5E-04
	Mean	1.4E-04	1.0E-04	1.1E-04	1.4E-03
	5th percentile	7.8E-07	2.7E-07	0.0E+00	0.0E+00
	95th percentile	7.6E-04	7.1E-04	7.8E-04	4.9E-03
Butyl benzyl phthalate	Median	8.5E-05	9.0E-05	8.1E-05	2.9E-03
	Mean	9.8E-05	1.1E-04	1.2E-04	3.1E-03
	5th percentile	3.3E-05	1.6E-05	8.7E-06	8.6E-04
	95th percentile	2.6E-04	2.0E-04	3.4E-04	7.0E-03
Di-methyl phthalate	Median	4.4E-06	7.1E-06	8.3E-06	8.4E-05
	Mean	4.6E-06	7.3E-06	2.0E-05	7.9E-05
	5th percentile	4.9E-07	6.1E-07	5.4E-07	1.0E-05
	95th percentile	9.3E-06	1.7E-05	9.6E-05	2.0E-04
Di-ethyl phthalate	Median	5.3E-04	1.0E-03	1.2E-03	1.7E-02
	Mean	7.1E-04	1.0E-03	2.1E-03	2.0E-02
	5th percentile	0.0E+00	4.6E-06	0.0E+00	1.8E-03
	95th percentile	2.0E-03	2.2E-03	8.6E-03	4.2E-02
Di-n-butyl phthalate	Median	1.8E-04	2.0E-04	2.2E-04	6.4E-03
	Mean	2.0E-04	2.7E-04	5.3E-04	6.7E-03
	5th percentile	4.4E-05	6.4E-05	6.4E-05	1.7E-03
	95th percentile	4.0E-04	8.0E-04	3.5E-03	1.2E-02
Bis (2-ethyl hexyl) phthalate	Median	3.8E-03	2.6E-03	3.3E-03	1.5E-01
	Mean	4.3E-03	3.0E-03	3.6E-03	2.3E-01
	5th percentile	6.6E-04	8.2E-04	5.1E-04	2.0E-02
	95th percentile	1.2E-02	7.6E-03	8.1E-03	9.6E-01
Tri-n-butyl phosphate	Median	3.3E-06	6.4E-06	1.1E-05	7.9E-05
	Mean	3.1E-06	5.9E-06	1.0E-05	1.3E-04
	5th percentile	1.6E-06	1.0E-06	6.9E-06	4.8E-05
	95th percentile	4.2E-06	1.4E-05	1.3E-05	2.5E-04
Tris-1-chloro isopropyl phosphate	Median	1.7E-05	1.8E-05	1.4E-05	9.3E-04
	Mean	8.6E-05	4.8E-05	4.9E-05	2.5E-03
	5th percentile	0.0E+00	0.0E+00	0.0E+00	4.4E-05
	95th percentile	6.0E-04	2.6E-04	2.0E-04	9.5E-03
Tri phenyl phosphate	Median	2.9E-05	1.7E-05	1.8E-05	2.7E-04
	Mean	1.1E-04	1.3E-04	1.6E-04	3.0E-03
	5th percentile	1.2E-05	7.9E-06	1.0E-05	8.3E-05
	95th percentile	4.3E-04	4.7E-04	5.8E-04	1.9E-02

Compound	Concentrations (μg/μg-squalene)	FH - 1	FH-2	FH-3	HW
Di-isobutyl phthalate	Median	2.3E-04	2.6E-04	2.8E-04	7.1E-03
	Mean	2.8E-04	3.2E-04	4.4E-04	1.5E-02
	5th percentile	0.0E+00	5.7E-05	4.2E-05	1.6E-03
	95th percentile	9.0E-04	8.6E-04	1.3E-03	8.2E-02
Squalene	Median	--	--	--	--
	Mean	--	--	--	--
	5th percentile	--	--	--	--
	95th percentile	--	--	--	--
Dioctyl terephthalate	Median	2.9E-03	1.9E-03	2.1E-03	1.5E-01
	Mean	7.9E-03	8.7E-03	9.6E-03	2.2E-01
	5th percentile	1.4E-04	1.3E-04	1.3E-04	4.2E-02
	95th percentile	3.2E-02	4.6E-02	5.6E-02	7.8E-01

Table 4-3: Percent recoveries of pollutants in wipe samples

Compounds	% FH-1	% FH-2	% FH-3	HW-1/FH-1, ratio
Octocrylene	51%	27%	22%	2.1 (0.03,11.8)
Homosalate	56%	28%	16%	1.2 (0.03,4.0)
Acetyl tributyl citrate	49%	38%	13%	1.2 (0.6,9.0)
Galaxolide	62%	22%	16%	1.7 (0,9.0)
Butyl benzyl phthalate	53%	31%	16%	3.9 (1.9,7.2)
Di-methyl phthalate	39%	31%	29%	1.1 (0.02,2.6)
Di-ethyl phthalate	39%	28%	32%	0.9 (0.5,22.4)
Di-n-butyl phthalate	47%	29%	23%	2.3 (0.7,5.2)
Bis (2-ethyl hexyl) phthalate	59%	23%	18%	0.3 (0.1,0.7)
Tris-1-chloro isopropyl phosphate	47%	36%	17%	1.8 (0.9,24.6)
Tri phenyl phosphate	58%	23%	19%	1.2 (0.8,5.8)
Di-isobutyl phthalate	46%	34%	20%	2.0 (0.9,18.8)
Squalene	52%	30%	18%	0.05 (0.006,0.8)
Dioctyl terephthalate	65%	23%	13%	2.5 (0.4,11.1)

Note: The 5th percentile and 95th percentile values are listed in parenthesis

We calculated the Pearson and Spearman rank correlation coefficients for the hand wipe (HW-1) and first forehead wipe sample (FH-1) in order to assess consistency between forehead and hand samples (Table 4-4). Octocrylene and homosalate, which are found in UV protection products, have a high Spearman correlation coefficient (0.83, $p<0.002$, 0.77 $p<0.005$) between these locations. Galaxolide, which is found in musk, also has a high Spearman correlation coefficient (0.71 $p<0.026$). We hypothesize that applying these products using hands has led to high correlation of the FH-1 and HW-1 concentrations. Di-iso butyl phthalate, bis-2-ethyl hexyl phthalate, dioctyl terephthalate and tris-1-chloro isopropyl phosphate also show a significant correlation between FH-1 and HW-1 based on their Pearson coefficients.

Table 4-4: Pearson and Spearman correlation coefficients between Forehead Wipe 1 (FH-1) and Hand Wipe 1 (HW-1)

Compound	Concentration (µg/cm^2)		Concentration (µg/µg-squalene)	
	Spearman Coefficient	Pearson Coefficient	Spearman Coefficient	Pearson Coefficient
Octocrylene	0.83 (0.002)	0.65 (0.031)	0.67 (0.023)	0.99 (<0.001)
Homosalate	0.77 (0.005)	0.96 (0)	0.49 (0.125)	1 (<0.001)
Acetyl tributyl citrate	0.4 (0.517)	0.3 (0.627)	0.7 (0.233)	0.65 (0.237)
Galaxolide	0.71 (0.015)	0.44 (0.174)	0.49 (0.125)	0.67 (0.025)
Butyl benzyl phthalate	0.26 (0.658)	0.24 (0.652)	0.2 (0.714)	0.13 (0.804)
Di-methyl phthalate	0.01 (0.979)	-0.59 (0.057)	0 (1)	0.38 (0.248)
Di-ethyl phthalate	-0.34 (0.312)	-0.38 (0.25)	-0.46 (0.151)	-0.42 (0.2)
Di-n-butyl phthalate	0.15 (0.67)	-0.04 (0.916)	-0.35 (0.298)	-0.37 (0.266)
Bis (2-ethyl hexyl) phthalate	0.65 (0.029)	0.83 (0.002)	0.34 (0.312)	0.7 (0.017)
Tri-n-butyl phosphate	-0.5 (1)	-0.66 (0.537)	-1 (0.333)	-0.89 (0.295)
Tris-1-chloro isopropyl phosphate	0.5 (0.117)	0.82 (0.002)	0.22 (0.519)	-0.04 (0.901)
Di-isobutyl phthalate	0.65 (0.029)	0.51 (0.107)	-0.07 (0.832)	0.07 (0.828)
Squalene	-0.14 (0.689)	-0.33 (0.325)	--	--
Dioctyl terephthalate	0.54 (0.089)	0.64 (0.035)	0.32 (0.34)	0.7 (0.017)

Note: p-values are listed in the parenthesis

In order to assess the likelihood of similar sources for the chemicals of interest, we applied both correlation analysis and ANOVA. We conducted an ANOVA to examine whether the concentration (µg/cm^2) are significantly different between FH-1 vs. FH-2 and FH-1 vs. FH-3. We found the levels of most compounds do not exhibit a significant difference between FH-1 and FH-2. But di-n-butyl phthalate and bis-(2-ethyl-hexyl) phthalate show a significant difference between FH-1 vs. FH-2 and FH-1 vs. FH-3. However, the same analysis on squalene-normalized chemical concentrations shows that di-methyl phthalate concentrations are significantly different between FH-1 vs. FH-2 and FH-1 vs. FH-3. Tri-n-butyl phosphate and di-ethyl phthalate concentrations also show a significant difference between FH-1 vs. FH-3. These concentration correlations suggest that compounds with the highest skin diffusion coefficients exhibit significant squalene-normalized concentration differences among consecutive wipe samples, but the data do not strongly support such a conclusion. The results of the ANOVA do not show any significant difference when normalizing the chemical concentrations by squalene concentrations, the results are shown in Appendix C, Table C-3.

We examined independently the correlation coefficients between the various chemicals in FH-1, FH-2, FH-3 and HW-1. We used both, the µg/cm^2 and µg-chemical/µg-squalene concentrations for the analysis. For all the wipe samples, octocrylene and homosalate are highly correlated, indicating that they likely have a common source. In FH-2, FH-3 and HW-1, di-methyl phthalate and di-ethyl phthalate also show a high correlation indicating those two compounds could also have a common source. Apart from the four compounds discussed we did not find any strong correlation between other chemicals in all the wipe samples (Appendix C, Table C-4A, C-4B, C-4C, C-4D).

Analysis of squalene concentrations

We attempted to deduce the depth from which a chemical is removed from skin by the skin wipe using the following two methodologies: (1) comparing measured squalene concentrations in the wipes to concentrations reported in the in literature as a function of skin depth (quantitative estimate) and (2) oxidation products of ozone-squalene reaction (qualitative evaluation)

1) Squalene concentrations implied by skin wipes compared to known squalene skin surface concentration from literature

Typical skin lipid concentrations range from 90-120 μg/cm^2 (Weschler and Nazaroff 2012). Other sources (Pappas et al., 2009) have reported that lipid concentration on the forehead can be as high as 150-300 μg/cm^2 due to high density of epidermal glands on the forehead. Based on estimates from Greene et al. (1970), squalene can on average comprise ~12.3% by mass of skin surface lipids, with mass concentration ranging from 12-14% of the total mass of skin surface lipids. In Table 4-6 we report the results of calculations performed to assess the depth of sampling of skin wipes. First, we use the literature referenced to estimate expected values of squalene in skin wipes. Next, we compare them to measured concentrations and use the ratio to calculate the skin-wipe sample depth of penetration for squalene extraction. We used the measured concentrations of squalene in skin wipes to develop a log-normal distribution of sample penetration depth and determined the geometric mean and standard deviation of this distribution. Based on the range of values reported in literature for squalene concentrations in the epidermis we inferred the following depth of sampling for each skin wipe:

Table 4-6: Squalene concentration and depth estimates of skin wipes

	Depth estimate (μm)	
Wipe	@ 150 μg/cm^2 total lipid concentration	@ 300 μg/cm^2 total lipid concentration
FH-1	1.1 (0.6-1.9)	0.6 (0.3-1.1)
FH-2	0.6 (0.4-0.9)	0.3 (0.2-0.5)
FH-3	0.4 (0.2-0.8)	0.2 (0.1-0.4)

Note: The depth estimates are additional depth from the previous wipe sample, and not the cumulative depth estimates. The values in parenthesis show the estimated depth ±1 SD

We provide a range of estimate depth of sampling, based on varying levels of skin lipids reported. It is more likely that skin lipid concentrations on the forehead are ~150 μg/cm^2, compared to 300 μg/cm^2, and we use those estimated depths in our analysis. From Table 4-6, assuming 150 μg/cm^2 of skin surface lipids, we expect that the first wipe sample typically removes chemical from about 1.1 μm of the stratum corneum, the second wipe gives us the concentration at an approximate depth of 1.7 μm and the third wipe sample gives us the concentration at a depth of 2.1 μm. This is a rough estimate and we expect that the skin wipe contributes to removal of lipids and chemicals from the top 1 μm of skin surface. Each consequent skin wipe removes lipids and chemicals from approximately an additional 0.5 μm of the stratum corneum.

2) Oxidation products of ozone and sapienic acid concentrations

The data on total squalene, squalene oxidation products, and sapienic acid also offers insight into how deep in the skin we extract chemicals when using sequential wipes. As reported by Wisthaler and Weschler (2010) squalene can react with ozone to form a variety of oxidation products in the skin surface. Ozone is not expected to penetrate much lower than the top 1 μm of

the stratum corneum (Froome et al. 2014). Squalene has six double bonds that can react with ozone to form various primary oxidation products. We screened our samples for the primary oxidation products of ozone listed in Wisthaler and Weschler (2010). Two oxidation products of ozone were detected in the LC-MS analysis: 4,9,13,17-tetramethyl-octadeca-4,8,12,16-tetraenoic acid (C-22 tetraenoic acid), and 5,9,13-trimethyl-tetradeca-4,8,12-trienoic acid (C-17-trienoic acid). The exact mass and the MS fragments of the compounds were used to confirm the detection of the compounds. Note, that we cannot quantify the compound recoveries because there are no available standards for these two compounds. However we can use the information on area under the curve as a proxy for concentration and study the decrease in concentration of oxidation products in each wipe sample.

In the literature there are reports of squalene (12.3% mass) in skin lipids and sapienic acid (5.4% mass) in lipids. This gives us a squalene/sapienic acid ratio of roughly 2 (12.3/5.4). In our wipe-sample analyses (Table 4-7), this ratio comes out at 0.9 and 0.19—values that are much lower at the skin surface and increase in subsequent wipe samples. We hypothesize that the sapienic acid-squalene ratios are lower at the surface due to the oxidation of squalene. However, the squalene/sapienic acid concentration ratios in lower layers are much higher than we expected from estimates in the literature. Ozone would not penetrate all layers of the stratum corneum, and its reactions would be limited to roughly the top 1 μm of the stratum corneum. These data also suggest of a depth profile with sequential wipes. More experimentation is needed to develop this hypothesis. Table 4-7 lists the area under the curve measured for each oxidation product, and the sapienic acid concentration in comparison to squalene concentration. These results imply that squalene at the surface is oxidized, primarily by ozone and probably by various other oxidative species that are present in the indoor environment

Table 4-7: Oxidation products of squalene and sapienic acid concentrations

	Sample 1			Sample 2			Unit
	FH-1/ FH-1	FH-2/ FH-1	FH-3/ FH-1	FH-1/ FH-1	FH-2/ FH-1	FH-3/ FH-1	
C-22 tetraenoic acid	1.00	0.52	0.43	1.00	0.51	0.61	Ratio of area under the curve
C-17 trienoic acid	1.00	0.50	0.39	1.00	0.41	0.55	Ratio of area under the curve
Sapienic acid	647	124	117	3343	1010	172	μg/wipe
Squalene	629	520	546	633	633	607	μg/wipe
Squalene/sapienic acid	0.97	4.20	4.68	0.19	0.63	3.53	Ratio

Transport of chemicals in the stratum corneum

This section explores the potential for using skin wipes as a passive sampler for estimated recent indoor exposures to chemicals. Typically, more intrusive sampling methods are needed (such as a blood/urine samples) to reflect recent exposures in different microenvironments. In addition, for chemicals such as phthalates, which have a short half-life in the human body, the sequential skin wipe samples can provide a complimentary estimate of recent exposures. Phthalates diffuse through the stratum corneum somewhat slowly such that sequential skin wipe samples in combination with bio-monitoring data on blood and urine can be used to construct a time profile

of recent exposures. Here we propose the use of chemical equilibrium partitioning ratios in combination with mass-transport modeling to estimate a metric called the equivalent time of exposure (ETE). The ETE is the amount of time a subject would have to be exposed to a constant concentration of the chemical to produce and skin-depth concentration profile corresponding to skin wipes concentrations obtained various depths in the stratum corneum.

Inherent chemical properties determine equilibrium partitioning of SVOCs among the different media indoors: such as partitioning among the gas-phase, dust-phase, skin lipids, organic surfaces. The properties that determine the partitioning of SVOCs are: the octanol-air partitioning coefficient K_{oa}, the octanol-water partitioning coefficient K_{ow}, and the air-water partitioning coefficient K_{aw}. These coefficients (e.g. K_{oa}) provide an estimate of the equilibrium partitioning concentration of chemical in one media, (e.g. octanol) compared to another (e.g. air). The octanol phase is commonly used to represent an organic phase. With the assumption that equilibrium or near equilibrium conditions prevail, partitioning relationships can be used to derive the concentration of chemical in different media from known concentrations in one media. With more detailed dynamic mass balance models these relationships can be used to infer concentrations in conditions that have not achieved equilibrium. Air-to-skin transfer of chemical can be modeled using mass transfer relationships similar to those used for air-to-soil, air-to-vegetation surfaces, and other air-to-organic phase partitioning of chemicals. The appropriate modifications to estimate a lipid-gas partitioning coefficient from the octanol-air partitioning coefficient are detailed in previous work on the topic (Weschler and Nazaroff 2014). From our wipe samples, data on skin lipid-normalized concentrations from the first forehead wipe sample (FH-1) provide an opportunity to calculate corresponding indoor gas-phase and dust concentrations by applying the equations of Weschler et al. (2012), which are summarized below. To further validate our assumption of near-equilibrium conditions, we first calculate the time to equilibrium when chemical partitions from air-to-skin lipids. The time to equilibrium in the skin lipid surface for the compounds of interest is estimated using the following equations (Weschler and Nazaroff 2010).

$$\tau_s = \frac{K_{lg} \times \delta}{v_d} \tag{4-1}$$

$$\log(K_{lg}) = 0.74 \times \log(K_{ow}) + \log(H) + \log(RT) \tag{4-2}$$

where,
τ_s = time to equilibrium (s)
K_{lg} = lipid-gas partitioning coefficient (no units)
δ = thickness of lipid film (m)
v_d = gas-phase mass transfer coefficient to skin (m/s)
K_{ow}= octanol-water partitioning coefficient (no units)
H = Henry's law constant (mol/Pa-m^3)
R = Universal gas constant (Pa-m^3/mol)
T = Temperature (K)

The thickness of lipid film is assumed to be 1 μm, and the gas-phase mass transfer coefficient from air to skin is 0.000167 m/s (6 m/h) (Weschler and Nazaroff 2010). Since we do not expect

the mass transfer coefficient to change significantly among different SVOCs, we assume a constant value for the SVOCs of interest in our study. Table 4-8 summarizes the values obtained from Equation 4-1. Here we see that the three compounds of interest for dermal modeling have a short time to equilibrium, which is of particular importance when assessing the usefulness of skin wipe samples as a passive measure over some prior period.

Table 4-8: Time to equilibrium for SVOCs

Compound	$\log(K_{lg})$	τ_s (h)
Di-methyl phthalate	1.7	8E-6
Di-ethyl phthalate	2.9	1E-4
Di-n-butyl phthalate	5.1	2E-2

We use the lipid-air partition coefficient K_{lg} and the octanol-air partition coefficient K_{oa} in combination with our wipe-based skin lipid surface concentrations to infer the indoor air and dust concentrations that correspond to skin concentrations.

$$C_{gas} = \frac{C_{lipid}}{K_{lg}} \tag{4-3}$$

$$X_{dust} = \frac{C_{gas} \times f_{om-d} \times K_{oa}}{\rho_d} \tag{4-4}$$

where,
C_{gas} = concentration of chemical in gas phase (µg/m^3-air)
C_{lipid} = concentration of chemical in skin surface lipid (µg/m^3-lipid)
X_{dust} = chemical concentration in dust (µg/g-dust)
f_{om-d} = fraction organic matter in dust
ρ_d = density of dust particles (kg/m^3)

The fraction of organic matter in dust (f_{om-d}) is assumed to be 0.20 (Shin et al. 2014), and the density of dust is assumed to be 1500 kg/m^3.

To obtain air and dust concentrations from skin surface wipes, we assume that all the chemicals in the FH-1 wipe samples were transferred via air to skin. Direct transfer for chemicals in FH wipe samples is assumed to be minimal or negligible. To ensure that the concentrations estimated here are within reasonable ranges, we compare our estimated dust concentrations to prior work that report measured dust concentrations in residential environments for our selected chemicals (Blanchard et al. 2014; Shin et al. 2014; Bonvallot et al. 2010; Rudel et al. 2013). Comparisons are illustrated in Figure 4-2. The estimated concentrations compare well for the following chemicals: di-ethyl phthalate, di-methyl phthalate and di-ethyl hexyl phthalate. The estimated concentrations are lower than measured concentrations for di-n-butyl phthalate. We compare the dust concentrations primarily to verify that the estimates of dust, and gas phase concentrations derived using partitioning modeling are reasonable and comparable to previously measured concentrations. Our estimated gas-phase concentrations are used to model the air-to-skin uptake and the corresponding concentration profile in the stratum corneum.

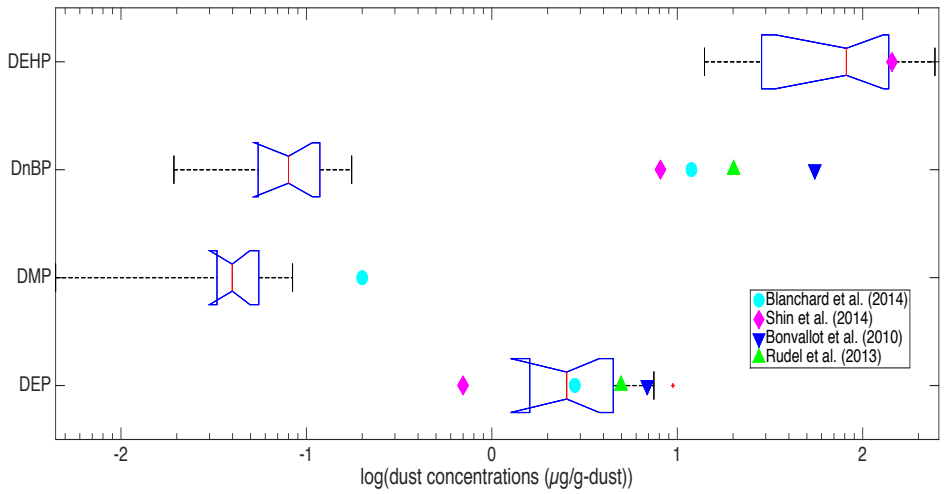

Figure 4-2: Measured versus estimated dust concentrations of di-ethyl phthalate, di-methyl phthalate, di-n-butyl phthalate and di-ethyl hexyl phthalate

Note: The red lines reflect the median of the distribution, and the box plot represent the inter quartile range (25^{th} percentile – 75^{th} percentile).

Modeling dermal uptake

Once we have made estimates of residential dust and air concentrations using partitioning modeling, we model the movement of chemical through skin using a dynamic mass transfer model in order to determine and concentration-depth profile within the stratum corneum. We conduct the analysis for each subject. In the mass-transfer model, the diffusion coefficient determines the rate of movement of chemical through skin. Limited measured data is available for diffusion coefficients. They are typically derived from permeability coefficients, which govern the rate of chemical transport through skin under steady state conditions. Many empirical models are available to calculate permeability coefficients based on fitting the variation of permeability measurement with variations of chemical properties such as K_{ow} and molecular weight (MW) (Guy and Potts 1992; Mitragotri 2003). Lian et al. (2007) analyzed the goodness of fit of various empirical models to available measured data on permeability coefficients. Based on ease of use of the equation and goodness of fit, we use the Potts and Guy (1992) model, which is also suggested for use in the United States Dermal Exposure Assessment document (USEPA 2007). We use the permeability coefficient from EPA's dermal exposure guidance (USEPA 2007) to determine the corresponding diffusion coefficient. The EPA dermal exposure model expresses permeability for a chemical migrating from water on the skin surface. The permeability constant, is the permeation rate or effective transport velocity (m/s) of a chemical through the skin, under steady state conditions. Because the EPA-estimated permeability includes an implicit estimate of the diffusion coefficient in the stratum corneum, we the EPA permeability model as a starting point to determine the diffusion coefficient of gas phase chemicals through the stratum corneum. The EPA estimated the water-based permeability coefficient with the following empirical relationship.

$$\log(k_p) = 0.71 \times \log(K_{ow}) - 0.0061 \times MW - 2.72 \quad (4\text{-}5)$$

This equation gives the permeability (in cm/s rather than m/s) of a chemical through the stratum corneum from an aqueous medium on the skin. The EPA guidance report uses this permeability coefficient determine the effective diffusion coefficient based on the following equation:

$$D_{sc} = \frac{k_p \times l}{K_{ow}} \tag{4-6}$$

where, K_{ow} = octanol-air partitioning coefficient, or the vehicle-skin partitioning coefficient
l = diffusion path length through the stratum corneum = 15 μm
k_p = Permeability coefficient determined from Equation 4-5

With a value of the diffusion coefficient available we can solve the governing equation for movement of chemical through a porous membrane such as skin:

$$\frac{\partial C}{\partial t} = -D_{sc}\frac{\partial^2 C}{\partial x^2} \tag{4-7}$$

Where,

C = concentration of pollutant in skin ($\mu g/m^3$)
D_{sc} = diffusion coefficient for a chemical through skin (m^2/s)
t = time (s)
x = depth in the stratum corneum (m) corresponding to concentration C

The equation is subject to the following conditions:
Initial conditions :

1. C (t=0, x>0) = 0
2. C (t=0, x=0) = C_0

Boundary conditions :

1. At the stratum corneum-viable epidermis surface the concentration -> 0
2. C (t, x=0) = $K_{lg} \times C_{air}$ (which is our measured surface concentration)

The stratum corneum offers the greatest resistance to movement of chemical through skin, hence we choose to focus our transport modeling on the stratum corneum. Modeling concentrations in the upper layers of the stratum corneum also allows us to compare model results to the concentrations obtained from wipe samples in order to assess other key metrics such as time to equilibrium and the mass distribution in stratum corneum under both steady state and dynamic conditions.

Given that the air and dust concentrations estimated from the skin lipid concentrations are comparable to previous studies with random samples from indoor environments, we use our air concentrations in Equation 4-7 to obtain the concentration profile in the stratum corneum. This model is used to illustrate the use of skin as a passive sampler for recent exposures. The equations were solved using a partial differential equation solver (pdepe) in MATLAB. The concentration in the stratum corneum was modeled for each subject for DEP and DnBP. We

model the concentrations for 6 hours of exposure. The concentration at the following three depths in skin are assessed: 1.1 μm, 1.7 μm and 2.1 μm.

The equivalent time of exposure (ETE), which is introduced above, was calculated from the apparent depth in skin to which the wipe samples penetrated to extract chemicals. The ETE is used to answer the following question: how long would a person have to be exposed at a fixed air concentration to develop the given concentration profile. In general, since the chemicals of interest in this study are ubiquitous in indoor environments it is reasonable to assume that the exposure is relatively constant over time. We thus only model the exposure over a small time frame (6-hours) in which the subjects are assumed to be indoors.

The difference between the modeled and measured concentrations at the three depths to which the wipe samples extracted chemicals are evaluated at various times within the model simulation. We use the root mean square error estimates to find the optimum matching depth profile and thus the corresponding ETE for the compounds.

The mean error, is calculated by taking the absolute different between the measured and modeled concentrations

$$\mathrm{ME} = \sum_i \left| C_{\mathrm{measured,i}} - C_{\mathrm{modeled,i}} \right| \tag{4-8}$$

where,
$C_{\mathrm{modeled,i}}$ = Concentration modeled using Ficks second law of diffusion (μg/m^3-lipid)
$C_{\mathrm{measured,i}}$ = Concentration measured in the skin wipe samples (μg/m^3-lipid)
i = index for depth in the stratum corneum

The mean error is minimized to find the ETE. The point of minimum inflexion for the mean error curve is used to find the optimum ETE. Across the 13 subjects we evaluate the ETE for di-ethyl phthalate, di-n-butyl phthalate and di-methyl phthalate. The ETE's, for each wipe sample and for each individual pollutant are listed in the table.

Table 4-9: ETE for DEP, DnBP and DMP

Compound	ETE (h)
Di-ethyl phthalate	4.3 ± 1.2
Di-n-butyl phthalate	> 6
Di-methyl phthalate	3 ± 0.8

SVOCs have much lower diffusion coefficients compared to VOCs, which contributes to their slow rate of movement through the skin. There is potential for significant dermal exposures to SVOCs as long as the source remains constant, as is typical in residential environments (Gong et al. 2014). The ETE indicates that for the SVOCs considered in this study, two, or three wipe samples could be indicative of exposures from 3-4 hours prior to sampling. Given the potential for air-to-skin transfer of SVOCs, the sequential wipe samples also serve as a proxy for recent inhalation exposures.

DISCUSSION

This chapter focuses on improving our understanding of dermal exposure indoors and how we can improve the utility of skin-wipes to assess indoor passive and activity-driven dermal uptake. We use a combination of analytical methods and modeling techniques to assess the transport and retention of SVOCs in the human skin surface. The role of the skin membrane as a passive personal sampler is examined. The analytical methods involved collecting wipe samples from 13 subjects. The wipe samples were analyzed for a variety of chemicals and skin surface lipids. The skin surface lipids were sampled for squalene, sapienic acid and oxidation products of ozone. We collected three consecutive wipe samples at the forehead and one hand wipe sample. The samples were collected to contrast between passive and active (contact driven) dermal uptake of chemicals. In each wipe sample we also quantified the total amount of squalene. For a limited sample size of two subjects we measured the sapienic acid and squalene-ozone oxidation product concentrations. In the forehead wipe samples, we find that concentrations of chemicals typically decrease in each wipe as we take consecutive samples. The hand wipe samples had much higher concentrations compared to forehead wipes. We found that the HW-1 concentrations were on average 1-1.5 times greater than the FH-1 concentrations. This highlights that while contact driven uptake can be a dominant exposure pathway, that passive uptake is also potentially significant. The wipe samples are reported as two different metrics $\mu g/m^2$ and µg/g-squalene.

As seen in other environmental monitoring metrics such as blood and urine samples, it was very informative to adjust the concentration of pollutant in wipe samples with a known proxy. Squalene serves as a proxy measure for the amount of lipid and lipid-dissolved chemical that is removed from the surface in each wipe sample. The measurement data allows us to construct a depth profile of concentration of chemical in each consecutive skin wipe. However, this metric has its limitations due to biological intra-person variability of squalene levels. Squalene levels also vary by location on the body. Squalene is the single largest individual molecule in terms of mass on the skin surface. Other limitations include the lack of sebaceous glands on areas such as the hand, which could skew the concentration estimates when reporting them as µg/g-squalene. The squalene-normalized concentrations highlight the importance of skin surface lipids and their role in passive uptake of chemicals from air. The skin surface lipids also react with oxidative species such as ozone in indoor environments. We studied the relative concentrations of squalene-ozone oxidation products, in wipe samples. The oxidation product concentrations were highest in the first surface wipe sample and decrease with each consecutive wipe.

We used a mass-transfer model for the stratum corneum to develop the "equivalent time of exposure" or ETE metric, and the numbers were compared to times to steady state obtained from the model. The ETE was used to highlight the potential for using skin wipes, to estimate recent personal exposures. The ETE is directly proportional to the diffusion coefficient of the pollutants. For some compounds, which are highly persistent in the lipid phase, a biomonitoring sample would provide estimate of long-term exposure. However, sequential wipe samples would reflect recent exposures more accurately. In general, the first skin wipe, which removes chemicals from about 1µm of the skin surface, can be a good indicator of prior exposures for SVOCs with $K_{lg} < 7$. With sequential skin wipes, we can estimate the personal exposures over a larger time frame (6 to 8 hours). Skin wipe sampling in conjunction with other measurements provides valuable input as an exposure assessment tool.

CHAPTER 5. Conclusions

My research objectives were to identify and address data gaps in our understanding of multiple pathways of exposure to chemicals in indoor environments. I used a combination of modeling and analytical measurements to evaluate the inhalation and passive dermal uptake pathways. I also assessed the effect of building operation parameters on indoor fate and human exposure. I present here summary of results and a discussion of significant findings from each of the three research topics that I carried out to address my research objectives.

Classification of pollutants in commercial buildings – Hazard evaluation and chemical property screening

In the first part of my dissertation research, which I present with results Chapter 2, I conducted a literature review to evaluate the reported occurrence of a number of contaminants in commercial buildings. I compared measured concentrations to regulatory health thresholds to develop hazard indices that could identify potential problem substances. The regulatory health thresholds were based on various health and perception endpoints: acute toxicity, intermediate and chronic non-cancer toxicity, cancer potency, reproductive toxicity, and odor and pungency. In addition to the health-based screening, I also evaluated the pollutants based on their chemical properties to assess their fate, partitioning, and transport in indoor environments. The screening analysis showed that 22 pollutants found in commercial buildings had reported concentrations that exceeded regulatory-health and/or odor thresholds. The chemical property screening analysis allowed me to classify pollutants into groups that rank the extent to which exposure can be altered by ventilation. The pollutants in Group 3,4&5 are most sensitive to changes in ventilation rates and pollutants in Groups 1&2 pollutants are least sensitive. The pollutants in Group 3,4&5 typically have low octanol-air partitioning coefficients and they volatile organic compounds. The pollutants in Group 1&2 have high octanol-air partitioning coefficients and they are semi-volatile organic compounds. Figure 5-1 provides a diagram of the methods and results for this screening analysis.

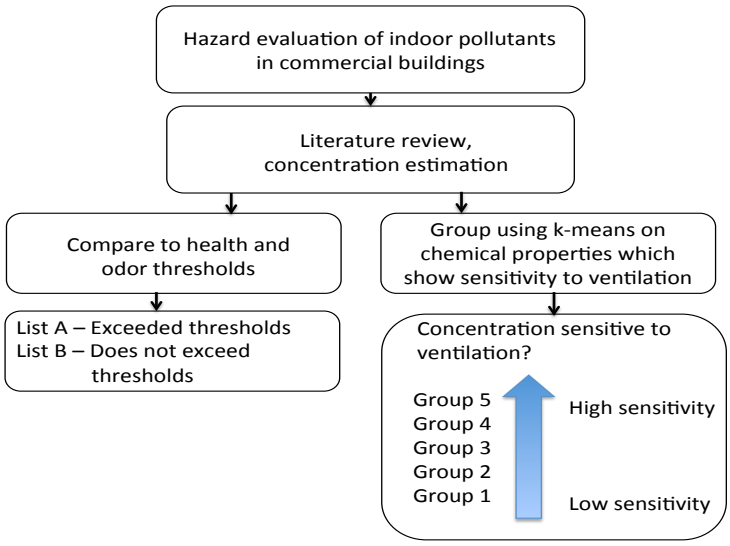

Figure 5-1: Conceptual diagram of hazard evaluation and chemical property screening for pollutants in commercial buildings

The chemicals that are in List A and Group 5, include acetaldehyde, benzene,1,3-butadiene, butyl acetate, carbon tetrachloride, chloroform, decanal, 1,4-dichloro benzene, di-chloromethane,

ethyl benzene, formaldehyde, hexanal, d-limonene, naphthalene, nonanal, octanal, pentanal, a-pinene, tetrachloroethane, tatrachloroethene, tri-chloroethylene, toluene, and m/p-xylene. The concentrations of pollutants in Groups 3 and 4 are also sensitive to changes in ventilation rates, but to a lesser extent compared to Group 5. Ventilation rates can have an impact on inhalation exposures to these compounds, provided they have indoor sources. Most of these compounds have strong indoor sources, highlighting the importance of considering sources locations and exposures for these substances in the process of setting minimum ventilation rate standards to protect public health.

Modeling fate and transport of pollutants in commercial building – Exposure assessment

In Chapter 3, I developed and applied an air-only mass-balance model and a fugacity-based mass-balance model to explore in more detail the dependence of indoor concentration and pollutant removal by ventilation and filtration. I used a well-mixed room model to study the effect of ventilation on criteria air pollutants and radon. The fugacity-based model includes multiple indoor compartments and allows for the exchange of chemical mass among air, particles, and several indoor surfaces (carpet and vinyl flooring, walls, etc.). I ran the fugacity model under a variety of scenarios (1) indoor source location of VOCs and SVOCs, with an outdoor PM source (2) outdoor source of VOCs and SVOCs with an outdoor PM source (3) indoor source of PM only. I evaluated the effect of ventilation and filtration on the exposure metrics for these compounds, by simulating the model runs under varying ventilation rates and filtration efficiencies. The exposure metrics used are inhalation intake fraction(iF) for PM and VOCs/SVOCs of indoor origin, indoor proportion of outdoor particles (iPOP) for PM of outdoor origin, and concentration ratio (CR) for VOCs/SVOCs of outdoor origin. The inhalation iF represents the fraction of the total pollutant emitted from the source that is inhaled. The iPOP and CR are the ratios of PM/pollutant concentration indoors and outdoors. The iF is a measure of intake, while the iPOP and CR are metrics of availability for uptake.

My results showed that for inorganic gases, increasing ventilation does not impact exposures since most of these pollutants typically do not have indoor sources. For particles of indoor and outdoor origin, high efficiency filters were the most effective method to manage exposures. Increased ventilation is seen to increase indoor concentrations of particles from outdoor air, however the effect of ventilation is modest when a moderate or high efficiency filter is used, since filtration removes PM coming in from outdoors. My modeling efforts showed that increased ventilation decreases indoor concentrations of indoor-origin particles, however, the effect of ventilation is modest when a moderate or high efficiency filter is used. In summary, filtration is seen to be effective at reducing exposure to both indoor and outdoor origin PM. For VOCs that exist primarily in the gas phase in air (Groups 1, 2, and 3), ventilation is the dominant removal mechanism when sources are located indoors. If the sources are located outdoors, increasing ventilation is seen to increase exposures to the pollutants. For SVOCs that partition preferentially to the particle phase in air (Groups 4 and 5), the effect of ventilation is evaluated further based on the octanol-air partitioning coefficient of the compound. For SVOCs with $9 < \log(K_{oa}) < 12$ (mostly Group 4) ventilation is seen to have a modest impact on exposures when sources are located indoors. For SVOCs with $\log(K_{oa}) > 12$ (Group 5), ventilation has a minimal impact on exposures. These compounds exist mostly in the particle phase in air, when PM are removed by filtration the total SVOC concentration is also seen to decrease. The chemical

properties of VOCs and SVOCs are seen to be parameters of importance, to model effects of ventilation rates and filtration on exposures to these compounds.

Passive dermal uptake of pollutants

The third project of my dissertation explores activity based vs. passive dermal uptake of semi-volatile organic compounds. The hypothesis of the study is that since SVOCs are ubiquitous in indoor environments, I should find a suite of SVOCs in the samples I collect. It was a random convenience sample of thirteen adults. I collected an opportunity sample from the subjects, and found a wide suite of SVOCs in all the samples. To evaluate the difference between the passive and active dermal uptake pathways by I collected wipe samples from two locations (forehead and hand) that are most likely to provide a study of the contrast between the two exposure pathways.

I analyzed the wipe samples for skin lipids: squalene, sapienic acid and two oxidation products of ozone-squalene oxidation reaction. The depth of sampling by a skin wipe is also evaluated, by comparing the measured squalene concentrations to levels reported in literature. I inferred that the first wipe sample collected removes the first layer of skin surface lipids of approximately 1 μm depth. The sequential wipes remove about 0.5 μm of the stratum corneum respectively. I also measure the levels of sapienic acid and two oxidation products of ozone-squalene reactions. I modeled the concentration depth profile in the stratum corneum for some compounds (di-methyl phthalate, di-ethyl phthalate, di-n-butyl phthalate and di-ethyl hexyl phthalate) with a combination of partitioning modeling and mechanistic modeling. I calculate the equivalent time of exposure (ETE) metric is by comparing the sampled and modeled concentration depth profile. The ETE is the amount of time to which the subject is exposed to a known air concentration of chemical. The sequential skin wipes are seen to provide estimates of exposure up to 6 hours prior for the compounds modeled depending on the diffusion coefficient.

The effort evaluate the skin lipid concentrations along with chemical concentrations in wipe samples provided me with a novel opportunity to better understand air-to-skin transfer of SVOCs. Standard practice currently is to report the skin wipe concentrations in $\mu g/cm^2$-area-wiped. Given the variation of chemical distribution across the various skin locations, normalizing the skin wipe samples by lipid concentrations provides more consistency for lipid-soluble chemicals in reporting their concentrations, especially when evaluating air-to-skin transfer of SVOCs. Dermal wipe sampling provides a lower cost and less intrusive measure of exposure compared to biomonitoring studies. When used in conjunction with other samples (dust, indoor surface wipes, air) these skin samples can provide a more complete picture of overall indoor chemical exposures of occupants.

Recommendations for future work

Scientific guidance for minimum ventilation rate standards

The previously published results of this work (Parthasarathy et al. 2012; Parthasarathy 2014) were used in a health risk assessment by Chan et al. (2014). The authors evaluate the inhalation health risks associated with changes in ventilation rates and filtration efficiencies in commercial buildings. The authors focus on office buildings, schools, retail stores and grocery stores. The inhalation risks in commercial buildings are seen to be low, although not trivial, compared to risks in residences. The primary driver of non-cancer risk in commercial buildings is PM2.5, and high efficiency air filters (MERV 8 rating and higher) are seen to be effective at lowering PM2.5

concentrations. Using high efficiency filters is shown to be a more energy efficient method to lower health risks associated with indoor particulate matter, compared to increasing ventilation rates. Minimum ventilation rate standards are based on occupant satisfaction with indoor air quality. In commercial buildings it is important to set ventilation rate standards that meet indoor air quality goals without compromising energy efficiency. The following diagram (Figure 5-2) provides an overview of for setting minimum ventilation rate standards.

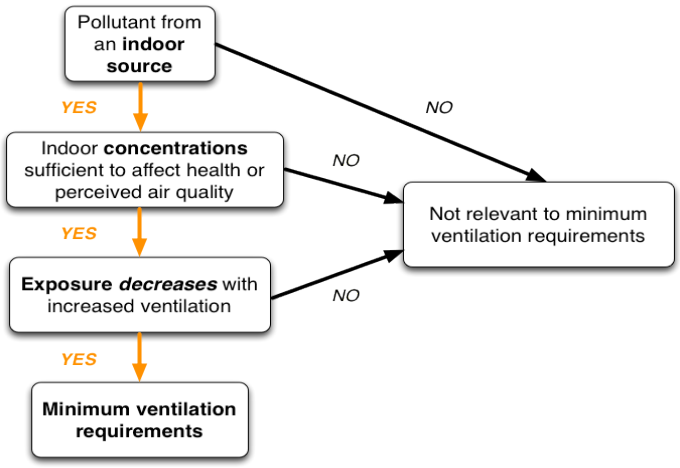

Figure 5-2: Conceptual diagram of indoor pollutants and relevance to ventilation standards

Comprehensive indoor air quality management plan

A study by Fisk et al. (2014) highlighted that commercial buildings meet or exceed current ventilation rate requirements. In spite of meeting the ventilation standards the concentrations of formaldehyde and acrolein are seen to exceed health and pungency thresholds in commercial buildings (Fisk et al. 2016). Policy makers need to employ other strategies such as air-cleaning and source reduction to maintain air quality, to balance energy efficiency requirements. Source control strategies such as the Composite Wood Products Airborne Toxic Control Measure, by California Air Resources Board (CARB) need to be adopted. The CARB measure requires composite material used indoors (flooring, hardwood, plywood particleboard) to emit low levels of formaldehyde. Hence, it important to evaluate compounds whose concentrations can exceed health and perception thresholds, evaluate associated health risks, examine their relevance to minimum ventilation rate requirements, and manage indoor air quality.

Dermal uptake pathway

From my research, the dermal uptake of pollutants is seen to be potentially significant exposure pathway. Other research has also shown that dermal uptake could be comparable to inhalation uptake of SVOCs. My study highlights the importance of considering multiple pathway exposures from a single medium (air), and a more comprehensive exposure framework to address the dermal exposure pathway. Future studies could collect sequential wipe samples from multiple locations, and evaluate the quantity of chemicals and skin lipids. More studies on ozone and other reactive oxidative species reactions with skin lipids is also needed to understand oxidative stress on skin, and how it affects exposures to multiple chemicals.

Concluding statement

In modern buildings, occupants are exposed to a suite of pollutants, VOCs and SVOCs, which are emitted by various indoor materials (furnishing, walls, flooring, cleaning products, consumer goods, paints, etc.). My research goal was to develop a better understanding of exposures to pollutants in indoor environments. My research shows that inhalation and passive dermal exposure pathways for occupants in commercial buildings and residences can contribute to uptake of pollutants from air. I use various exposure metrics to evaluate the effect of building operation parameters such as filtration and ventilation on indoor concentrations and subsequently, the inhalation intake of pollutants. I use a combination of analytical measurement and modeling to study the passive dermal uptake pathway.

My research provides insight in designing future studies to assess the health effects of SVOCs. My research can also assist to shape policy on introducing new chemicals in commerce, given our improved understanding the complexity of human interactions with indoor environments, and the relevance of these interactions to health effects. My work also highlights the need for a comprehensive exposure assessment policy with an emphasis on the role of different media and multiple exposure pathways, when evaluating new chemicals to be introduced into indoor environments.

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APPENDIX

Appendix A: Chapter 2

Table A1: VOCs of interest reported in previous studies, and their chemical properties

Compound	CAS No.	Chem. Class[1]	VP[2]	Mol.Wt[2]	Schools[3]	Other buildings[4]
			mm Hg	g/mol		
Acetaldehyde	75-07-0	Ald	902	20	CAS,LAS, 4PC	L,B,T,S,EE, A,H
Acetic acid	64-19-7	Acid	16	118		H
Acetophenone	98-86-2	Ket	0.4	120		T,D,Sh,EE, A,H
Acrolein	107-02-8	Ald	274	53		H
Acrylonitrile	107-13-1	Misc	109	53		
Benzene	71-43-2	Arom	95	80	CAS,LAS, MS	L,B,D,Ek,S, EE,A,H
Benzaldehyde	100-52-7	AromAld	1	179	CAS	T,D,S,EE,A, H
Benzyl chloride	100-44-7	HaloAro	1	127		
Bromomethane (methyl bromide)	74-83-9	Halo	1620	95		
1,3-Butadiene	106-99-0	Alke	2110	54		L,H
1-Butanol	71-36-3	Alc	7	118		B,T,EE,H
2-Butanone	78-93-3	Ket	91	80	MS,4PC	B,T,Ek,EE, H
2-Butoxyethanol	111-76-2	Gly	1	171		B,T,D,Sh,S, EE
Butylacetate	123-86-4	Est	12	126		B,T,D,EE
Butylated hydroxytoluene	128-37-0	AromAlc	0.01	265		
Butylbenzene	104-51-8	Arom	1	134		
t-Butyl methyl ether (MTBE)	1634-04-4	Ethr	250	20	LAS,	L,Ek,EE
n-Butyraldehyde	123-72-8	Ald	111	72	CAS	EE
Carbon disulfide	75-15-0	Misc	359	76		
Carbon tetrachloride	56-23-5	Halo	115	154	CAS,LAS,	L,Ek,S
Chlorobenzene	108-90-7	HaloAro	12	113		
Chloroform	67-66-3	Halo	197	119	CAS,LAS, MS	L,Ek,S,EE
Chloromethane	74-87-3	Halo	4300	50		B,Ek
Cyclohexanone	108-94-1	Ket	4	156		
p-Cymene	99-87-6	Alke	2	177	MS	
n-Decane	124-18-5	Alka	1	174		B,T,D,Sh,E E,A,H
Decanal	112-31-2	Ald	0.1	156	4PC	S,EE

Compound	CAS No.	Chem. Class[1]	VP[2]	Mol.Wt[2]	Schools[3]	Other buildings[4]
			mm Hg	g/mol		
1,2-Dichlorobenzene	95-50-1	HaloAro	1	147		
1,3-Dichlorobenzene	541-73-1	HaloAro	2	147	MS	
1,4-Dichlorobenzene	106-46-7	HaloAro	2	147	LAS	L,B,T,S,EE
Dichlorodifluoromethane	75-71-8	HaloAro	4850	121		B,Ek
1,2-Dichloroethane (ethylene dichloride)	107-06-2	Halo	79	99		
Dichloromethane (methylene chloride)	75-09-2	Halo	435	85	LAS	L,T,D,Ek,S,EE
1,2-Dichloropropane	78-87-5	Halo	53	113		
Diethyl phthalate	84-66-2	Est	0.002	298		S,A
Di(ethylene glycol) butyl ether	112-34-5	Est	0.02	162		T,EE
1,4-Dioxane	123-91-1	Ethr	38	101		
Di(propylene glycol)methyl ethers (DPGME)	34590-94-8	Ethr	1	148		T
Dodecane	112-40-3	Alka	0.1	216		B,T,D,Sh,EE,A,H
Ethanol	64-17-5	Alc	59	78		T,D,Ek
Ethyl acetate	141-78-6	Est	93	77		B,D,Ek,EE
Ethylbenzene	100-41-4	Arom	10	136	CAS,LAS,MS	L,B,D,Ek,Sh,S,EE,A,H
2-Ethyl-1-hexanol	104-76-7	Alc	0.1	183		B,T,EE
2-Ethyltoluene	611-14-3	Arom	3	120		D,H,EE
3/4-Ethyltoluene	620-14-4	Arom	3	120		EE
4-Ethyltoluene	622-96-8	Arom	3	120		B,EE
Ethylene glycol	107-21-1	Gly	0.1	19		EE
Formaldehyde	50-00-0	Ald	3890	30	CAS,LAS,4PC	L,B,T,S,EE,A,H
n-Heptane	142-82-5	Alka	46	98		D,Ek,EE,A,H
n-Hexadecane	544-76-3	Alka	0	287		Sh,EE,A,H
n-Hexane	110-54-3	Alka	151	69		B,D,Ek,S,EE,A,H
Hexanal	66-25-1	Ald	11	128	CAS,4PC	B,T,D,S,EE,A,H
Isopropylbenzene	98-82-8	Arom	5	120		
d-Limonene	5989-27-5	Alke	1	177	LAS,MS	B,T,D,Sh,S,EE,A,H
Methylcyclohexane	108-87-2	Alke	46	100		D,H,EE
Methylcyclopentane	96-37-7	Alke	138	72		D,H
3-Methylhexane	589-34-4	Alke	62	91		D,H,EE
1-Methyl-2-	872-50-4	Misc	0.3	99	4PC	EE

Compound	CAS No.	Chem. Class[1]	VP[2]	Mol.Wt[2]	Schools[3]	Other buildings[4]
			mm Hg	g/mol		
pyrrolidinone						
4-methyl-2-pentanone (MIBK)	108-10-1	Ket	20	117	MS	B,T,Ek,EE
Naphthalene	91-20-3	Arom	0.1	128	MS,4PC	B,T,S,EE,A,H
Nonanal	124-19-6	Ald	0.4	195	4PC	B,S,EE,H
Nonane	111-84-2	Alka	4	151		B,T,D,EE,H
Octane	111-65-9	Alka	14	126		B,D,Sh,EE,A,H
Octanal	124-13-0	Ald	1	174		T,S,EE,H
n-Pentadecane	629-62-9	Alka	0.003	270		Sh,EE
Pentanal (valeraldehyde)	110-62-3	Ald	26	103	CAS	B,T,D,EE,A,H
Pentane	109-66-0	Alka	514	36		D
Phenol	108-95-2	Alc	0.4	182	MS,4PC	B,T,S,EE,H
4-Phenylcyclohexene	4994-16-5	Alke	0.05	158	4PC	
α-pinene	80-56-8	Terp	5	155	LAS,MS	B,Sh,S,EE,A,H
β-pinene	127-91-3	Terp	3	166	LAS	
Propanal	123-38-6	Ald	317	58	CAS	EE
2-Propanol (isopropanol)	67-63-0	Alc	45	82		T,D,Ek,EE
2-Propanone (acetone)	67-64-1	Ket	232	56		B,T,D,Ek,S,EE,A,H
n-Propylbenzene	103-65-1	Arom	3	120		EE
Styrene	100-42-5	Arom	6	145	MS	L,B,D,Ek,S,EE,A,H
D4 Siloxane	556-67-2	Est	1	297		Sh,EE
D5 siloxane[7]	541-02-6	Est	0.2	371		T,Sh,S,EE
Terpineols	98-55-5	TerpAlc	0.04	154	4PC	S
Tetrachloroethane	79-34-5	Halo	13	168		D
Tetrachloroethene	127-18-4	Halo	19	166	CAS,LAS,MS	L,B,T,Ek,Sh,EE,A,H
n-Tetradecane	629-59-4	Alka	0.01	252		Sh,EE
Tetrahydrofuran	109-99-9	Misc	162	72	MS,	Ek
TMPD-DIB[5]	6846-50-0	Est	0.009	280		B,T,S,EE
TMPB-MIB[6]	25265-77-4	Est	0.010	244		B,EE
Toluene	108-88-3	Arom	28	111	CAS,LAS,MS,4PC	L,B,T,D,Ek,Sh,S,EE,A,H
1,2,4-Trichlorobenzene	120-82-1	HaloAro	0.5	181	MS	
1,1,1-Trichloroethane (Methyl chloroform)	71-55-6	Halo	124	133	CAS	B,T,D,S,EE

Compound	CAS No.	Chem. Class[1]	VP[2]	Mol.Wt[2]	Schools[3]	Other buildings[4]
			mm Hg	g/mol		
Trichloroethene (Trichloroethylene)	79-01-6	Halo	69	131	MS	L,D,Ek,S
Trichlorofluoromethane	75-69-4	Halo	803	137		T,D,Ek,EE
Trichlorotrifluoroethane	76-13-1	Halo	363	187		Ek
1,2,4-Trimethylbenzene	95-63-6	HaloAro	2	120	MS,4PC	B,T,D,Ek,Sh,EE
1,2,3-Trimethylbenzene	526-73-8	HaloAro	2	120		D,H,EE
1,3,5-Trimethylbenzene	108-67-8	HaloAro	2	120	MS	B,D,Ek,EE
Trimethylcyclohexenone	78-59-1	Misc	0.4	138		
2,2,5-Trimethylhexane	3522-94-9	Alka	17	124		D,H
2,2,4-Trimethylpentane	540-84-1	Alka	49	114		Ek
n-Undecane	1120-21-4	Alka	0.4	196		B,T,D,Sh,EE
o-xylene	95-47-6	Arom	8	106	CAS,LAS, MS	L,B,D,S,EE
mp-xylene	1330-20-7	Arom	8	106	CAS,LAS, MS	L,B,T,D,Ek, Sh,S,EE,A, H
1,2,3-trichlorobenzene	87-61-6	HaloAro	0.2	181	MS	
1,2,3-trichloropropane	96-18-4	Halo	4	147	MS	
β-Methacrolein	4170-30-3	Ald	30	70	CAS	
3-Methylbutyraldehyde	590-86-3	Ald	50	86	CAS	EE
2,5-Dimethylbenzaldehyde	5779-94-2	Ald	0.1	134	CAS	
4-methylbenzaldehyde	620-23-5	Ald	0.4	120	CAS	
op-tolualdehyde	529-20-4	Ald	0.4	120	CAS	
Caprolactam	105-60-2	Ket	0.002	113	4PC	EE
1,2-dichloropropane	78-87-5	Halo	53	113		
Ethenyl acetate	108-05-4	Est	90	86	4PC	
1-Butoxy-2-Propanol	5131-66-8	Alc	0.4	132		EE
1-Piperidinecarboxaldehyde	2591-86-8	Ald	0.1	204		EE
2-(2-Ethoxyethoxy) Ethanol	111-90-0	Alc	0.1	134		EE
2-Ethoxyethyl acetate	111-15-9	Acid	2	132		EE
2-Ethyl-1-hexanoic Acid	149-57-5	Acid	0.0	144		EE
2-Heptanone	110-43-0	Ket	3.9	114		EE
Benzoic Acid	65-85-0	Acid	0.001	122		EE
Hexanoic acid	142-62-1	Acid	0.044	206		EE
Longifolene	475-20-7	Ket	0.02	204		EE

Compound	CAS No.	Chem. Class[1]	VP[2]	Mol.Wt[2]	Schools[3]	Other buildings[4]
			mm Hg	g/mol		
Menthol	89-78-1	Alc	0.06	156		EE
N,N-Dibutyl Formamide	761-65-9	Amine	0.03	157		EE
N-butyl-1-Butanamine	111-92-2	Amine	3	129		EE
Nonanoic acid	112-05-0	Acid	0.002	172		EE
Propylene Glycol	57-55-6	Alc	0.1	76		EE
Tridecane	629-50-5	Alka	0.1	184		EE

Notes: 1) Alc = alcohol; Ethr = ether; Gly = glycol ether; Ket = ketone; Ald = aldehyde; Estr = acetates and other esters; Acid = carboxylic acid; Alka = alkane HC; Alke = alkene HC; Cycl = cyclic HC; Terp = terpene HC; Arom = aromatic HC; ClAro = chlorinated aromatic HC; Halo = halogenated aliphatic HC; Misc = miscellaneous category.

2) Vapor pressure and molecular weight were generated using EPISUITE

3) CAS-CADPH (2004), MS-Godwin et al. (2007), LAS-Shendell et al. (2004), 4PC-Hodgson et al. (2004)

4) L-Loh et al. (2007), B-Apte et al. (2000), T-Hotchi et al. (2007), S-SMCB (2010), D-Daisey et al. (1994), Sh-Shields et al. (1992), Ek-Eklund et al. (2007), EE-East End (2003)
5) 2,2,4-Trimethyl-1,3-pentanediol monisobutyrate (combined isomers 1 & 3)
6) 2,2,4-Trimethyl-1,3-pentanediol diisobutyrate
7) Decamethylcyclopentasiloxane

Table A2 - Comparing concentrations in other commercial buildings to health and odor-based thresholds

Compound	Conc. Used	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index
	ppb					
Acetaldehyde	54	**1.E+01**		**2.E+01**	**4.E+01**	
Acetophenone	0.9				3.E-03	1.E-03
Benzene	10	**4.E+00**	**1.E+01**	**4.E+01**	4.E-03	
Benzaldehyde	0.1				3.E-03	
Benzothiazole	0.05					
1,3-Butadiene	53	**7.E+01**		**5.E+03**	**3.E-01**	
1-Butanol	0.2				3.E-02	
2-Butanone	1.5	1.E-03				
2-Butoxyethanol	1.1	4.E-03			4.E-03	
Butylacetate	1.2				**7.E-01**	
t-Butyl methyl ether (MTBE)	0.2	3.E-04				
Carbon tetrachloride	0.01	2.E-03		**2.E-01**	2.E-06	
Chloroform	0.1	6.E-03		**2.E-01**	3.E-05	
Chloromethane	0.8	2.E-02				

Compound	Conc. Used ppb	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index
n-Decane	0.1				2.E-04	
Decanal	0.2				**6.E-01**	
1,4-Dichlorobenzene	7.5	**8.E-01**		**4.E+01**	**2.E-01**	1.E-02
Dichlorodifluoromethane	5.5					
Dichloromethane (methylene chloride)	0.2	2.E-03		6.E-02		
Diethyl phthalate	0.002					4.E-05
Di(ethylene glycol) butyl ether	0.01					
Di(propylene glycol)methyl ethers (DPGME)	1					
Dodecane	0.05				5.E-04	
Ethyl acetate	1.7				7.E-03	
Ethylbenzene	3.3	2.E-02		**6.E+00**	**6.E-01**	
2-Ethyl-1-hexanol	0.3				1.E-03	2.E-03
2-Ethyltoluene	0.01				2.E-04	
4-Ethyltoluene	0.3				4.E-02	
Formaldehyde	43	**7.E+00**		**2.E+01**	1.E-01	**6.E-01**
n-Hexadecane	0.3					
n-Hexane	3.7	2.E-02			3.E-03	
Hexanal	1.3				**2.E-01**	
d-Limonene	1.8				**1.E-01**	
Methylcyclohexane	0.01				7.E-05	
Methylcyclopentane	0.03				2.E-05	
3-Methylhexane	0.01				1.E-05	
1-Methyl-2-pyrrolidinone	0.1		2.E-03			
4-methyl-2-pentanone (MIBK)	1.8	3.E-03			1.E-02	
Naphthalene	9.4	**2.E+01**		**1.E+02**	**7.E-01**	
Nonanal	16				**5.E+01**	
Nonane	1					
Octane	0.02				1.E-05	
Octanal	0.1				**1.E+01**	
n-Pentadecane	0.05					
Pentanal (valeraldehyde)	0.05				**1.E-01**	
Pentane	0.4				3.E-04	
Phenol	0.2	4.E-03			4.E-02	

Compound	Conc. Used ppb	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index
α-pinene	0.4				2.E-02	
2-Propanone (acetone)	43	4.E-03			5.E-02	
Styrene	1.1	6.E-03			3.E-02	
D4 Siloxane	0.002					
D5 siloxane	78					
Terpineols	0.01					
Tetrachloroethane	0.01			**4.E-01**		
n-Tetradecane	0.07					
TMPD-DIB	0.003					
TMPB-MIB	0.7					
Toluene	16	**2.E-01**	**1.E-01**		**2.E-01**	
1,1,1-Trichloroethane (Methyl chloroform)	15	9.E-02				
Trichloroethene (Trichloroethylene)	0.06	6.E-04		**7.E-02**	2.E-05	
1,2,4-Trimethylbenzene	0.08				7.E-04	
1,2,3-Trimethylbenzene	0.02					
1,3,5-Trimethylbenzene	0.3				2.E-03	
2,2,5-Trimethylhexane	0.003				4.E-06	
n-Undecane	0.1				1.E-04	
o-xylene	1.3	3.E-02			4.E-03	
mp-xylene	13	**6.E-01**			**4.E-01**	
Caprolactam	21					
1-Butoxy-2-Propanol	1.6		2.E-02		1.E-02	
1-Piperidinecarboxaldehyde	1.3					
2-(2-Ethoxyethoxy) Ethanol	3.8					
2-Ethoxyethyl acetate	0.8	**3.E-01**			2.E-02	
2-Ethyl-1-hexanoic Acid	0.9					
2-Heptanone	3.9				**9.E-01**	
Benzoic Acid	4					
Hexanoic acid	1				**1.1.E+00**	
Longifolene	1.7					
Menthol	0.9					
N,N-Dibutyl Formamide	5.3					
N-butyl-1-Butanamine	25					
Nonanoic acid	0.9					

Compound	Conc. Used ppb	Non-cancer toxi. Index	Rep. toxi. Index	Cancer toxi. Index	Odor Index	Pungency Index
Propylene Glycol	3.9	**5.E-01**				
Tridecane	1.3					

Note: Conc. - Concentration

Appendix B: Chapter 3

Mass balance model

The Bennett-Furtaw fugacity-based indoor-fate model was adapted for this study to provide a dynamic chemical mass-balance model for commercial buildings (Bennett and Furtaw 2004). We provide below the relevant equations and inputs used in the model. The overall mass balance scheme is shown in Figure B1. The model tracks chemical inventory in a set of relevant indoor compartments such that the mass of the chemical pollutant and particles in the room were treated as state variables. The model was set-up and run using Matlab (differential equations were solved using ode15s). Preliminary mass balance equation set-up to assess the concentration of the chemical pollutants in the room is as follows. Flux between compartments such as walls and floors are driven by advective and diffusive processes. The model also incorporated the flow of particulate matter from outdoor air, the flow of PM is modeled along with chemical flow and equilibrium

Table B1: Building parameters obtained by ranking individual parameters for various buildings

Room configurations	Office	Retail	Grocery	Schools	Units
Total floor area (A)	1645	3716	790	895	m^2
Height of room (h)	3.7	7	3.6	3	m
$PM_{10\text{-}o}$ (outdoor concentration)	26.8	23.6	23.6	19.6	$\mu g/m^3$-air
$PM_{2.5\text{-}o}$ (outdoor concentration)	12.1	9.6	12.5	13.6	$\mu g/m^3$-air
ACH_i	0.56	0.75	1.16	0.6	h^{-1}
ACH_r	1.4	3	0	3	h^{-1}
% area of carpeted flooring (f_c)	80%	80%	80%	80%	no units
% area of vinyl flooring (f_v)	20%	20%	20%	20%	no units

Table B2 : Parameters used in model

Gas constant – R	8.314	J/mol K	Shin et al. (2012)
Temperature - T	298	K	Shin et al. (2012)
Boundary layer thickness - δ_{bl}	0.026	m	Shin et al. (2012)
Carpet layer thickness - δ_{car}	0.016	m	Shin et al. (2012)
Vinyl layer thickness - δ_{vin}	0.0018	m	Shin et al. (2012)
Thicnkess of organic film - δ_{film}	1.00E-07	m	Bennett and Furtaw (2004)
Thicnkess of wall - δ_{wall}	0.015	m	Shin et al. (2012)
Density of organic film - ρ_{film}	826	kg/m^3	Bennett and Furtaw (2004)
Diffusion coefficient of gases, D_{ab}	0.023	m^2/h	Parthasarathy et al. (2011)
Density of dust loading on vinyl - ρ_v	8.50E-05	$\mu g/m^2$	Bennett and Furtaw (2004)

Density of dust loading on carpet - ρ_{car}	0.01	$\mu g/m^2$	Bennett and Furtaw (2004)
Density of particles - ρ_d	1500	kg/m^3	Bennett and Furtaw (2004)

Deposition rates of particles - T_d			
Bin 1 : 10 µm - 2.5 µm - $T_{d,1}$	6.50E-01	h^{-1}	Siegel et al. (2013)
Bin 2 : 2.5 µm - 1.0 µm - $T_{d,2}$	3.90E-01	h^{-1}	Siegel et al. (2013)
Resuspension rates of particles - T_r			
Bin 1 : 10 µm - 2.5 µm - $T_{r,1}$	7.00E-06	h^{-1}	Bennett and Furtaw (2004)
Bin 2 : 2.5 µm - 1.0 µm – $T_{r,2}$	5.00E-07	h^{-1}	Bennett and Furtaw (2004)
Fraction of organic carbon - $f_{oc,j}$			
Bin 1 : 10 µm - 2.5 µm - $f_{oc,1}$	0.3	no unit	Bennett and Furtaw (2004)
Bin 2 : 2.5 µm - 1.0 µm - $f_{oc,2}$	0.3	no unit	Bennett and Furtaw (2004)
Dust loading on carpet : Fractions			
Bin 1 : 10 µm - 2.5 µm	0.06	no unit	Shin et al. (2012)
Bin 2 : 2.5 µm - 1.0 µm	0.02	no unit	Shin et al. (2012)
Dust loading on vinyl : Fractions			
Bin 1 : 10 µm - 2.5 µm	0.09	no unit	Shin et al. (2012)
Bin 2 : 2.5 µm - 1.0 µm	0.04	no unit	Shin et al. (2012)
Cleaning efficiency			
Carpet	0.008	d^{-1}	Shin et al. (2012)
Vinyl	0.06	d^{-1}	Shin et al. (2012)

Table B3

Efficiency of particulate air filter	MERV 6	MERV 8	MERV 13	Source
Bin 1 : 10 µm - 2.5 µm	0.70	0.81	0.87	Fisk et al. (2002)
Bin 2 : 2.5 µm - 1.0 µm	0.20	0.41	0.70	Fisk et al. (2002)

Basic equations
Volume, $V = A \times h$
Total air change rate, $ACH = ACH_i + ACH_r$

Fugacity capacity calculation
The fugacity capacities for the various compartments are first listed:
Pure air : $Z_{air} = \frac{1}{R \times T}$
Where, R is the universal gas constant, T is the standard room temperature in K

Particles in air : $Z_{ap,j} = \frac{K_{p,j} \times \rho_d \times 10^9}{R \times T}$

Where, $Z_{ap,j}$ is the fugacity capacity of the particles in each size bin, j
$\log(K_{p,j}) = \log(K_{oa}) + \log(\frac{f_{oc,j}}{0.74}) - 11.91$, $K_{p,j}$ is the particle air partition coefficient ($m^3/\mu g$),

Total air : $Z_{airT} = \sum_j \left(\frac{Z_{ap,j} \times \rho_{p,j}}{\rho_d \times 10^9}\right) + Z_{air}$
Where, $\rho_{p,j}$ = mass concentration of particles in size bin j, calculated from mass balance of particles indoors

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Carpet : $Z_{carpet} = \frac{K_{ca}}{R \times T}$
$K_{ca} = 10^{3.82 - 0.62 \times \log(VP)}$, K_{ca} is the carpet-air partitioning coefficient
VP = Vapor pressure of pollutant in Pa

Particles on carpet : $Z_{cd} = \sum_j Z_{ap,j} \times f_{c,j}$
Where, $f_{c,j}$= fraction of particles settled on carpet in size bin j
Total carpet : $Z_c = \frac{Z_{carpet} \times \delta_c + Z_{cd} \times \frac{\rho_{car}}{\rho_d}}{\delta_c + \frac{\rho_{car}}{\rho_d}}$
Where, ρ_{car} = particle loading on carpet in $\mu g/m^2$

Vinyl : $Z_{vinyl} = \frac{K_v}{R \times T}$
$K_{va} = 10^{5.2 - 0.68 \times \log(VP)}$, K_{va} is the vinyl-air partitioning coefficient

Particles on vinyl : $Z_{vd} = \sum_j Z_{ap,j} \times f_{v,j}$
Where, $f_{v,j}$= fraction of particles settled on vinyl in size bin j
Organic film : $Z_{film} = \frac{0.48 \times K_{ow} \times f_{ocf} \times \rho_{film}}{H \times 1000}$
Where, f_{ocf} is the fraction of organic carbon in film

Total vinyl : $Z_v = \frac{Z_{film} \times \delta_{film} + Z_{vinyl} \times \delta_v + Z_{vd} \times \frac{\rho_v}{\rho_d}}{\delta_{film} + \delta_v + \frac{\rho_v}{\rho_d}}$
Where, ρ_v = particle loading on vinyl in $\mu g/m^2$

Mass balance equations and transfer factor calculations

Mass balance for pollutant in air

$$\frac{dM_a}{dt} = ACH \times M_{out} + T_{va}M_v + T_{wa}M_w + T_{ca}M_c + M_{indoor-source} - ACH \times M_a - T_{av}M_a - T_{aw}M_a - T_{ac}M_a$$

Mass balance for pollutant in carpet

$$\frac{dM_c}{dt} = T_{ac}M_a - T_{ca}M_c - \theta k_{cl} M_c$$

Mass balance for pollutant in vinyl

$$\frac{dM_v}{dt} = T_{av}M_a - T_{va}M_v - k_{cl} M_v$$

Mass balance for pollutant in walls

$$\frac{dM_w}{dt} = T_{av}M_a - T_{va}M_v - k_{cl} M_v$$

Mass balance for particles in air

$$\frac{dM_{pm-a}}{dt} = ACH \times M_{pm-oa} + T_{r-va}M_{pm-v} + T_{r-ca}M_{pm-v} - ACH_{i+r}M_{pm-a} - T_d (M_{pm-a} + M_{pm-a})$$

Mass balance for particles in carpet

$$\frac{dM_{pm-c}}{dt} = T_{d-ac}M_{pm-a} - T_{r-ca}M_{pm-c} - \theta k_{cl} M_{pm-c}$$

Mass balance for particles in vinyl

$$\frac{dM_{pm-v}}{dt} = T_{d-av}M_{pm-a} - T_{r-va}M_{pm-v} - k_{cl} M_{pm-v}$$

Mass of particles removed by filter

$$M_{pm-f} = ACH_i M_{pm-oa} + ACH_r M_{pm-a} - \eta (ACH_i M_{pm-oa} + ACH_r M_{pm-a})$$

where,

M_{out} = mass of pollutant entering room from outdoors (µg)
M_v = mass of pollutant in vinyl (µg)
M_c = mass of pollutant in carpet (µg)
M_w = mass of pollutant in wall (µg)
M_a = mass of pollutant in room air (µg)
$M_{indoor-source}$ = Indoor source of pollutant, where applicable (µg/h)
ACH = ventilation rate in room, comprising of filtration and ventilation (h-1)
M_{pm-oa} = mass of PM entering room from outdoors (µg)
M_{pm-v} = mass of PM on vinyl (µg)
M_{pm-c} = mass of PM on carpet (µg)
M_{pm-a} = mass of PM in room air (µg)

$M_{indoor-source}$ = Indoor source of pollutant, where applicable (µg/h)

Transfer factors for the compartments are comprised of an advective and diffusive component

T_{ac} is the transfer factor from air to carpet

$$T_{ac} = f_c\left(\frac{Y_{ac}}{Z_{airT}h} + \frac{v_v}{h} \times \frac{Z_{ap}}{Z_{airT}} \times \frac{\rho_{pm}}{\rho_d}\right)$$

$$Y_{ac} = \begin{cases} 10^{-1.64-0.31\times\log(VP)} \text{ if } VP > 35.4 \text{ Pa} \\ \frac{D_{air}Z_{air}}{\delta_{bl}} \text{ if } VP \leq 35.4 \text{ Pa} \end{cases}$$

ρ_{pm} is the mass concentration of particles in air (µg/m^3)

$$\rho_{pm} = \frac{M_{pm-a}}{V}$$

T_{av} is the transfer factor from air to vinyl

$$T_{av} = f_v\left(\frac{Y_{av}}{Z_{airT}h} + \frac{v_v}{h} \times \frac{Z_{ap}}{Z_{airT}} \times \frac{\rho_{pm}}{\rho_d}\right)$$

$$Y_{av} = \begin{cases} 10^{-2.83-0.33\times\log(VP)} \text{ if } VP > 0.16 \text{ Pa} \\ \frac{D_{air}Z_{air}}{\delta_{bl}} \text{ if } VP \leq 0.16 \text{ Pa} \end{cases}$$

T_{aw} is the transfer factor from air to wall

$$T_{av} = \left(\frac{Y_{aw}}{Z_{airT}(\sqrt[0.25]{A})}\right)$$

$$Y_{aw} = \begin{cases} 10^{-1.77-0.35\times\log(VP)} \text{ if } VP > 15.7 \text{ Pa} \\ \frac{D_{air}Z_{air}}{\delta_{bl}} \text{ if } VP \leq 15.7 \text{ Pa} \end{cases}$$

T_{ca} is the transfer factor from carpet to air

$$T_{ca} = \left(\frac{Y_{ac}}{Z_c\delta_c} + \frac{v_{rc}}{\delta_c} \times \frac{Z_{cp}}{Z_c} \times \frac{\rho_{pm-c}}{\rho_d}\right)$$

$$Y_{ac} = \begin{cases} 10^{-1.64-0.31\times\log(VP)} \text{ if } VP > 35.4 \text{ Pa} \\ \frac{D_{air}Z_{air}}{\delta_{bl}} \text{ if } VP \leq 35.4 \text{ Pa} \end{cases}$$

ρ_{pm} is the mass concentration of particles on carpet (µg/m^2)

$$\rho_{pm-c} = \frac{M_{pm-c}}{A_c}$$

T_{va} is the transfer factor from vinyl to air

$$T_{va} = \left(\frac{Y_{av}}{Z_v \delta_v} + \frac{v_{rv}}{\delta_v} \times \frac{Z_{vp}}{Z_v} \times \frac{\rho_{pm-v}}{\rho_d}\right)$$

$$Y_{ac} = \begin{cases} 10^{-1.64-0.31 \times \log(VP)} & \text{if } VP > 35.4 \text{ Pa} \\ \frac{D_{air} Z_{air}}{\delta_{bl}} & \text{if } VP \leq 35.4 \text{ Pa} \end{cases}$$

ρ_{pm-v} is the mass concentration of particles on vinyl ($\mu g/m^2$)

$$\rho_{pm-v} = \frac{M_{pm-v}}{A_c}$$

T_{wa} is the transfer factor from wall to air

$$T_{wa} = \left(\frac{Y_{aw}}{Z_w \delta_w}\right)$$

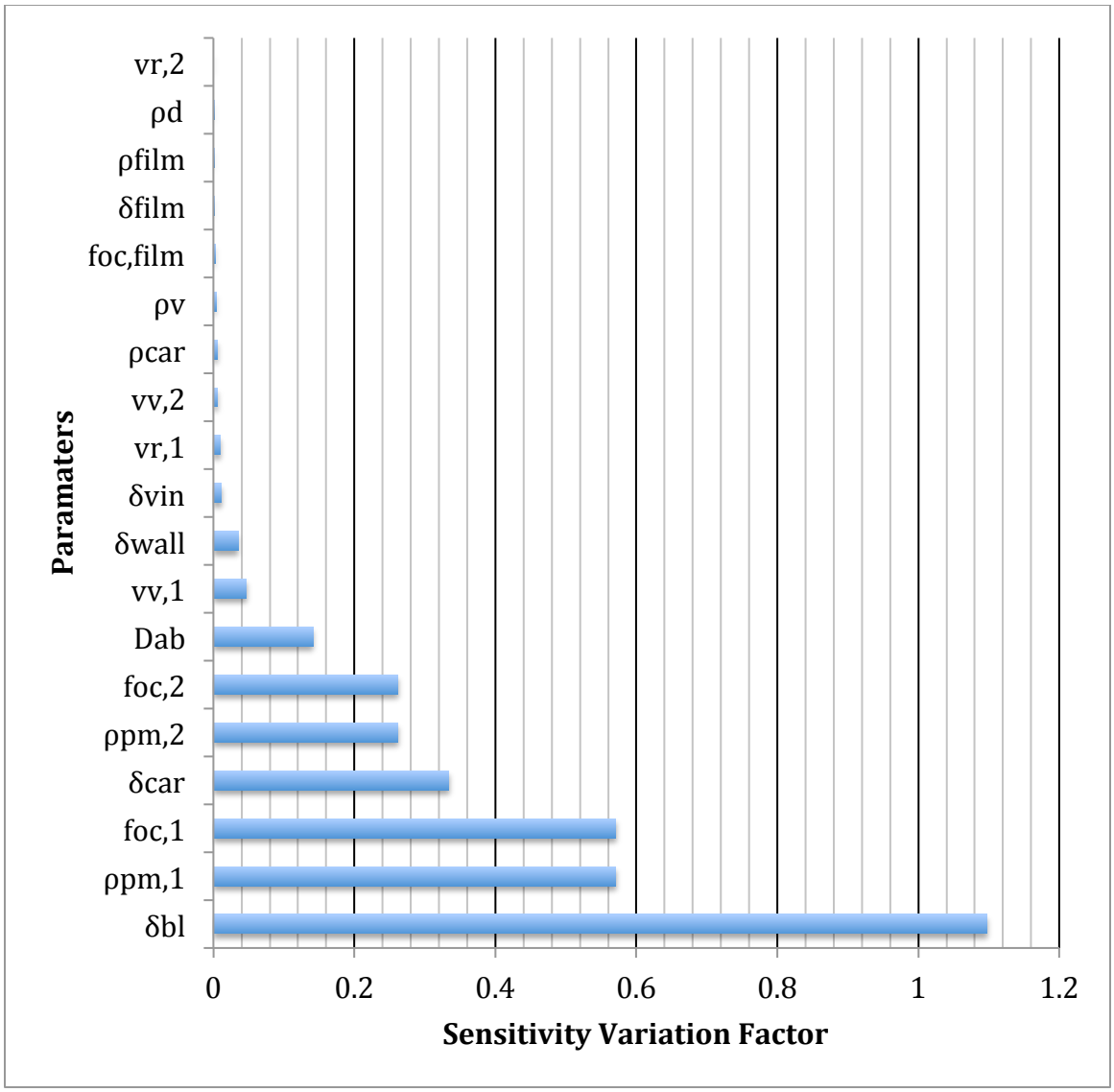

Figure B1: The Sensitivity Variation Factor (SVF), for various parameters in the model. Parameters with lower SVF are not shown in the Figure.

Appendix C: Chapter 4

Wipe sampling protocol for sample collection

This protocol described the step-by-step procedure to collect wipe samples from participants
The following equipment are needed for wipe sample collection

1. MG Chemicals Wipe Cleanroom Cotton Dry, Pack of 100 wipes, 4x4", Allied Electronics
2. Phthalate free glass bottle for isopropyl alcohol (reagent grade)
3. Aluminum foil
4. Phthalate free latex gloves
5. Labels
6. Forceps, one pair
7. Phthalate free plastic bags for storing samples
8. Cooler for storing samples

The wipes and aluminum foil are pre-cleaned using the following procedure
Foil cleaning procedure

1. Wipe the aluminum foil clean using isopropyl alcohol
2. Hang the foil out to dry for 12 hours
3. Place dry foil in phthalate free plastic bags

Wipe cleaning procedure

1. The wipes (60 wipes) are placed in the Soxhlet extractor using forceps
2. Add 1:1 mixture of hexane and acetone, extract for 24 hours
3. Transfer wipes to pre-cleaned aluminum foil and dry in the oven
4. Place wipes in foil, wrap and place in phthalate free plastic bags

The study team member collects the samples from subjects in their homes
Preparing work surface in subjects home

1. Study team member will place a square sheet of aluminum foil on a flat surface

Collection of hand wipe

1. The participants are asked to refrain from washing their hand atleast 1 hour prior to sampling
2. Study team member puts on a new pair of phthalate-free latex gloves for each subject
3. Saturate Wipe#1 with isopropyl alcohol from glass bottle
4. Apply gentle pressure, wipe the participants palm. Wipe only the palm side of the hand, and the area between the fingers.
5. Fold the wipe and wrap it in foil. Place the foil in labeled plastic bag and place in cooler for transport back to lab. The wipes are stored at -20 °C until analysis.

Collection of forehead Wipes

1. Study team member puts on new set of phthalate-free latex gloves for each subject.
2. Saturate Wipe#2 with isopropyl alcohol from glass bottle
3. Apply gentle pressure, wipe the participants forehead.
4. Fold the wipe and wrap it in foil. Place the foil in labeled plastic bag and place in

cooler for transport back to lab. The wipes are stored at -20 °C until analysis.

5. Repeat steps 2 through 5 for Wipe #3 and Wipe #4.

Blank samples

1. Blanks should account for atleast 5% of total samples taken.
2. Study team member puts on new set of phthalate-free latex gloves for each subject.
3. Saturate Wipe #5 with isopropyl alcohol
4. Wipe the gloved hand of the study team member
5. Fold the wipe, and wrap it in foil. Place foil in a Ziploc bag (or an inert brown glass bottle) and place in cooler for transport back to the lab. The wipes should be frozen at -20 °C until analysis.

Table C1: Operating conditions for Liquid Chromatography/Quantitative Time of Flight Mass Spectrometry

LC-QTOF-MS Method		Agilent 6530 Accurate-Mass Q-TOF LC/MS
Injection Volume		10 µL
LC Settings		
Mobile Phases	A (pos)	miliQ water + 0.1% formic acid
	B (pos)	acetonitrile + 0.1% formic acid
	A (neg)	miliQ water + 1 mM ammonium fluoride
	B (neg)	acetonitrile
Solvent Flow		0.35 mL/min
Gradient		2% B for 1.5 min
		2%-100% B in 15 min
		100% B for 5 min
		equilibration to initial conditions for 3 min
Column		Zorbax Eclipse Plus (100 mm length, 2.5 mm ID, 1.8 µm particle size)
Column Temperature		30°C
MS Settings		
Gas Temperature		300 °C
Drying Gas Flow		12 l/min
Nebulizer		25 psig
Sheath Gas Temperature		350 °C
Sheath Gas Flow		11 l/min
Vcap		3500 (pos), 3000 (neg)
Fragmentor		110 V
Scan Range		50-1050 m/z
Scan Speed		4 spectra/s

All-Ions Acquisition		Collision Energy (CE): 0, 10, 20, 40
Reference Mass Correction		pos: none, neg: masses 112.9855, 1033.9881

Table C2: Operating conditions for Gas Chromatography/Quantitative Time of Flight Mass Spectrometry

GC-QTOF-MS Method	Agilent 7200 Accurate-Mass Q-TOF GC/MS
GC-EI-MS Method	
Injection Volume	2.5 µL
Injection Mode	splitless
Purge Flow to Split Vent	33 mL/min at 0.75 min
Inlet Temperature	280 °C
GC Settings	
Column	HP-5MS (30m x 0.25mm, 025 µm)
Initial Oven Temperature	35 °C, hold 3 min
Ramp 1	8°C/min to 325 °C, hold 3 min
Optimized He Flow for RT locking	0.776 mL/min, constant flow
Transfer Line Temperature	280 °C
MS Settings	
N2 Collision Gas	1.5 ml/min
Source Temperature	300 °C
Emission Current Filament	35 µA
Electron Energy	70 eV
Scan Range	35-1000 m/z
Scan Speed	4 spectra/sec
Reference Mass Correction	internal mass correction after every second sample

Table C3: ANOVA p-values for FH-1 versus FH-2 and FH-1 versus FH-3

Compound	Concentration (μg/m^2)		Concentration (μg/μg-squalene)	
	FH-1 vs. FH-2	FH-1 vs. FH-3	FH-1 vs. FH-2	FH-1 vs. FH-3
Octocrylene	0.90	0.51	0.54	0.97
Homosalate	0.92	0.62	0.47	0.82
Galaxolide	0.51	0.28	0.71	0.75
Di-methyl phthalate	0.19	0.20	0.08	0.10
Di-ethyl phthalate	0.41	0.23	0.19	0.08
Di-n-butyl phthalate	0.07	0.01	0.37	0.22
Bis (2-ethyl hexyl) phthalate	0.01	0.00	0.25	0.55
Tri-n-butyl phosphate	0.37	0.93	0.33	0.01
Tris-1-chloro isopropyl phosphate	0.49	0.30	0.46	0.47
Di-isobutyl phthalate	0.24	0.09	0.70	0.24
Squalene	0.01	0.00	--	--
Dioctyl terephthalate	0.29	0.17	0.88	0.76

Table C4-A: Correlation coefficients for FH-1 concentrations in µg/m^2

Compound	Octocrylene	Homosalate	Galaxolide	DMP	DEP	DnBP	DEHP	Tri-n-butyl phosphate	DiBP	Squalene	Dioctyl terephthalate
Octocrylene	1.00	1.00	-0.08	0.90	0.21	-0.17	-0.19	-0.17	-0.27	0.83	-0.22
Homosalate	1.00	1.00	-0.08	0.90	0.21	-0.17	-0.19	-0.17	-0.26	0.84	-0.21
Galaxolide	-0.08	-0.08	1.00	-0.02	-0.24	-0.04	0.30	0.33	-0.11	-0.08	0.07
DMP	0.90	0.90	-0.02	1.00	0.26	0.09	-0.08	-0.12	-0.03	0.93	-0.07
DEP	0.21	0.21	-0.24	0.26	1.00	0.00	-0.17	0.04	0.02	0.29	0.59
DnBP	-0.17	-0.17	-0.04	0.09	0.00	1.00	0.02	0.00	0.13	0.12	-0.11
DEHP	-0.19	-0.19	0.30	-0.08	-0.17	0.02	1.00	0.01	0.22	0.11	-0.15
Tri-n-butyl phosphate	-0.17	-0.17	0.33	-0.12	0.04	0.00	0.01	1.00	-0.05	-0.28	-0.08
DiBP	-0.27	-0.26	-0.11	-0.03	0.02	0.13	0.22	-0.05	1.00	0.10	0.29
Squalene	0.83	0.84	-0.08	0.93	0.29	0.12	0.11	-0.28	0.10	1.00	-0.13
Dioctyl terephthalate	-0.22	-0.21	0.07	-0.07	0.59	-0.11	-0.15	-0.08	0.29	-0.13	1.00

Table C4-B: Correlation coefficients for FH-2 concentrations in µg/m^2

Compound	Octocrylene	Homosalate	Galaxolide	DMP	DEP	DnBP	DEHP	Tri-n-butyl phosphate	DiBP	Squalene	Dioctyl terephthalate
Octocrylene	1.00	1.00	-0.06	0.58	0.83	-0.22	-0.28	-0.13	-0.16	0.44	-0.17
Homosalate	1.00	1.00	-0.06	0.58	0.83	-0.22	-0.28	-0.13	-0.16	0.43	-0.17
Galaxolide	-0.06	-0.06	1.00	-0.25	0.10	-0.04	0.72	0.34	-0.08	0.12	0.03
DMP	0.58	0.58	-0.25	1.00	0.74	0.38	-0.21	-0.18	0.46	0.47	0.01
DEP	0.83	0.83	0.10	0.74	1.00	0.23	-0.19	0.10	-0.05	0.51	-0.05
DnBP	-0.22	-0.22	-0.04	0.38	0.23	1.00	-0.32	-0.01	0.18	-0.16	0.09
DEHP	-0.28	-0.28	0.72	-0.21	-0.19	-0.32	1.00	0.35	0.25	0.24	0.02
Tri-n-butyl phosphate	-0.13	-0.13	0.34	-0.18	0.10	-0.01	0.35	1.00	-0.01	0.55	-0.08
DiBP	-0.16	-0.16	-0.08	0.46	-0.05	0.18	0.25	-0.01	1.00	0.42	0.28
Squalene	0.44	0.43	0.12	0.47	0.51	-0.16	0.24	0.55	0.42	1.00	-0.17
Dioctyl terephthalate	-0.17	-0.17	0.03	0.01	-0.05	0.09	0.02	-0.08	0.28	-0.17	1.00

Table C4-C: Correlation coefficients for FH-3 concentrations in µg/m^2

Compound	Octocrylene	Homosalate	Galaxolide	DMP	DEP	DnBP	DEHP	Tri-n-butyl phosphate	DiBP	Squalene	Dioctyl terephthalate
Octocrylene	1.00	1.00	-0.08	0.23	0.16	-0.01	-0.28	-0.08	-0.19	0.65	-0.14
Homosalate	1.00	1.00	-0.08	0.23	0.16	-0.01	-0.29	-0.09	-0.19	0.64	-0.14
Galaxolide	-0.08	-0.08	1.00	0.00	0.00	-0.24	0.29	0.18	-0.16	-0.10	-0.01
DMP	0.23	0.23	0.00	1.00	0.74	0.58	-0.28	0.13	-0.02	-0.06	0.12
DEP	0.16	0.16	0.00	0.74	1.00	0.40	0.22	0.51	0.24	0.37	0.35
DnBP	-0.01	-0.01	-0.24	0.58	0.40	1.00	-0.20	0.06	0.16	-0.19	0.12
DEHP	-0.28	-0.29	0.29	-0.28	0.22	-0.20	1.00	0.36	0.29	0.16	0.20
Tri-n-butyl phosphate	-0.08	-0.09	0.18	0.13	0.51	0.06	0.36	1.00	0.11	0.52	-0.07
DiBP	-0.19	-0.19	-0.16	-0.02	0.24	0.16	0.29	0.11	1.00	0.09	0.41
Squalene	0.65	0.64	-0.10	-0.06	0.37	-0.19	0.16	0.52	0.09	1.00	0.04
Dioctyl terephthalate	-0.14	-0.14	-0.01	0.12	0.35	0.12	0.20	-0.07	0.41	0.04	1.00

Table C4-D: Correlation coefficients for HW-1 concentrations in µg/m^2

Compound	Octocrylene	Homosalate	Galaxolide	DMP	DEP	DnBP	DEHP	Tri-n-butyl phosphate	DiBP	Squalene	Dioctyl terephthalate
Octocrylene	1.00	0.83	-0.55	-0.35	-0.19	0.17	0.06	0.31	-0.09	0.25	-0.14
Homosalate	0.83	1.00	-0.40	-0.55	-0.11	-0.25	-0.15	0.05	-0.10	-0.16	-0.31
Galaxolide	-0.55	-0.40	1.00	-0.12	0.01	-0.52	-0.16	0.22	-0.30	-0.21	0.47
DMP	-0.35	-0.55	-0.12	1.00	0.60	0.26	0.09	-0.12	0.41	0.22	0.25
DEP	-0.19	-0.11	0.01	0.60	1.00	-0.03	0.11	0.10	0.45	0.16	0.37
DnBP	0.17	-0.25	-0.52	0.26	-0.03	1.00	0.34	0.40	0.43	0.78	0.20
DEHP	0.06	-0.15	-0.16	0.09	0.11	0.34	1.00	0.06	0.13	0.20	0.24
Tri-n-butyl phosphate	0.31	0.05	0.22	-0.12	0.10	0.40	0.06	1.00	-0.01	0.79	0.57
DiBP	-0.09	-0.10	-0.30	0.41	0.45	0.43	0.13	-0.01	1.00	0.07	0.29
Squalene	0.25	-0.16	-0.21	0.22	0.16	0.78	0.20	0.79	0.07	1.00	0.33
Dioctyl terephthalate	-0.14	-0.31	0.47	0.25	0.37	0.20	0.24	0.57	0.29	0.33	1.00

Figure C1: Concentrations of pollutants in wipe samples