보건학박사 학위논문

Secondhand Smoke in Living Environments of Korea: Exposure, Health Risks, and Impacts of Smoke-free Regulation

한국의 생활환경에서 간접흡연: 노출, 건강 위해 및 실내 금연정책의 효과

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Abstract

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Secondhand smoke (SHS) exposure is causally linked to many adverse health effects in adults and children. There is no risk-free level of SHS exposure. To reduce SHS exposure, many countries have implemented smoke-free regulations in indoor public places. In hospitality venues such as restaurants and bars, the Korean government has granted gradual implementation of smoke-free regulations based on the size of the establishment. These regulations were implemented for restaurants and bars $\geq$150 m^2 starting July 1, 2013. However, the effects of reduction of SHS exposure due to the implementation of the smoke-free regulations have not been established. Scientific evidence of the impact of smoke-free regulations is essential to support expansion of smoke-free policies to other indoor public places.

Although smoke-free regulations have been implemented in indoor public places, such regulations might not be applicable to residences. Home environments are sources of SHS exposure. Even in smoke-free homes, residents could be exposed to SHS because of tobacco smoke migrating

between neighboring units in multi-unit housing (MUH) via a process known as "SHS incursion." Although the risks of SHS exposure in smoke-free MUH homes have received increased attention, little information on residents' exposure and health effects due to SHS incursion were available. The main objectives of this study were to determine the effects of smoke-free regulations in indoor public places and to establish scientific evidence regarding the risks of exposure and the health effects due to SHS incursion into smoke-free homes.

In the first study, the effects of smoke-free regulations in restaurants and bars were examined in terms of air quality, biomarker levels, and health effects on staff. This study measured indoor fine particles ($PM_{2.5}$) in 146 hospitality venues, and urinary cotinine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) levels in 101 non-smoking staff members in 77 hospitality venues, before and 1 month after the regulations were implemented. Self-reported respiratory and sensory symptoms (i.e., eye/nose/throat irritation) were measured during both phases. In total, 121 venues and 95 non-smoking staff members in 71 venues were included in the final analysis. The geometric mean (GM) of indoor $PM_{2.5}$ concentrations was significantly reduced in bars ≥150 m^2, from 93.2 μg/m^3 (geometric standard deviation, GSD = 2.2) before regulation to 55.3 μg/m^3 (GSD = 2.2) after regulation ($p<0.05$). Although the urinary cotinine concentrations of staff in all venues did not change following regulation, the GM of total NNAL concentrations of staff in bars ≥150 m^2 was significantly reduced, from 12.1

pg/mg creatinine (Cr) (GSD = 2.0) before regulation to 7.3 pg/mg Cr (GSD = 1.7) after regulation ($p<0.05$). The levels of $PM_{2.5}$ and biomarker did not change in staff in restaurants ≥150 m^2 due to the few smokers prior to regulation. The health effects on staff show that only sensory symptoms improved significantly in venues ≥150 m^2, decreasing from 52% before regulation to 40% after regulation ($p<0.05$). These findings indicate that the implementation of smoke-free regulations significantly reduced the levels of $PM_{2.5}$ and total NNAL concentrations of staff in bars ≥150 m^2 and improved the sensory health of staff in venues ≥150 m^2. However, no improvement was observed in the measured data or the health effects in venues <150 m^2 where indoor smoke-free regulations were not applied.

The purpose of the second study was to determine the prevalence of SHS incursion, and to establish the relationship between SHS incursion and socio-demographic and built environmental factors in MUHs. Population-based samples representing 2,600 adult residents living in MUH in Seoul, Korea were obtained through a web-based selection panel. Residents completed a questionnaire detailing socio-demographic factors, smoking status, frequency of SHS incursion, and built environmental factors. The presence of a personal smoke-free home rule was determined by residents declaring that no one smoked inside the home. Of the 2,600 participants, non-smoking residents who lived in homes with a personal smoke-free rule were selected for further analysis (n = 1,784). SHS incursion had been experienced by 74.7% of the residents within the previous 12 months. A multivariate ordinal logistic

regression analysis indicated that residents who spent more time at home, lived with children, supported the implementation of smoke-free regulations in MUH, lived in small homes, lived in homes with natural ventilation provided by opening a front door or both the windows and a front door, and lived in homes with more frequent natural ventilation were more likely to have SHS incursion into their homes. Most non-smoking residents living in smoke-free homes in MUHs experienced SHS incursion into their homes.

The purpose of the third study was to determine the relationship between SHS incursion and allergic symptoms in children living in homes without smokers in MUHs. We conducted a cross-sectional study in 2015 in Seoul, Korea. Children were recruited from elementary schools, kindergartens, and daycare centers. In total, 16,676 children between 1 and 13 years of age living without smokers in MUH were included in the analysis. Allergic symptoms during the previous 12 months (current wheeze, rhinitis, and eczema) and home environmental factors, including the frequency of SHS incursion during the previous 12 months, were examined using a questionnaire filled out by the parents or guardians of the children. The prevalence of current allergic symptoms in children was 4.9% for wheeze, 42.0% for rhinitis, and 28.1% for eczema. The prevalence of SHS incursion into children's homes was 61.6%. In a multivariate logistic regression analysis adjusted for demographic and home environmental factors, children living in homes with SHS incursion (either no more than once a month or more than once a month) were more likely to have current wheeze, rhinitis, and eczema than those with no SHS

incursion. Thus, SHS incursion into homes was associated with current wheeze, rhinitis, and eczema symptoms in children living in homes without smokers in MUH.

The objective of the final study was to determine urinary cotinine concentrations in non-smoking residents of smoke-free homes and to establish the association of urinary cotinine with housing type and socio-demographic and SHS exposure factors. Data from the Korean National Environmental Health Survey (KoNEHS) I (2009–2011) were used. We examined 814 non-smoking adult residents who were not residing with smokers in apartments and in attached and detached housing and spent their time mainly indoors at home. In Korea, detached housing includes single-family and multifamily (e.g., single room studio) houses. Urinary cotinine was detected in 88% of the 814 non-smoking residents. The urinary cotinine concentrations of residents living in attached [GM: 1.25 ng/ml; 95% confidence interval (CI): 1.00–1.55] and detached housing (GM: 1.37 ng/ml; 95% CI: 1.09–1.73) were significantly higher than those of residents who lived in apartments (GM: 0.81 ng/ml; 95% CI: 0.64–1.01). Urinary cotinine concentrations were significantly higher in residents who were men, had a household income ≤1000 USD/month, were former smokers both with >1 year and ≤1 year of not smoking, and who experienced tobacco smoke odor every day. A multivariate regression analysis showed that housing type, former smoking status, and frequency of experiencing tobacco smoke odor were significantly associated with urinary cotinine concentrations (R^2 = 0.15). The associations detected by

multivariate analysis were similar to those detected for creatinine corrected urinary cotinine concentration. The results of this study indicate that the majority of non-smoking residents of smoke-free homes had detectable levels of urinary cotinine, and housing type, former smoking status, and frequency of experiencing tobacco smoke odor were predictors of urinary cotinine concentrations among study participants. Relationships between urinary cotinine concentrations and the frequency of experiencing tobacco smoke odor implied that non-smoking residents in smoke-free homes might be exposed to tobacco smoke pollutants from SHS incursion and from third-hand smoke in the home.

This study aimed to determine the effects of smoke-free regulations in indoor public places and to establish scientific evidence of the risks of exposure and the health effects due to SHS incursion into smoke-free homes. The implementation of smoke-free regulations in restaurants and bars ≥150 m^2 reduced the indoor $PM_{2.5}$ concentrations and total NNAL concentrations of non-smoking staff in bars ≥150 m^2 and improved the sensory symptoms of non-smoking staff in venues ≥150 m^2. Improvement was not observed in the measured data or health effects at venues <150 m^2, where indoor smoke-free regulations were not applied. These findings might be useful in supporting the expansion of smoke-free regulations to all indoor public places, including restaurants and bars <150 m^2. Further study is needed to determine the effect of ongoing regulations, their longer-term health effects, and the possible social determinants of change over time.

Most non-smoking residents (74.7%) of MUH living in homes with a personal smoke-free rule, where smoke-free regulations were not applied, experienced SHS incursion into their homes. Several socio-demographic and built environmental factors were associated with the experience of SHS incursion. In the study of children living in homes without smokers in MUH, dose-dependent relationships were observed between the frequency of SHS incursion and current wheeze, rhinitis, or eczema in children, even following adjustment for demographic and home environmental factors. These results indicate that SHS incursion was associated with these allergic symptoms in children living in MUH without smokers. KoNEHS I data showed that most non-smoking residents (88%) not residing with smokers had detectable levels of urinary cotinine. Housing type, former smoking status, and frequency of experiencing tobacco smoke odor were predictors of urinary cotinine concentrations.

The findings of the studies conducted in residences indicated that, even in smoke-free homes, most non-smoking residents were at risk of exposure and the health effects due to SHS incursion in their homes. Because KoNHES I data showed that urinary cotinine concentrations were associated with housing type, housing type-specific approaches to the creation of smoke-free environments (e.g., the development of a smoke-free policy, education, or promotional materials) are necessary; this may be particularly important for attached and detached housing. The findings of these studies could be used to support the expansion of smoke-free policies in indoor public places as well

as to inform the public about the need for smoke-free environments in all indoor living spaces.

Key words: allergy, hospitality venue, incursion, multi-unit housing, secondhand smoke, smoke-free home, smoke-free regulation

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Introduction

Background

Characteristics of secondhand smoke

Secondhand smoke (SHS), also known as environmental tobacco smoke, is tobacco smoke inhaled by nonsmokers and smokers from someone else's cigarette, cigar, or pipe smoke in indoor and outdoor environments.[1] It is composed of exhaled mainstream smoke (MS) and air-diluted sidestream smoke (SS). MS is the smoke drawn through the cigarette into the smoker's mouth during puffing, whereas SS is the smoke emitted from the burning cone and through the cigarette paper between puffs.

The International Agency for Research on Cancer (IARC) reported that about 4,000 compounds are present in MS.[2] The qualitative compositions of MS, SS, and SHS are similar. SS is generated at a lower temperature (approximately 600°C between puffs vs. 800–900°C for MS during puffs)[3] and tends to have higher concentrations of cigarette smoke compounds compared to MS.[4] The National Research Council found that SS and MS have different compositions, and some compounds were present in SS at levels ten-fold those in MS.[5]

Following release into the environment, the physical form and chemical composition of SHS are altered by physical and chemical processes.[6] Dispersion (e.g., turbulent mixing) causes SHS concentrations to be more uniformly distributed in indoor environments. SHS particles are mostly liquid droplets of 0.02 to 2 μm in diameter, with a mass median diameter of

0.2 μm. The size of indoor SHS particles measured by number concentrations is less than 0.4 μm with a peak size of approximately 0.1 μm.[7] The composition and size of SHS particles is altered by volatilization and changes in the moisture content of gaseous components in the air over time.[1] SHS particle concentrations decrease following dilution in the environment and impaction on surfaces, including human skin and lung tissue. Gaseous elements in SHS can be adsorbed onto materials.

SHS is primarily composed of organic compounds with a vapor pressure low enough to stay in the condensed phase. Organic compounds are formed during smoking by pyrolysis, volatilization, and partial oxidation of the components of tobacco products. By the year 2000, 69 carcinogens in tobacco smoke had been identified. These included 10 *N*-nitrosamines, 4 aromatic amines, 7 *N*-heterocyclic amines, 10 species of polynuclear aromatic hydrocarbons (PAHs, also known as polycyclic aromatic hydrocarbons or polyaromatic hydrocarbons), 6 heterocyclic hydrocarbons, 4 volatile hydrocarbons, 2 aldehydes, 16 miscellaneous organic compounds, and 9 inorganic compounds.[8]

Adverse health effects of SHS exposure

SHS is a complex mixture of more than 7,000 chemicals including 69 known carcinogens.[9] Exposure to SHS is associated with stroke, nasal irritation, coronary heart disease, and lung cancer in adults.[10–12] Furthermore,

SHS exposure has adverse effects on the female reproductive system (e.g., low birth weight).[12] Based on 2004 data from 192 countries, 603,000 premature deaths were attributable to SHS exposure, equivalent to 1.0% of the mortality rate worldwide.[13] The US Environmental Protection Agency (EPA) reported that the estimated annual number of excess deaths attributable to SHS exposure is about 3,400 (range, 3,423 to 8,866) from lung cancer, 46,000 (22,700 to 69,600) from cardiac-related illnesses, and 430 from sudden infant death syndrome (SIDS).[1]

In children, SHS exposure is associated with middle-ear disease, impaired lung function, lower respiratory illness, SIDS, asthma, and other respiratory symptoms.[12,14] The US EPA estimated that SHS exposure is responsible for about 202,300 episodes of childhood asthma, 150,000 to 300,000 cases of lower respiratory illness, and about 789,700 cases of middle-ear infections annually.[1] Furthermore, the EPA estimated that 24,300 to 71,900 low-birth-weight or preterm deliveries annually can be attributed to SHS exposure. Based on the adverse health effects of SHS exposure, the US Surgeon General reached the following major conclusions.[1]

1) SHS causes premature death and disease in children and in adults who do not smoke.
2) Children exposed to SHS are at an increased risk for SIDS, acute respiratory infections, ear problems, and more severe asthma. Smoking by parents causes respiratory symptoms and slows lung growth in their

children.

3) Exposure of adults to SHS has immediate adverse effects on the cardiovascular system and causes coronary heart disease and lung cancer.

4) The scientific evidence indicates that there is no risk-free level of exposure to SHS

5) Many millions of Americans, both children and adults, are still exposed to SHS in their homes and workplaces despite substantial progress in tobacco control.

6) Eliminating smoking in indoor spaces fully protects nonsmokers from exposure to SHS. Separating smokers from nonsmokers, cleaning the air, and ventilating buildings cannot eliminate exposures of nonsmokers to SHS.

Environmental markers of SHS exposure

SHS exposure can be quantified using environmental markers. Airborne particulate matter <2.5 μm ($PM_{2.5}$) is frequently used as an indirect marker of SHS in indoor environments,[15–20] because most particles in tobacco smoke are <1 μm in diameter. Although there are other sources of particles (e.g., cooking, candles, and outdoor pollution), cigarette smoking is the most important source of $PM_{2.5}$ in indoor environments where smoking is permitted.[21]

$PM_{2.5}$ concentrations can be measured using direct reading or active

sampling. Direct-reading devices use a light-scattering sensor to measure $PM_{2.5}$ concentrations in real time.[22] However, the $PM_{2.5}$ concentrations obtained must be calibrated against those determined gravimetrically because the scattering per unit mass is a function of particle size and refractive index. In active sampling, $PM_{2.5}$ can be collected using filters and $PM_{2.5}$ concentrations (or those of PAHs or metals) can be estimated by gravimetric analyses.

Airborne nicotine is a specific marker of SHS exposure.[22] The majority of nicotine in SHS is in the vapor phase and nicotine is widely used as a tracer for the mixture of chemicals in SHS. The airborne nicotine concentration has been used to quantify SHS exposure in indoor environments.[23–25] However, nicotine is sensitive to photodegradation and is more rapidly eliminated than other SHS components.[26] Because it can be absorbed onto and desorbed from surfaces,[27] nicotine can be detected in indoor environments even in the absence of tobacco smoke. Thus, although airborne nicotine is used as a specific marker of SHS exposure, it is not ideal.[28] Nicotine is typically measured by passive sampling using a 35 mm polystyrene sampling cassette containing a sodium bisulfate-treated filter and covered by a diffusion screen.[29] Depending on the nicotine concentration, passive monitors are deployed for days to weeks because of their low sampling rate (25 mL/min). Nicotine levels can also be measured using active sampling using an adsorbent tube or treated filters over a span of hours. The nicotine concentration can be quantified by gas chromatography

(GC) with mass spectrometry (MS) or a nitrogen/phosphorus detector (NPD).

3-Ethenypyridine (3-EP), formed by pyrolysis of nicotine, is unique to tobacco smoke and is more stable than nicotine in indoor environments.[30] Many studies, mostly funded by the tobacco industries,[22] used 3-EP as a marker for SHS and 3-EP concentrations were strongly correlated with nicotine concentrations than other SHS markers.[31] However, concentration of 3-EP is relatively lower than that of nicotine resulting significant non-detectable samples.[22] 3-EP concentration can be measured using active or passive sampling and is analyzed by GC-MS or NPD in laboratory analysis.

Carbon monoxide (CO) is a byproduct of incomplete combustion and is used as a marker of SHS exposure.[32,33] Although CO is not specific to tobacco, it enables discrimination between non-smoking and smoking environments.[34,35] The CO concentration can be measured using a direct-reading device equipped with an electronic sensor.

SHS contains tobacco-specific nitrosamines (TSNAs) such as 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, also known as nicotine-derived nitrosamine ketone (NNK). The IARC classifies NNK as a group 1 carcinogen.[36] The data on NNK and TSNA concentrations in indoor air after smoking are sparse; most studies have measured airborne NNK concentrations in controlled settings.[22,37] Thus, further studies are needed to characterize the utility of airborne NNK as a marker of SHS in indoor environments.

Other environmental markers of SHS exposure are respirable suspended

particulates (RSPs), PAHs, nitrogen oxides (NOx), aldehydes, and volatile organic compounds (VOCs);[22] however, these are not tobacco-specific markers.

Biological markers of SHS exposure

Nicotine is present in almost all tobacco products but significant amounts are not found in food.[38] On average, one cigarette contains 12 mg nicotine (range, 7 to 18 mg).[2] Nicotine is primarily metabolized in the liver. On average, 75% of nicotine is metabolized to its proximate metabolite cotinine by the liver enzyme cytochrome P450.[39] Cotinine has a longer half-life (16 h) than nicotine (2 h) in urine, blood, and saliva.[40] Because the cotinine concentration in urine is four- to six-fold that in blood and saliva, it has greater sensitivity for evaluating SHS exposure. The cotinine concentration in body fluids has been used in studies on the effects of smoke-free regulations in indoor public places.[41–45]

Cotinine in urine is a specific and sensitive biomarker of SHS exposure,[46] and samples can be collected in a non-invasive manner.[40] The cotinine concentration can be measured in blood (not typically performed), serum, or plasma. The serum cotinine concentration has lower sensitivity for SHS exposure than urinary cotinine concentration. Urinary but not serum cotinine concentration mostly be adjusted for urinary creatinine concentration. Salivary cotinine concentrations parallel those in sera.[40] Measurement of

salivary cotinine levels is non-invasive and samples are easy to collect. Saliva samples have a greater risk of contamination than serum samples and the cotinine concentrations therein may be affected by age, sex, ethnicity, oral pH, diet, dehydration, and drug treatment. The cotinine concentration in human fluids can be measured using GC-MS/MS or liquid chromatography (LC)-MS/MS.

The nicotine level in hair can be used as biomarker of SHS exposure.[47,48] Hair can contain nicotine when the nicotine was present in the circulation. Environmental contamination could be minimal after hair samples is washed.[49] Because hair grows approximately 1 cm/month, the nicotine concentration in 1 cm of hair adjacent to the scalp represents the SHS exposure in the prior month. Hair nicotine can characterize time and exposure because it represents longer SHS exposure period than other biomarkers. The mean hair nicotine concentration is relatively unaffected by fluctuations in exposure, metabolism, and nicotine elimination.[40] Hair is easily collected and transported and can be stored at room temperature without degrading for up to 5 years.[49] However, chemical treatment of hair can reduce nicotine concentrations by 9% to 30%,[40] and hair nicotine concentrations might differ between sexes and among ethnicities. In analytical methods, The nicotine concentration in hair can be measured using LC-ultraviolet (UV) or GC-MS.

NNK is metabolized to 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and NNAL-glucuronides in the body; these metabolites are

typically measured together (total NNAL). Total NNAL is tobacco specific and can be measured in urine.[40] Total NNAL remains in the body longer than cotinine (half-life, ≤3 weeks). Because it is a lung carcinogen, determination of total NNAL in urine in nonsmokers exposed to SHS can be used to assess the link between SHS exposure and lung cancer. However, this requires expertise in analytical chemistry and costly equipment. Whether metabolism differs among age or other factor is unclear.[40] Total NNAL in urine can be quantified by GC-thermal energy analyses, GC-MS/MS, and LC-MS/MS.

Smoke-free regulations for indoor public places in other countries

The US Surgeon General concluded that only the elimination of indoor smoking could protect non-smokers from SHS exposure.[1] Separating smokers from nonsmokers, cleaning the air, and ventilation of building cannot eliminate exposure of nonsmokers to SHS. Therefore, only smoke-free regulations without exemptions can protect nonsmokers, including children, from SHS exposure.

Article 8 of the WHO Framework Convention on Tobacco Control (FCTC), the first global public health treaty, encourages countries to "protect citizens from exposure to tobacco smoke in the workplaces, public transport and indoor public places."[50] As of 2015, 168 countries had signed, and 180 countries including the Republic of Korea had ratified the FCTC.[51]

Many European countries have adopted smoke-free regulations for

indoor public places. Adoption of the WHO FCTC Guidelines on Protection from Tobacco Smoke in 2007 prompted the European Council to promulgate the Recommendation on a Smoke-Free Environment in 2009.[52] The recommendation stipulates that member states must "provide effective protection from exposure to tobacco smoke in indoor workplaces, indoor public places, public transport, and as appropriate, other public places." The scope of implementation of smoke-free regulations for indoor workplaces and public places varies among EU member states. Hungary, Bulgaria, Spain, Ireland, the United Kingdom, Malta, Greece, the Former Yugoslav Republic of Macedonia, and Turkey have 100% smoke-free regulations for enclosed workplaces and public places, including restaurants and bars.[52] Although 10 member states (Belgium, Cyprus, Finland, France, Italy, Latvia, Lithuania, Sweden, Poland, and Slovenia) and Norway and Iceland have smoke-free regulations for workplaces and enclosed public places, these countries allow separate and enclosed smoking rooms under certain conditions. The remaining member states (Denmark, the Netherlands, Luxembourg, Romania, Portugal, Austria, Germany, the Czech Republic, Estonia, and Slovakia) and the Republic of Serbia and Croatia have smoke-free regulations with exemptions for certain public places.

The United States is not a party to the FCTC and has no federal smoke-free law. However, as of January 2, 2018, 25 states, along with the District of Columbia, Puerto Rico, and the US Virgin Islands, had 100% smoke-free regulations for non-hospitality workplaces, restaurants, and bars.[53] This

represents 58.4% of the US population. Across the US, 22,661 municipalities have 100% smoke-free regulations for non-hospitality workplaces, restaurants, and/or bars; this represents 81.6% of the US population.

Many Asian countries have smoke-free regulations for indoor public places. All Association of Southeast Asian Nations (ASEAN) countries except Indonesia are parties to the FCTC.[54] However, the scope of implementation of smoke-free regulations for indoor workplaces and public places slightly differed among ASEAN member states. All member states except for the Philippines and Indonesia have national smoke-free regulations. Five member states (Brunei, Cambodia, Lao PDR, Myanmar, and Thailand) have 100% smoke-free regulations for workplaces and most public places including restaurants and bars. Although four other member states (Indonesia, Philippines, Singapore, and Vietnam) have such regulations for workplaces and public places, smoking rooms are allowed. Malaysia implemented smoke-free regulations for workplaces and public places with exemptions for bars and non-air-conditioned restaurants. In East Asia, China, Japan, Mongolia, and the Republic of Korea are parties to the FCTC. While Mongolia and the Republic of Korea have national smoke-free regulations for indoor public places including restaurants and bars, China and Japan have local smoke-free regulations for indoor public places.

Smoke-free regulations for indoor public places in Korea

Since enacting the National Health Promotion Act (NHPA) in 1995, the Korean government has expanded smoke-free regulations for indoor public places. However, certain indoor public places were granted exemptions or allowed to implement smoke-free regulations gradually. In 1995, the NHPA designated smoke-free areas in large buildings, academies, tourist accommodations, indoor gymnasia, and medical facilities.[55] In 2003, smoking restriction was expanded to schools, computer game rooms, comic rooms, and large restaurants and bars. Because the Korean government signed the FCTC, it has strengthened smoke-free regulations for indoor public places. In 2011, the Korean government amended Article 9 of the NHPA to enforce smoke-free regulations for indoor public places including public institutions and hospitality venues. Smoke-free regulations were implemented for commercial hospitality venues such as restaurants and bars on July 1, 2013 for venues ≥150 m^2, January 1, 2014 for those ≥100 m^2, and January 1, 2015 for venues of all sizes. The Korean government only implemented smoke-free regulations for large sports facilities (≥1,000-person capacity) in 2013. The NHPA restricted smoking in all indoor facilities including billiard rooms and driving ranges on December 3, 2017.

Effects of smoke-free regulations for indoor public places

Many countries have evaluated the effects of implementation of smoke-free regulations in indoor public places, and have found that it has resulted in positive effects on health. For example, hospital admissions for cerebrovascular disease and chronic obstructive pulmonary disease (COPD),[56] and the incidence of non-hospital emergency visits for bronchospasm[57] and of acute exacerbated COPD[58] decreased after implementation of smoke-free regulations. A meta-analysis of 18 studies showed that smoke-free regulations in public and work places were associated with reductions in the incidence of acute myocardial infarction.[59] In children, reduced numbers of emergency department visits for asthma, ear infections, and upper respiratory infections were observed after implementation of smoke-free regulations,[60] and the risks of low birth weight, preterm birth, and small for gestational age were decreased.[61]

Implementation of smoke-free regulations in indoor public places resulted in a significant reduction in indoor air pollution (e.g., $PM_{2.5}$, RSP, VOCs, and CO).[15–20,32,33,62] Furthermore, implementation of smoke-free regulations in indoor public places resulted in reduced levels of biomarkers of SHS exposure (e.g., cotinine and NNAL)[41–45] and improved the health (e.g., respiratory or sensory symptoms, or cardiovascular risk) of the non-smoking staff of hospitality venues.[63–65]

Scientific evidence on the impact of smoke-free regulations has been critical for expansion the regulations to other indoor public places. The

benefit of smoke-free regulations for indoor public places has been evaluated in other countries, but not in Korea. Smoke-free regulations went into effect on July 1, 2013 for only ≥150 m^2 restaurants and bars, providing an opportunity to evaluate their effects. Scientific evidence on the effects of smoke-free regulations can be used to support expansion of the regulations to other indoor public places.

Emerging risks of SHS exposure in multi-unit housing

A large number of people live in multi-unit housing (MUH) worldwide. In the United States, one-quarter of the population (25.8%) or 79.2 million people, live in MUH.[66] In Korea in 2015, more than two-thirds of the population (74.5%) lived in MUH (i.e., apartments or attached housing). The home environment is a significant source of SHS exposure.[1] Because people tend to spend the majority of their time in their homes, SHS exposure at home can be a significant contributor to the total SHS exposure. On average, people spend about 65% in the United States[67] and 59% in Korea of their time at home.[68]

Even in smoke-free homes in MUH, residents can be exposed to SHS. In the United States, 73% of children living in homes in which nobody smoked inside the home had detectable serum levels of cotinine.[69] The serum levels of cotinine of children living in apartments were 45% higher than those living in detached houses. In that study, children living in smoke-free homes

in MUH might be exposed to SHS transferred from neighboring units or from outside;[70-73] this is known as "SHS incursion."

Movement of SHS depends on airflow that arises from pressure differences between units due to the stack effect, wind effect, and mechanical effect.[74] The stack effect arises from differences in the indoor to outdoor air density due to temperature and moisture differences, thus causing airflow between units through small pathways. The wind effect is caused by a pressure difference between the windward and leeward sides of a building, thus promoting air transfer through cracks between units or through exterior walls. The mechanical effect is caused by pressure differences generated by air conditioning, ventilation, and/or heating systems.

SHS incursion in MUH is widespread in other countries.[75] Most studies measured SHS incursion in homes in MUH using self-report methods (e.g., questionnaires, telephone, or verbal). Table 1 lists reports of SHS incursion in MUH by year of publication. Residents of MUH reported a prevalence of SHS incursion of 16% to 80%.[73,76–86] However, this value should be interpreted with caution due to the diverse methods used to inquire about SHS incursion.

Table 1-1. Summary of peer-reviewed literature reporting SHS incursion in multi-unit housing*

Reference	Location	Housing type	Sample size	Survey method	Experience of SHS incursion		Other assessed indicators
					Subpopulation	%	
Hennrikus, Pentel et al. 2003	Minnesota, USA	Rented MUH	301	Paper	-	46	Existing smoke-free policy; attitude toward difficulty of enforcement; attitude toward policy; health beliefs
King, Cummings et al. 2010	New York, USA	General MUH	5,936	Telephone	Among home with personal smoke-free policy (*n* = 3,326)	46	Existing smoke-free policy; attitude towards policy
Baezconde-Garbanati, Weich-Reushe et al. 2011†	California, USA	Low-income housing	142	Verbal	-	55	Attitudes towards SHS; bothering of SHS; attitudes towards smoke-free policies
Baezconde-Garbanati, Weich-Reushe et al. 2011†	California, USA	Rented MUH	409	Telephone	-	63	Attitudes towards smoke-free policies; attitudes towards SHS
Hewett, Ortland et al. 2012	Minnesota, USA	Common interest communities	495	Paper; telephone	-	28	Existing smoke-free policy; bothering of SHS; perceived market for smoke-free units; attitudes towards existing policy

MUH = multi-unit housing; SHS = secondhand smoke.
*Modified and update from previous report.[75]
†Each of two method (verbal and telephone) in the paper described separately.

Table 1-1. Summary of peer-reviewed literature reporting SHS incursion in multi-unit housing (continued)

Reference	Location	Housing type	Sample size	Survey method	Experience of SHS incursion		Other assessed indicators
					Subpopulation	%	
Licht, King et al. 2012	National, USA	General MUH	418	Telephone	Among home with personal smoke-free policy (*n* = 339)	44	Existing smoke-free policy, attitude towards policy
Ballor, Henson et al. 2013	Washington, USA	Public housing	229	Paper	-	64	Attitude towards smoking rule and policies; personal smoking behaviors (smoking frequency, intention to quit smoking, knowledge about cessation assistance)
Koster, Brink et al. 2013	Denmark	General MUH	2,183	Internet	Among home with no smoking reported†	28	Attitude towards smoke-free policy
Wilson, Torok et al. 2014	National, USA	General MUH	731	Telephone, internet	Among home with no smoking for the past 3 month (*n* = 532)	16	Existing smoke-free policy
Leung, Ho et al. 2015	Hong Kong	General MUH	61,810	Paper	-	16	Respiratory symptoms of children

MUH = multi-unit housing; SHS = secondhand smoke.

Table 1-1. Summary of peer-reviewed literature reporting SHS incursion in multi-unit housing (continued)

Reference	Location	Housing type	Sample size	Survey method	Experience of SHS incursion		Other assessed indicators
					Subpopulation	%	
Nguyen, Gomez et al. 2016	National, USA	General MUH	17,467	Telephone	Among home with personal smoke-free policy†	34	Type of tobacco use (combustible only, noncombustible only, both, no current tobacco use), existing smoke-free policy
Delgado-Rendon, Cruz et al. 2017	LA, USA	General MUH	403	Electronic tablet	-	80	Health literacy, personal home smoking policy and enforcement, support for a smoke-free building policy, knowledge, attitudes, self-efficacy, and intention to change behavior to protect themselves from smoke exposure
Gentzke, Hyland et al. 2017	3 community pairs across the USA	Subsidized and market-rate MUH	1,565	Telephone	Among home with smoke-free rule (*n* = 1259)	50	Preferences towards smoke-free policies

MUH = multi-unit housing; SHS = secondhand smoke.

Few countries have policies to restrict smoking in MUH. The United States has led the way in restricting or prohibiting smoking in private units in MUH with municipal law or Housing Authority policies.[87] As of January 2, 2018, 38 municipalities had implemented 100% smoke-free MUH, defined as prohibition of smoking in 100% of private units in all specified types of privately and publicly owned MUH. Moreover, 541 municipalities have smoke-free policies for publicly owned MUH. The majority of public-owned MUH are managed by the Public Housing Authority. Delaware, Hawaii, Maine, Montana, New Hampshire, North Carolina, and Washington have state laws and policies that prohibiting smoking in private units of MUH. Public Housing Agencies are required by the US Department of Housing and Urban Development to implement a smoke-free policy for all of their public housing properties by August 2018. In Canada, federal and provincial law allows owners of MUH to adopt smoke-free policies.[88] In 2012, 8 and 10 jurisdictions (i.e., provinces and territories) had 100% smoke-free policies for public and private MUH, respectively.

Smoke-free regulations have not been applied to MUH in Korea. From September 3, 2016, the Korean government allowed smoking in shared areas such as corridors, stairwells, elevators, or underground parking lots to be restricted if demanded by at least half of MUH residents. However, this law did not include private areas. In 2017, the government amended the Multi-family Housing Management Act to allow management authorities (e.g., apartment managers) to recommend or educate smokers in MUH to stop

smoking at home. However, there were limitations in regulating private spaces and some MUH, such as small apartments or attached houses, do not have a management authority.

Scientific evidence on the risk and health effects of SHS exposure of residents due to incursion in smoke-free homes in MUH in Korea is limited. Most studies in other countries focused on the relationships of SHS incursion into homes in MUH with socio-demographic factors, attitude toward smoke-free policies, or smoking behaviors, but not built environmental factors. Identifying built environmental factors associated with SHS incursion would enhance our understanding of SHS incursion-related exposure in MUH. Although SHS incursion in smoke-free homes in MUH is widespread, its health effects are unclear. Children are particularly at risk for SHS exposure in homes because they spend the majority of their time at home. SHS exposure is a risk factor for allergic diseases such as asthma, allergic rhinitis, and eczema in children.[89–93] A study in Hong Kong reported that SHS incursion into smoke-free homes is associated with respiratory symptoms in adolescents.[80] Further research on the effects of SHS incursion on the health of children living in smoke-free homes would provide insight into the risks of SHS exposure in residences. SHS incursion in smoke-free homes was measured using self-reporting methods. Only one study involving United States children in smoke-free homes examined SHS exposure using serum cotinine concentration.[69] Further research studies are needed to characterize SHS exposure using biomarkers of non-smoking residents in smoke-free MUH.

Objectives

The overall objectives of this study were to determine the effects of smoke-free regulations in indoor public places and to establish scientific evidence of the risks of exposure and the health effects due to SHS incursion in smoke-free homes in Korea. The specific objectives were as follows:

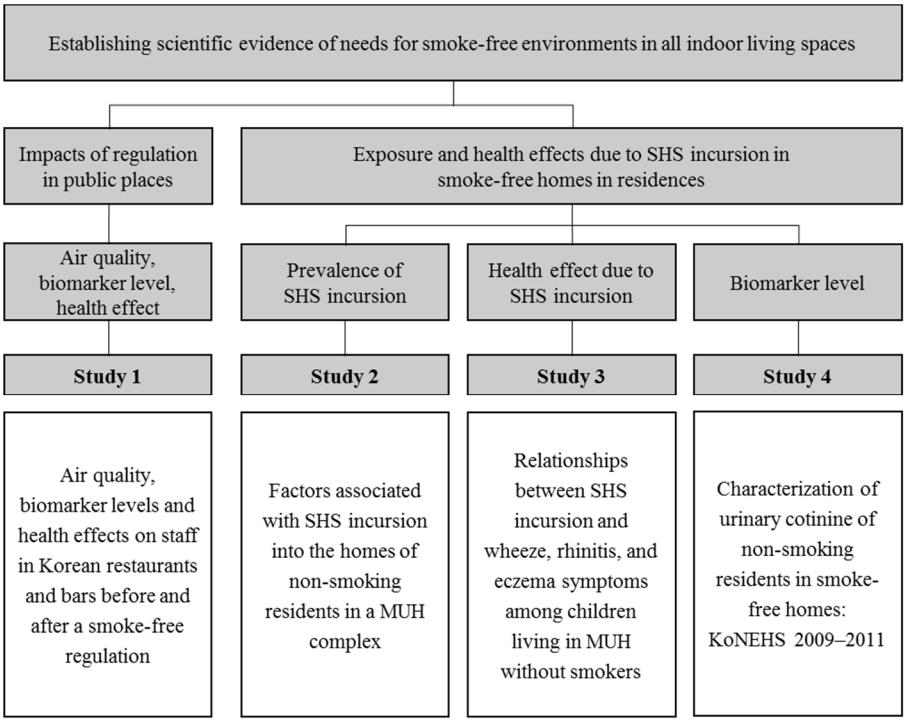
1) To determine the effects of Korean smoke-free regulations in restaurants and bars on air quality, biomarker levels, and staff health.

2) To determine the prevalence of SHS incursion in MUH and the relationship between SHS incursion into the homes of non-smoking residents and socio-demographic and built environmental factors.

3) To investigate the relationship between SHS incursion and allergic symptoms in children living in smoke-free MUH.

4) To characterize the urinary cotinine concentrations of non-smoking residents of smoke-free homes, and to establish their relationship with housing type and other socio-demographic and SHS exposure factors.

An outline of the study is shown in Figure 1-1. First, we evaluated the effects of smoke-free regulations for restaurants and bars in terms of air quality, biomarker levels, and the health of non-smoking staff. The findings can support expansion of smoke-free policies to other indoor public places. Second, we determined the prevalence and predictors of SHS incursion among non-smoking residents of smoke-free MUH using self-reporting methods. Third, we examined the relationships between SHS incursion and wheeze, rhinitis, and eczema symptoms in children living in MUH without smokers using questionnaires. Fourth, we identified factors associated with the urinary cotinine concentrations of non-smoking residents of smoke-free homes using data from the Korean National Environmental Health Survey. Such studies might provide insight into the exposure and health effects of residents due to SHS incursion in smoke-free homes. The findings might also be used to support the expansion of smoke-free policies to indoor public places and to inform the public about the benefits of smoke-free environments in indoor living spaces.



Abbreviation: SHS = secondhand smoke; MUH = multi-unit housing; KoNEHS = Korean National Environmental Health Survey

Figure 1-1. Overall outline of the study.

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Chapter 2.

Air quality, biomarker levels and health effects on staff in Korean restaurants and bars before and after a smoke-free regulation

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Abstract

The Korean government implemented a smoke-free regulation at square floor area of ≥150 m^2, rather than <150 m^2, restaurants and bars from July 2013. This study examined the effects of the smoking regulations in restaurants and bars by measuring indoor air quality, biomarker levels and health effects on staff. Particulate matter smaller than 2.5 μm ($PM_{2.5}$) was measured in 146 venues before and one month after the regulation. The urinary cotinine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) levels were measured in 101 staff members at 77 venues before and one month after the regulation. We measured self-reported respiratory and sensory symptoms on both phases. Of the 146 venues, 121 venues were included in the $PM_{2.5}$ analysis. In bars ≥150 m^2, geometric means (GM) of indoor $PM_{2.5}$ concentration was significantly reduced from 93.2 μg/m^3 (geometric standard deviation, GSD = 2.2) before the regulation to 55.3 μg/m^3 (GSD = 2.2) after the regulation ($p<0.05$). While the urinary cotinine concentrations of the staff in all venues were not changed after the regulation, the GM of total NNAL concentrations of the staff in bars ≥150 m^2 was significantly reduced from 12.1 pg/mg creatinine (Cr) (GSD = 2.0) before the regulation to 7.3 pg/mg Cr (GSD = 1.7) after the regulation ($p<0.05$). The health effects on staff show that only sensory symptoms significantly improved in venues ≥150 m^2 from 52% before the regulation to 40% after the regulation ($p<0.05$). The smoke-free regulation significantly reduced the levels of $PM_{2.5}$ and total NNAL

concentrations in bars ≥150 m^2 and improved sensory health among staff in venues ≥150 m^2. The results of this study could be useful in supporting an expansion of the smoke-free regulation in all indoor places, including restaurants and bars <150 m^2.

Introduction

Second-hand smoke (SHS) contains a mixture of more than 7,000 chemicals, including more than 69 known carcinogens.[1] SHS is well known to be associated with lung cancer, coronary heart disease, cardiovascular disease, asthma and other respiratory symptoms.[2-5] Thus, governments from many countries have legislated smoke-free regulations in indoor spaces and work places due to the growing scientific evidence of the adverse health effects of SHS.

Implementation of smoke-free regulations is associated with an improvement in health effects. In Piedmont, Italy, the rates of hospital admission for acute myocardial infarction were compared before and after smoke-free regulation with the rates of admissions during the same periods in 12 months before.[6] The number of admissions decreased among patient under aged 60.0 years (odd ratio, 0.89; 95% confidence interval, 0.81-0.98). After comprehensive national smoke-free regulations in all indoor public places in Rome, Italy, acute coronary events decreased by 11.2% for 35- to 64-year-olds and 7.9% for 65- to 74-year-olds.[7]

Indoor smoke-free regulations effectively reduce indoor air pollutants and the biomarker levels of SHS. In Ireland, particulate matter smaller than 2.5 μm ($PM_{2.5}$) and benzene concentrations were reduced by 83% and 80.2%, respectively, in bars 1 year after smoking was banned.[8] A significant reduction in urinary cotinine levels was previously reported after the smoke-free

regulation. Urinary cotinine levels decreased from 35.9 to <5 ng/ml among non-smoking bar workers after the smoke-free regulation in Michigan.[9]

SHS contains tobacco-specific nitrosamines, such as 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK).[10] NNK has been demonstrated to be a lung carcinogen in an animal model[11] and was recently classified as a group 1 carcinogen for humans by the International Agency for Research on Cancer (IARC).[12] The total NNAL, which consists of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and its glucuronides (NNAL-Glucs), is metabolised from NNK.[13] Many studies have used cotinine levels as biomarkers for SHS exposure. However, total NNAL has rarely been used to determine changes in these levels due to smoke-free regulations in hospitality venues.

In Korea, smoking has been banned in public places, such as hospitals, schools, and airports, because of the passing of Article 7 of the Enforcement Rules of the National Health Promotion Act (ERNHPA) in 2003. However, most commercial hospitality venues, such as restaurants and bars, were not subject to a complete smoke-free regulation. The amendment of the ERNHPA in 2011 banned smoking in restaurants and bars with net indoor floor areas of ≥150 m^2 starting in July 2013.

The smoke-free regulation going into effect in only restaurants and bars ≥150 m^2 provided a good opportunity to evaluate the effect of the smoke-free regulation before and after it was enforced and compares venues that enforced the regulation and those that did not. The purpose of this study was to

determine the effects of Korean smoke-free regulation in restaurants and bars by measuring indoor air quality, biomarker levels and health effects on staff.

Methods

Study design and participants

In Seoul, the capital of the Republic of Korea, restaurants and bars were targeted for a determination of smoking-free regulation effect. The research team requested lists of restaurants and bars from the Health Center in 6 districts in Seoul. The lists included the type, net indoor area, name, address, and phone number of the venues. The research team randomly called venues on the lists to determine whether they allowed smoking. A flow chart describing the recruitment of restaurants and bars and their staff is shown in Figure 2-1. One hundred fifty-four venues that allowed smoking were selected according to non-proportional quota sampling based on the type (restaurants and bars) and size (<150 m^2 and ≥150 m^2) of venues. The venues were selected following a convenience sampling based on the accessibility of the venues to the research team.

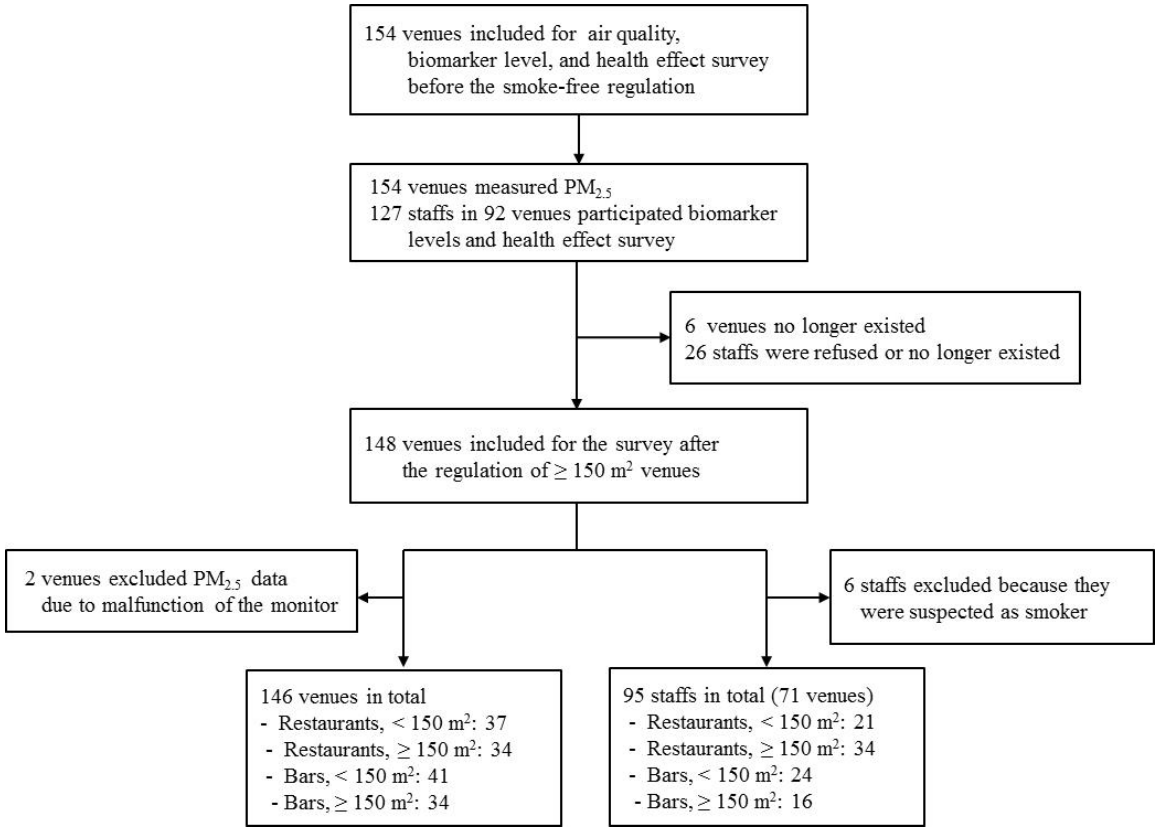

Figure 2-1. Flow chart of recruitment of restaurants and bars and their staff before and 1 month after the smoke-free regulation.

$PM_{2.5}$ levels were measured in the 154 venues before the introduction of the smoke-free regulation in venues ≥150 m^2 from April 29 to June 28, 2013 (first phase). The $PM_{2.5}$ measurements were repeated in 148 venues from August 1 to September 27, 2013 (second phase) at the same venues 1 month after the regulation. Six venues no longer existed during the second phase. Two venues were excluded due to a malfunction of the monitor that resulted in unusual $PM_{2.5}$ data. One hundred forty-six venues were assessed in both phases of the study.

The research team and the Health Center recruited 127 non-smoking staff from 92 of the 154 venues whose owners or managers were interested in participating in the study based on telephone interviews to assess biomarker levels and health effects on the staff. The research team visited the venues and selected staff members that had never smoked or ex-smokers who quit at least 3 months before the study and worked commonly in the main hall. One hundred one staff members completed both phases of the study, but 6 staff members were excluded because they were suspected of smoking based on their urinary cotinine levels. A total of 95 staff members (75%) in 71 venues were included in the analysis. Of the 95 participants, 4 participants came from 1 venue, 3 participants came from 3 individual venues, 2 participants worked at 15 individual venues and the remaining 52 participants were employed at 52 different venues.

Air quality assessment

Airborne $PM_{2.5}$ concentration was used as an indicator of SHS in restaurants and bars. Other studies have also used $PM_{2.5}$ concentration as a marker of SHS.[14,15] The $PM_{2.5}$ levels were measured using a portable nepheometer (SidePak AM510, TSI Inc., MN, USA). The monitor used a scattering of 670 nm wavelength light to determine the mass concentrations of aerosols. The monitor was fitted with a $PM_{2.5}$ impactor to remove particles larger than 2.5 μm and zero calibrated with a HEPA filter according to the manufacturer's specifications each day before measuring began. The monitor was set to a logging interval of 1 min at a flow rate of 1.7 l/min. As the scattering per unit of mass was a strong function of the size and refractive index of the particles, the measured value was converted by a factor of 0.295 such that it was suitable for SHS. The conversion factor had been previously determined from an experiment with SidePak collocated with gravimetric measurements.[16]

The research team visited restaurants between 18:00 and 20:00 and bars between 20:00 and 24:00 on weekdays to measure the indoor $PM_{2.5}$ concentrations. The monitor was concealed in a small bag and continuously collected sample every minute before and after entrance into the venues (outdoor) for 5 min (n = 10) and during the visit (indoor) for 30 min (n = 30). When outside the venues, the researchers placed the monitor in the front of the targeted venues away from direct emission sources (e.g., outdoor smokers and vents). The data points from each indoor and outdoor place were averaged

to provide an average $PM_{2.5}$ concentration. The monitor was placed on a table or seat in the venues at a location as central as possible away from any doors, windows, kitchen areas and direct puff of cigarettes. The research team recorded information on regarding the sampling location, size, and type of cooking fuel. The number of burning cigarettes (#bc) in each minute and number of customers in every five minute were noted during the indoor measurement. Smoking density was calculated as the number of burning cigarettes per 100 m^3 of indoor volume. Numbers of customers counted by each 5-min interval were averaged to produce average number of customers in each venue. The repeated measurement was evaluated after the regulation on the same day of the week as close as possible to the same time of day (± 30 min) at which the measurement before the regulation was performed.

Assessment of biomarker levels and health effects

Urinary samples and questionnaires for health effects on the staff in the venues were collected on the spot during off-hours or during the setup time of the day within 1 week of the $PM_{2.5}$ measurements. The urinary samples were frozen at -70 °C until analysis and sent in batches to the Center for Clinical Services, National Cancer Center (323 Ilsan-ro, Ilsandong-gu, Goyang-si, Gyeonggi-do, 410-769). The urinary samples were blinded to the type and area of venues, including the smoking history of participants.

The cotinine and total NNAL concentrations were used as biomarkers of SHS for staff at the venues. These biomarker have been used to assess human

SHS exposure in others studies.[9,17] The urinary cotinine concentrations were estimated using a method modified from a previous study.[18] The cotinine concentrations were measured by liquid chromatography-tandem mass spectrometry (LC-MS/MS) using electro spray ionisation. The liquid chromatography unit used was an Agilent 1100 series (Agilent Technologies, Santa Clara, USA), and the tandem mass spectrometer was an API 4000 machine (AB SCIEX, Framingham, USA) equipped with an atmospheric pressure chemical ionisation interface. The limit of quantification (LOQ) for cotinine was 2 ng/ml. The urinary total NNAL concentrations were measured by LC-MS/MS using modified methods that have been previously described.[19] The LOQ for the total NNAL was 0.25 pg/ml. The creatinine levels in the urine were measured via colorimetry (Toshiba 2090 FR; Toshiba, Tokyo, Japan).

The health effects were assessed via questionnaires that were based on those used in the Scotland and Ireland bar studies.[20,21] The questionnaires provided demographic data and information concerning SHS exposure, respiratory symptoms (wheezing/whistling, shortness of breath, morning cough, rest of day or night cough, phlegm production), sensory irritation (red or irritated eye, runny or sneezing nose, sore or scratchy throat), and job descriptions.

Participants whose cotinine levels exceeded 100 ng/ml were suspected of smoking.[22] All staff participated voluntarily and provided written informed

consent. The ethics committees at Seoul Medical Center reviewed and approved all procedures prior to the survey.

Statistical analysis

Normality of data distribution was verified by Shapiro-Wilk test. Indoor $PM_{2.5}$ (p = 0.40) and total NNAL concentration (p = 0.09) were log-normally distributed. Although log-normal distribution of cotinine concentrations were statistically rejected (p<0.05), the cotinine concentrations were closed to log-normal distribution. Thus, geometric mean (GM) and geometric standard deviation (GSD) of $PM_{2.5}$, cotinine, and total NNAL were computed. Wilcoxon's signed rank test was used to compare the differences between indoor and outdoor $PM_{2.5}$ concentrations and smoking density, indoor $PM_{2.5}$ concentrations, and number of customers in restaurants and bars between the two phases. Wilcoxon rank-sum test was used to compare cotinine and total NNAL levels among staff that live with smokers and staff that do not. Wilcoxon's signed rank test was employed to compare the differences in the cotinine and total NNAL concentrations in the venues between the two phases. Spearman's correlation was used to evaluate the associations among the smoking density, indoor $PM_{2.5}$ concentrations, cotinine and total NNAL concentrations of staffs in restaurants and bars as some variables were not normally distributed. When the urinary cotinine and total NNAL concentrations were below the limit of quantification, half of the LOQ were assigned. McNemar's test was employed to assess the changes in the number

of participants experiencing respiratory and sensory symptoms. Although respiratory and sensory symptoms were assessed via several questions, the respiratory and sensory symptoms were determined if participants have any of those related symptoms in the questionnaires. Wilcoxon rank-sum test was employed to compare indoor $PM_{2.5}$ concentrations in between bars ≥ 150 m^2 without smoking room and those with smoking room. SAS software (ver. 9.2; SAS Institute, Inc., Cary, NC) was used for all statistical analyses. A *p*-value of 0.05 was considered significant in all analyses. SigmaPlot 9.0 (Systat Software Inc., Chicago, IL, USA) were used to draw the graphs.

Results

Characteristics of restaurants and bars and their staffs

The general characteristics and demographic details of participants in restaurants and bars are summarised in Table 2-1. The majority of the 146 venues cooked in the kitchen (58%) and cooked with gas (79%). A slightly higher use of natural ventilation (55%) was observed in the venues. The majority of the 95 participants in the 71 venues were female (69%) and had at least some high school education (57%); 39% were permanent staff and 36% were owners. Ex-smokers comprised 19% of the cohort, and staff living with smokers at home constituted 40% of the cohort. The average age of the participants was 47.4 ± 11.5 years during the study period. The average working years and hours of the participants were 4.9 ± 5.1 years and 64.2 ± 16.2 hr/week, respectively.

Table 2-1. Characteristics of restaurants and bars and their staff

	Characteristics	Restaurants *n* (%)	Bars *n* (%)	Total *n* (%)
Venues (*n* = 146; of 148 venues)	Cooking places			
	Kitchen	13 (18)	72 (96)	85(58)
	Kitchen and table	58 (82)	3(4)	61(42)
	Type of Cooking			
	Gas	45 (63)	70 (93)	115 (79)
	Electric stove	1 (1)	5 (7)	6 (4)
	Charcoal	25 (35)	0 (0)	25 (17)
	Ventilation			
	Local	37 (52)	29 (39)	66 (45)
	Natural	34 (48)	46 (61)	80 (55)
Participants (*n* = 95; 71 of 148 venues)	Sex			
	Male	11 (20)	18 (45)	29 (31)
	Female	44 (80)	22 (55)	66 (69)
	Educational status			
	Less than high school	14 (25)	7 (18)	21 (22)
	High school	35 (64)	19 (48)	54 (57)
	More than high school	6 (11)	14 (35)	20 (21)
	Job position			
	Owner	19 (35)	15 (38)	34 (36)
	Manger	5 (9)	7 (18)	12 (13)
	Permanent staff	25 (45)	12 (30)	37 (39)
	Temporary staff	6 (11)	6 (15)	12 (13)
	Smoking history			
	Ex-smoker	8 (15)	10 (23)	18 (19)
	Currently living with smoker			
	Living with smoker	27 (49)	11 (28)	38 (40)
		AM ± SD	AM ± SD	AM ± SD
	Age	48.7 ± 10.2	45.7 ± 12.9	47.4 ± 11.5
	Year worked in the venues	5.5 ± 5.6	4.2 ± 4.4	4.9 ± 5.1
	Hours worked per week	69.2 ± 14.9	57.2 ± 15.4	64.2 ± 16.2

AM = arithmetic mean; SD = standard deviation.

Smoking observation, smoking density, and levels of $PM_{2.5}$

Of the 146 venues, 25 restaurants were excluded because they used charcoal for tableside cooking, which was suspected to be a significant indoor source of $PM_{2.5}$.[23] A total of 121 venues were included for further analysis.

In the total 121 restaurants and bars, the GM of the indoor and outdoor $PM_{2.5}$ concentration was 76.5 μg/m^3 (GSD = 2.2) and 35.6 μg/m^3 (GSD = 1.6) at baseline and 63.4 μg/m^3 (GSD = 2.4) and 28.7 μg/m^3 (GSD = 1.9) 1 month after the regulation in venues ≥150 m^2. Indoor $PM_{2.5}$ concentrations of each phase were significantly higher than outdoor $PM_{2.5}$ concentrations ($p<0.05$).

The number of smoking observed venues, smoking density, indoor $PM_{2.5}$ concentrations, and number of customers by type and area of the restaurants and bars before and after the smoke-free regulation are presented in Table 2-2. Before the smoke-free regulation at venues ≥150 m^2, the number of smoking observed venues was higher with respect to bars than with respect to restaurants. After the smoke-free regulation at venues ≥150 m^2, smoking was observed in one ≥150 m^2 restaurant and in ten bars ≥150 m^2.

Although smoking density decreased in restaurants ≥150 m^2 (n = 25), the difference was not statistically significant (p = 0.22). The arithmetic mean (AM) and standard deviation (SD) of smoking density in restaurants ≥150 m^2 was 0.18 ± 0.39 #bc/100 m^3 before the regulation and 0.07 ± 0.37 #bc/100 m^3 after the regulation. The smoking density in bars ≥150 m^2 (n = 34) significantly decreased after the regulation ($p<0.05$). The AM of smoking density in bars ≥150 m^2 was 1.09 ± 2.47 #bc/100 m^3 before the regulation 0.17

± 0.34 #bc/100 m^3 after the regulation. The smoking density levels in venues <150 m^2 were not significantly different across the two phases.

Indoor levels of $PM_{2.5}$ concentrations showed a similar trend. Although the indoor $PM_{2.5}$ concentrations decreased in restaurants ≥150 m^2 (n = 25), the differences were not statistically significant (p = 0.63) (Figure 2-2). The GM of the indoor $PM_{2.5}$ concentration in restaurants ≥150 m^2 was 51.9 μg/m^3 (GSD = 2.2) before the regulation and 42.1 μg/m^3 (GSD = 2.7) after the regulation. The indoor $PM_{2.5}$ concentrations in bars ≥150 m^2 (n = 34) significantly decreased after the regulation (p<0.05). The GM of the indoor $PM_{2.5}$ concentration in bars ≥150 m^2 was 93.2 μg/m^3 (GSD = 2.2) before the regulation and 55.3 μg/m^3 (GSD = 2.2) after the regulation. The $PM_{2.5}$ concentrations in venues <150 m^2 were not significantly different between the two phases.

Numbers of customers in all venues were not significantly different between before and after the regulation in venues ≥150 m^2. The AM and SD of number of customers in restaurants ≥150 m^2 was 23.0 ± 15.9 #venue before the regulation and 22.3 ± 20.7 #venue after the regulation. The number of customers in bars ≥150 m^2 was 31.3 ± 18.5 #venue before the regulation and 33.4 ± 19.9 #venue after the regulation.

Table 2-2. Number of smoking observed venues, smoking density, indoor $PM_{2.5}$ concentrations, and number of customer in all restaurants and bars before and after the smoke-free regulation at venues ≥150 m^2

	Type of venue	Area (m^2)	*n*		Before the regulation	After the regulation	Median changes from before the regulation (IQR)	*p*-value*
Smoking observation (#venue)	Restaurant	<150	21	# (%)	9 (43)	6 (29)	-	-
		≥150	25	# (%)	7 (28)	1 (4)	-	-
	Bar	<150	41	# (%)	39 (95)	28 (68)	-	-
		≥150	34	# (%)	23 (68)	10 (29)	-	-
Smoking density (#bc/100 m^3)	Restaurant	<150	21	AM ± SD	1.19 ± 1.77	0.80 ± 1.89	0.00 (-0.22 to 0.00)	0.38
		≥150	25	AM ± SD	0.18 ± 0.39	0.07 ± 0.37	0.00 (0.00 to 0.00)	0.22
	Bar	<150	41	AM ± SD	2.76 ± 2.68	2.10 ± 2.46	-0.67 (-1.46 to 1.39)	0.52
		≥150	34	AM ± SD	1.09 ± 2.47	0.17 ± 0.34	-0.36 (-0.88 to 0.00)	<0.05
Indoor $PM_{2.5}$ concentration(μg/m^3)	Restaurant	<150	21	GM (GSD)	49.5 (1.7)	45.8 (2.3)	-1.1 (-27.9 to 33.3)	0.93
		≥150	25	GM (GSD)	51.9 (2.2)	42.1 (2.7)	-7.8 (-18.0 to 19.2)	0.63
	Bar	<150	41	GM (GSD)	103.0 (2.0)	107.6 (2.0)	0.4 (-22.4 to 50.0)	0.62
		≥150	34	GM (GSD)	93.2 (2.2)	55.3 (2.2)	-22.5 (-57.1 to -6.5)	<0.05
Number of customer (#venue)	Restaurant	<150	21	AM ± SD	10.0 ± 7.7	12.6 ± 10.0	2.3 (-0.9 to 6.0)	0.09
		≥150	25	AM ± SD	23.0 ± 15.9	22.3 ± 20.7	1.0 (-5.8 to 4.3)	0.67
	Pub	<150	41	AM ± SD	21.1 ± 17.2	20.6 ± 16.2	-0.2 (-5.0 to 4.1)	0.98
		≥150	34	AM ± SD	31.3 ± 18.5	33.4 ± 19.9	1.1 (-2.3 to 6.4)	0.32

bc = burning cigarette; AM = arithmetic mean; SD = standard deviation; GM = geometric mean; GSD = geometric standard deviation; IQR = interquartile range.

* *p*-value between before and 1 month after the regulation, Wilcoxon's signed rank test.

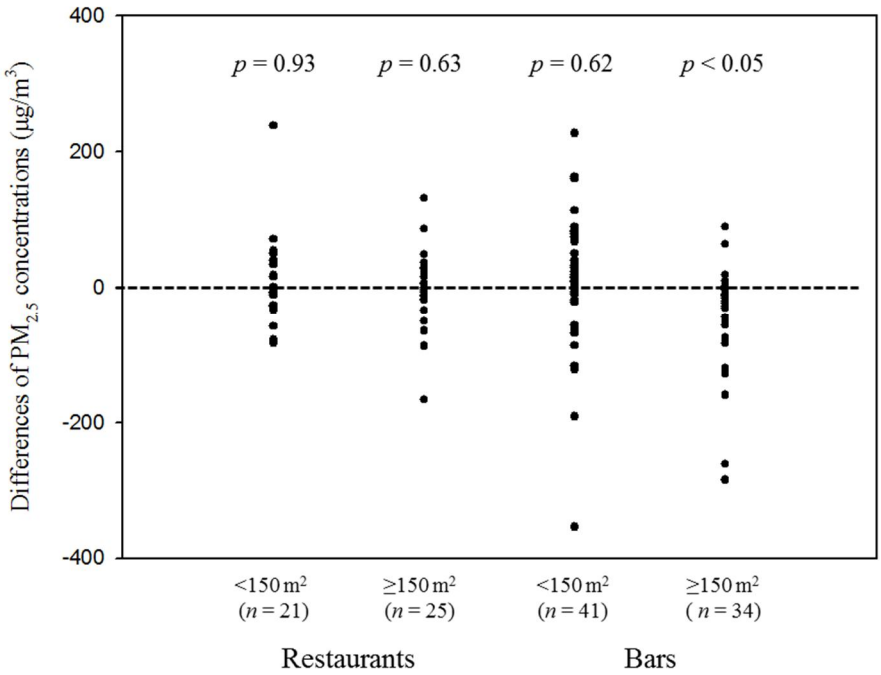

Figure 2-2. Difference in $PM_{2.5}$ concentrations in all restaurants and bars before and after the smoke-free regulation at venues ≥ 150 m^2.

Levels of cotinine and total NNAL

A total of 190 urine samples were collected both before and after the regulation from the 95 staff members at 71 venues. One hundred ninety samples were analysed for cotinine, and 126 of the 190 samples (66%) showed cotinine concentrations below the LOQ. The total NNAL concentrations was analysed for 182 of 190 samples, and all concentrations were above the LOQ. Eight of the 190 samples did not contain enough urine for the total NNAL analysis. A total of 190 samples for cotinine and 182 samples for total NNAL from the venues were included for further analysis.

Urinary cotinine and total NNAL concentrations of staffs were not significantly different between staffs that live with smokers and staff that do not at baseline. The urinary cotinine concentrations among staffs that live with smokers and staff that do not were 1.9 ng/mg creatinine (Cr) (GSD = 2.5) and 1.6 ng/mg Cr (GSD = 2.5), respectively (p = 0.56). The total NNAL concentrations among staffs that live with smokers and staff that do not were 7.7 pg/mg Cr (GSD = 2.3) and 6.3 pg/mg Cr (GSD = 2.4), respectively (p = 0.53).

The urinary cotinine and total NNAL concentrations among 95 staff members are shown in Table 2-3 by type and area of the venues before and after the smoke-free regulation. The cotinine concentrations in venues ≥150 m^2 were not significantly different before and after the regulation (Figure 2-3). However, the cotinine concentrations in bars <150 m^2 (n = 24) slightly increased after the regulation (p = 0.05). The GM of bars <150 m^2 was 2.6

ng/mg Cr (GSD = 3.1) before the regulation and 3.5 ng/mg Cr (GSD = 3.1) after the regulation.

The urinary total NNAL concentrations in bars ≥150 m^2 (n = 14) decreased significantly after the regulation ($p<0.05$) (Figure 2-4). The GM of total NNAL concentrations in bars ≥150 m^2 was 12.1 pg/mg Cr (GSD = 2.0) before the regulation and 7.3 pg/mg Cr (GSD = 1.7) after the regulation. The total NNAL concentrations in restaurants ≥150 m^2 and venues <150 m^2 were not changed after the regulation.

Table 2-3. Urinary cotinine and total NNAL concentrations of staff in all restaurants and bars before and after the smoke-free regulation at venues ≥150 m^2

Substance	Type of venue	Area (m^2)	*n*	Before the regulation	After the regulation	Median changes from before the regulation (IQR)	*p*-value*
				GM (GSD)	GM (GSD)		
Cotinine	Restaurant	<150	21	1.5 (2.0)	1.9 (2.4)	-0.03 (-0.3 to 0.6)	0.50
(ng/mg Cr)		≥150	34	1.3 (1.9)	1.4 (2.4)	0.02 (-0.4 to 1.0)	0.73
	Bar	<150	24	2.6 (3.1)	3.5 (3.1)	1.13 (-0.3 to 4.0)	0.05
		≥150	16	1.9 (2.9)	3.0 (4.1)	0.02 (-1.3 to 2.5)	0.57
Total NNAL	Restaurant	<150	20	6.4 (1.9)	6.2 (2.1)	-0.97 (-2.1 to 3.6)	0.68
(pg/mg Cr)		≥150	31	4.3 (2.3)	4.9 (2.1)	-0.06 (-3.3 to 2.8)	0.97
	Bar	<150	22	9.8 (2.3)	10.4 (2.3)	-0.80 (-3.2 to 4.2)	0.69
		≥150	14	12.1 (2.0)	7.3 (1.7)	-2.44 (-8.6 to 0.8)	<0.05

Cr = creatinine; IQR = interquartile range.
* *p*-value between before and 1 month after the regulation, Wilcoxon's signed rank test.

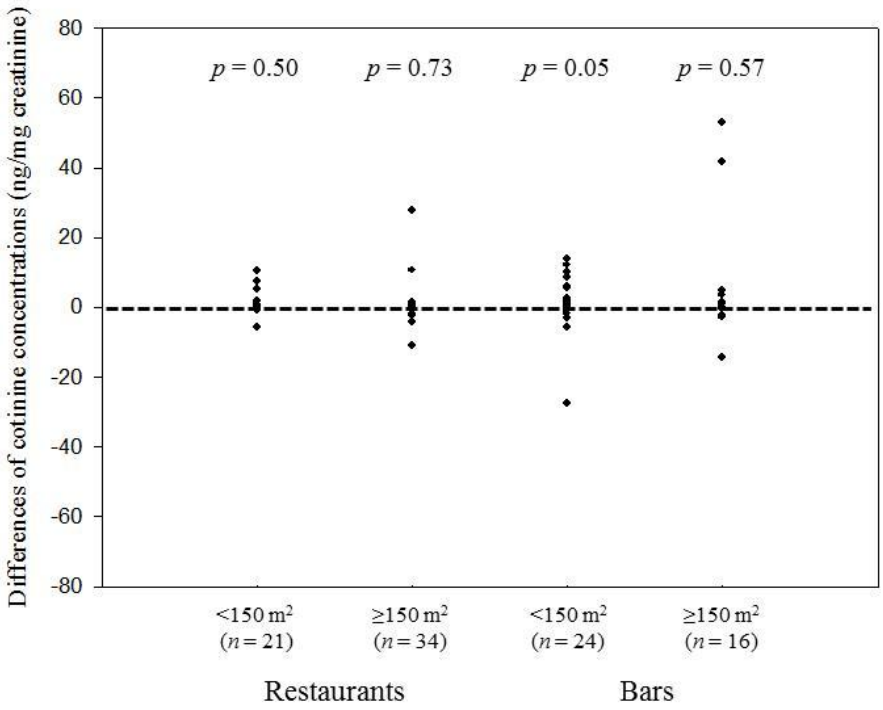

Figure 2-3. Difference in cotinine concentrations of staff members in all restaurants and bars between before and after the smoke-free regulation of venues ≥150 m^2.

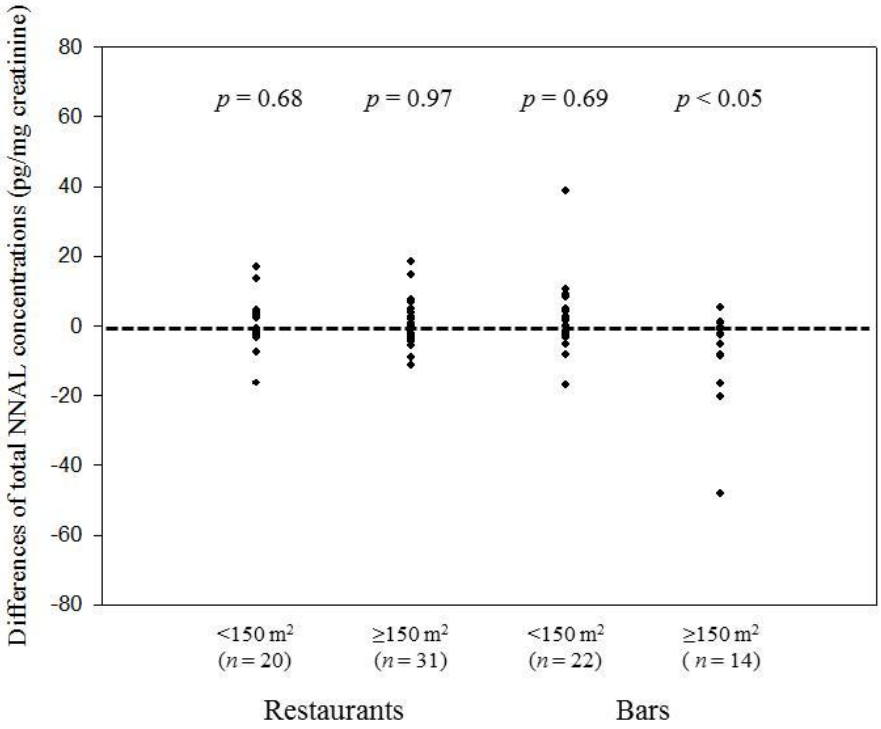

Figure 2-4. Difference in the total NNAL concentrations of staff at all restaurants and bars before and after the smoke-free regulation at venues ≥150 m^2.

Correlations among smoking density and exposure data

According to the Spearman test, smoking density, indoor $PM_{2.5}$, urinary cotinine, and total NNAL concentrations were significantly correlated at the baseline (Table 2-4). Smoking density showed higher correlations with indoor $PM_{2.5}$ concentrations (r = 0.47, n = 121, p<0.01) than cotinine (r = 0.33, n = 76, p<0.01) and total NNAL concentrations (r = 0.42, n = 68, p<0.01). Indoor $PM_{2.5}$ concentrations showed stronger associations with total NNAL concentrations (r = 0.36, n = 68, p<0.01) than with cotinine concentrations (r = 0.31, n = 76, p<0.01). The cotinine and total NNAL concentrations were significantly correlated (r = 0.33, n = 72, p<0.01)

Table 2-4. Spearman correlations among smoking density, indoor $PM_{2.5}$, urinary cotinine and total NNAL concentrations at the baseline

	Smoking density	Indoor $PM_{2.5}$ concentrations	Cotinine concentrations	Total NNAL concentrations
Smoking density	1.00 (121)			
Indoor $PM_{2.5}$ concentrations	0.47 (121)	1.00 (121)		
Cotinine concentrations	0.33 (76)	0.31 (76)	1.00 (80)	
Total NNAL concentrations	0.42 (68)	0.36 (68)	0.33 (72)	1.00 (72)

All variables were statistically associated at the 0.01 significant levels.
The numbers in the parentheses are the number of observations.

Self-reported health effects

The self-reported health effects on respiratory and sensory symptoms were estimated by area regardless of the type of venue due to the low incidence of symptoms in each type of venue. The sensory symptoms among the staff in venues ≥150 m^2 (n = 50) significantly decreased from 52% at baseline to 40% after the regulation (p<0.05), whereas the staff from venues <150 m^2 (n = 45) did not exhibit a significant change in symptoms (p = 0.45) (Table 2-5). The respiratory symptoms did not significantly differ among staff in any venues before and after the regulation.

Table 2-5. Respiratory and sensory symptoms of staff in all restaurants and bars before and after the smoke-free regulation at venues ≥150 m^2

Symptoms	Area (m^2)	*n*	Number with symptoms (%)		Number of changes with symptoms (%)	*p*-value*
			Before the regulation	After the regulation		
Respiratory	<150	45	18 (40)	12 (26)	-6 (-33)	0.15
	≥150	50	18 (36)	13 (26)	-5 (-28)	0.22
Sensory	<150	45	23 (51)	19 (42)	-4 (-17)	0.45
	≥150	50	31 (52)	20 (40)	-11 (-35)	<0.05

* *p*-value between before and 1 month after the regulation, McNemar's test.

$PM_{2.5}$ levels by presence of smoking room

After the smoke-free regulations in restaurants the bars ≥150 m^2, several bars ≥150 m^2 installed smoking room. Of the bars ≥150 m^2 that did not observe smokers after the regulation (n = 24), a smoking room was installed in 7 pubs. The GM of the indoor $PM_{2.5}$ concentrations in bars ≥150 m^2 without (n = 17) and with smoking room (n = 7) was 34.2 μg/m^3 (GSD = 1.9) and 102.1 μg/m^3 (GSD = 2.0), respectively. The indoor $PM_{2.5}$ concentrations in bars ≥150 m^2 with smoking room was significantly higher than those without smoking room (p<0.05, Wilcoxon rank-sum test).

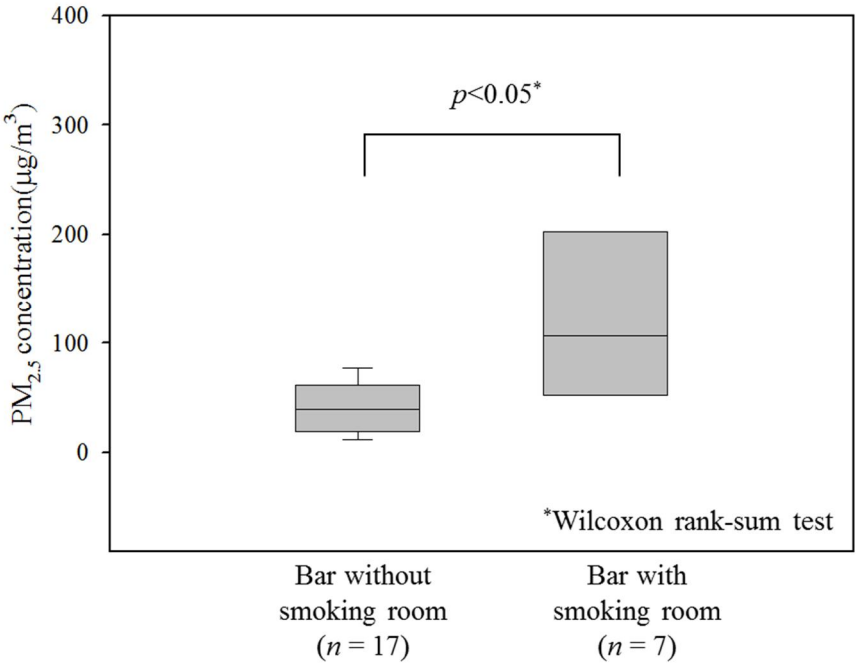

Figure 2-5. Distributions of $PM_{2.5}$ levels in bars ≥150 m^2 without and with smoking room after the smoke-free regulation at venues ≥150 m^2.

Discussion

One month after the smoke-free regulation at restaurants ≥150 m^2 and bars has led to a significant reduction in the indoor $PM_{2.5}$ concentrations in bars ≥150 m^2 while the $PM_{2.5}$ concentrations in restaurants ≥150 m^2 were not different after the regulation. Although the urinary cotinine concentrations of the staff in all venues were not decreased after the regulation, the urinary total NNAL concentrations of the staff in bars ≥150 m^2 were significantly reduced after the regulation. The total NNAL concentrations of the staff in restaurants ≥150 m^2 were not changed after the regulation. The self-reported questionnaires of the health effects on staff show that only sensory symptoms significantly improved in venues ≥150 m^2 after the regulation. However, none of the measured data and health effects showed improvements in venues <150 m^2 after the smoke-free regulation. Overall, the smoke-free regulation at venues ≥150 m^2 had positive effects in bars ≥150 m^2.

The GM of all outdoor $PM_{2.5}$ concentrations both before and after the smoke-free regulation was 32.0 μg/m^3 (GSD = 1.8). Most restaurants and bars involved in this study were located in the commercial area adjacent to the main road in Seoul. The levels of outdoor $PM_{2.5}$ concentrations were similar to those of outdoor $PM_{2.5}$ concentrations in the commercial area in Busan. Busan is the second largest city in Korea. Outdoor $PM_{2.5}$ concentrations were measured from January 2011 to December 2012 by local air quality

monitors.[24] Two-year average outdoor $PM_{2.5}$ concentrations in commercial areas were 28.9 ± 15.5 μg/m^3. Similar outdoor $PM_{2.5}$ levels were reported at bus stops in Seoul.[25] The average outdoor $PM_{2.5}$ concentrations at bus stops were 20.0 ± 11.8 μg/m^3 (n = 100) in November 2011 and 31.9 ± 14.9 μg/m^3 (n = 100) in March 2012.

Of each type and area of venues, the indoor levels of $PM_{2.5}$ concentrations in bars ≥150 m^2 significantly decreased by 41% after the regulation. Although the $PM_{2.5}$ concentrations in restaurants ≥150 m^2 decreased by 19%, this decrease was not statistically significant. The slight changes in the $PM_{2.5}$ concentration in restaurants ≥150 m^2 may have been due to the small sample size of such restaurants with low $PM_{2.5}$ levels at baseline. Because the studied venues that were observed or non-observed smoking included venues that were smaller, larger or equal to 150 m^2 in size between the two sampling phases, additional analyses were conducted by classifying the smoking status of the venues. When restaurants and bars were classified into an observed smoking venue at baseline and non-observed smoking venue during the second phase, the indoor $PM_{2.5}$ concentrations reduced by 39% in restaurants (n = 9) and by 52% in bars (n = 24). The reduced levels of $PM_{2.5}$ concentrations were consistent with those reported in a previous study. The indoor $PM_{2.5}$ concentrations in 41 bars in two Scottish cities were measured before and 2 months after the introduction of a smoke-free regulation.[14] The indoor $PM_{2.5}$ concentrations decreased by 86% from 246 to 20 μg/m^3. In Kentucky, USA, indoor $PM_{2.5}$ concentrations were reduced by 91%, from 199

to 18 μg/m^3 after a comprehensive smoke-free regulation in 10 hospitalities in Lexington.[16]

The urinary cotinine concentrations in venues ≥150 m^2 did not decrease after the regulation. Although a slight increase in cotinine concentrations was observed in bars <150 m^2, the cotinine concentrations among staff in all venues at baseline were close to 2 ng/mg Cr. The results may have been inconsistent with the $PM_{2.5}$ levels because the staff at venues ≥150 m^2 may be intermittently exposed to SHS during times of weak enforcement, such as late at night. When all venues were classified as either an observed smoking venue at baseline or non-observed smoking venue after the regulation (n = 9), the cotinine concentrations were reduced by 36%. Another possible reason might be because low detection rate of urinary cotinine samples, which might be limited to examine changes of the cotinine concentrations of the staffs before and after the regulation. Other studies indicate significantly reduced cotinine concentrations, with higher levels observed at baseline. The GM of the urinary concentrations of 25 non-snuffing, non-smoking staff in Norwegian bars and restaurants was 9.5 μg/g Cr before smoking was banned, and this mean concentration was reduced to 1.4 μg/g Cr 3 months after the smoke-free regulation.[26] The urinary cotinine levels among 40 non-smoking bar staff members decreased from 35.9 to <5 ng/ml after the smoke-free air law in Michigan, USA.[9]

The decrease in the urinary total NNAL concentrations appears to be consistent with the observed decrease in the $PM_{2.5}$ levels. The total NNAL

concentrations were reduced by 40% in bars ≥150 m^2 after the regulation. When bars were classified into observed smoking venues at baseline and non-observed smoking venues after the regulation (n = 7), the total NNAL levels were reduced by 57%. The significantly reduced levels in the total NNAL concentrations observed in this study are consistent with those reported in previous studies of non-smoking staff in hospitality venues. Forty non-smoking bar staff members showed a 60% reduction in levels of NNAL 6-10 weeks after the implementation of the smoke-free regulation in Michigan, USA.[9] In Minnesota, NNAL levels among 24 non-smoking staff members in the hospitality industry (restaurants, bars, bowling alley, and others) were reduced by 57% 4 to 8 week after comprehensive smoke-free laws went to effect.[27]

The changes in the urinary cotinine and total NNAL concentrations were not consistent. Staff members showing changes greater than 10 ng/mg Cr in their cotinine levels exhibited different trends in their total NNAL levels. Although two staff members in bars ≥150 m^2 showed significantly increased cotinine levels (Figure 2-3), their total NNAL levels were slightly decreased. The mean difference in the cotinine and total NNAL concentrations of the two members were 47.3 ng/mg Cr and -3.9 pg/mg Cr, respectively. However, the cotinine and total NNAL levels of one staff member in bars ≥150 m^2 showing reduced cotinine levels of <-10 ng/mg Cr were -14.5 ng/mg Cr and -16.5 pg/mg Cr, respectively. These two biomarkers may have yielded inconsistent results because cotinine more sensitively reflects SHS exposure than does the

total NNAL level. Cotinine, a specific and sensitive biomarker of SHS exposure, has an average half-life of 16 hours and is removed from the body within 3-4 days after the last SHS exposure.[28,29] NNAL, metabolites of the tobacco-specific lung carcinogen NNK, remain in the body longer than cotinine with a half-life of up to 21 days.[29] Although the smoke-free regulation had been introduced in venues ≥150 m^2, the staff at the venues may have intermittently been exposed to SHS, which may be directly reflected in the cotinine levels. However, the total NNAL levels may reflect the overall decrease in SHS exposure after 1 month, although there was intermittent exposure to SHS.

Sources of SHS can be inside (e.g., late at night) and outside the restaurants and bars (e.g., home, outdoor, and other hospitality venue). However, identifying sources of SHS for the staff were limited in the present study. In addition, one of the sources of SHS for staff can be smokers at home. Approximately 40% of the staffs were reported that they were living with smokers. However, differences in cotinine (-0.3 ng/mg Cr) and total NNAL concentrations (-1.4 pg/mg Cr) between staff who live with smokers and staff that do not were not significant. This is might be because smokers living with the staffs smoked outside homes. Thus, staff living with smokers may minimally affect cotinine and total NNAL concentrations.

The smoking density was positively correlated with the indoor $PM_{2.5}$, cotinine, and total NNAL concentrations. The smoking density had the higher correlation coefficients with indoor $PM_{2.5}$ concentrations than with cotinine

and total NNAL concentrations. Smoking density, calculated by the number of burning cigarettes/100 m^3, was commonly used as an indicator for indoor smoking.[15,16] Because smoking density and indoor $PM_{2.5}$ concentrations were measured at the same time and location, the correlation coefficients may be higher than the cotinine and total NNAL concentrations.

$PM_{2.5}$ concentrations measured by the SidePak were adjusted by using a conversion factor of 0.295. The conversion factor that used in the present study was consistent with other study, which average SidePak conversion factor of $PM_{2.5}$ was 0.29 for SHS in a chamber experiment.[30] Although the conversion factor was suitable for SHS, a light-scattering aerosol monitor might vary according to the type of particles and ambient conditions.[31,32] In a study of the non-smoking and smoking sections of the hospitality venues in Louisville, KY, measured values using a light-scattering aerosol monitor were 2.6 to 3.1 times higher than those obtained using the gravimetric method, which would be equivalent to a conversion factor between 0.32 and 0.38.[31] Other study showed that the light-scattering aerosol monitors were subject to overestimation risk at a relative humidity (RH) exceeding 60%.[32] In the present study, 72 of 242 indoor samples on two phases were above 60% of RH with AM of 65.5 ± 3.6%. Because those samples were not significantly higher than 60% of RH, $PM_{2.5}$ samples were not adjusted. Although applying the conversion factor of 0.295 might cause error, such variation is not likely to cause significant effects.

The smoke-free regulation in venues ≥150 m^2 led to immediate beneficial effects on sensory symptoms, whereas respiratory health was not significantly different after the regulation. Because the survey evaluated symptoms before and 1 month after the smoke-free regulation, the intervention period may have been too short to verify respiratory health effects. A previous study conducted a survey 2 months after a regulation. When longitudinal studies were conducted, the self-reported health effects among 191 bar workers decreased from 69% to 57% for respiratory symptoms (p = 0.02) and from 75% to 64% (p = 0.02) for sensory symptoms at 1-year follow-up in Scotland.[20] However, a per-protocol analysis conducted following a smoke-free regulation in Tayside, Scotland, indicated that the respiratory symptoms among 77 non-asthmatic and asthmatic non-smoking bar staff members were significantly reduced from 62.3% before the smoke-free regulation to 41.5% 1 month after the regulation and to 27.3% 2 months after the regulation.[33]

Presence of smoking room in bars ≥150 m^2 was associated with indoor $PM_{2.5}$ concentrations after the regulation. Indoor $PM_{2.5}$ concentrations in bar ≥150 m^2 with smoking room were about 3 times higher than those without smoking room. High levels of $PM_{2.5}$ concentrations in bars ≥150 m^2 with smoking room might be because SHS particles in smoking room drifted into main hall of the bar. Indoor smoking room should be eliminated to protect customer and staffs in these venues from SHS exposure.

This study is the first to evaluate the impact of the Korean smoke-free policy on restaurants and bars. Few studies have simultaneously assessed the

effects of the law by measuring indoor air quality, biomarker levels and self-reported health effects of staff members in restaurants and bars. In addition, this study included non-enforced venues in addition to evaluating enforced venues to examine the influences of the smoke-free regulation at venues ≥150 m^2. The urinary total NNAL, which are metabolites of lung carcinogenic NNK, were used to assess human exposure to SHS.

This study featured several limitations. Restaurants and bars were not randomly selected, which suggests that the results of this study may not be representative of all restaurants and bars. In addition, the exclusion of 25 charcoal-burning restaurants from the $PM_{2.5}$ analysis may have led to biased results. The exposure data and health effects may have been underestimated if more smoking customers frequented excluded venues than those examined in the study.

Although we determined via telephone that all venues allowed smoking, smokers were not observed in several venues. In addition, during the second phase, some venues ≥150 m^2 were observed, and some venues <150 m^2 were not observed. These practices may have obscured the effects of the smoke-free regulation. However, this study represents a more realistic setting because it was conducted quasi-experimentally.

Another limitation is that spot urine samples may have underestimated the cotinine and total NNAL levels. However, other studies have indicated that spot urine may be useful as an alternative to 24 hr urine collection for biomarkers for SHS.[34] The linear correlations for the creatinine-corrected

nicotine equivalents (nicotine-N-glucuronide, cotinine, cotinine-N-glucuronide, trans-3'-hydroxycotinine, and trans-3'-hydroxycotinine-O-glucuronide) and the total NNAL correlated well in these 24 hr samples. Thus, the use of spot urine samples in the current study may have yielded reasonable estimates of cotinine and total NNAL levels.

The 1-month follow-up after the smoke-free regulation may have been too short to evaluate the effects of the smoke-free regulation among non-smoking staff members in restaurants and bars. The urinary cotinine and respiratory symptoms did not significantly decrease. Further study is needed to determine the effect of ongoing regulations, their longer-term health effects, and the possible social determinants of change over time.

Conclusions

Of the 152 restaurants and bars examined in this study, a total of 121 venues and 95 staff members in 71 venues were included to evaluate the effects of the smoke-free regulation in Korea in venues with net indoor floor areas of ≥150 m^2 by measuring indoor air quality, biomarker levels, and health effects. The smoke-free regulation significantly reduced the levels of indoor $PM_{2.5}$ and total NNAL for staff members in bars ≥150 m^2. In addition, the staff at venues ≥150 m^2 reported significantly improved sensory health. The measured data and health effects did not show improvement at venues <150 m^2. The results of this study could be useful to support an expansion of the smoke-free regulation in all indoor public places, including restaurants and bars <150 m^2.

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Chapter 3.

Factors associated with secondhand smoke incursion into the homes of non-smoking residents in a multi-unit housing complex: a cross-sectional study in Seoul, Korea

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Abstract

In a multi-unit housing (MUH) complex, secondhand smoke (SHS) can pass from one living space to another. The aim of this study was to determine the prevalence of SHS incursion, and to establish the relationship between SHS incursion and socio-demographic and built environmental factors in MUH in Korea. A population-based sample of 2,600 residents (aged ≥19 years) living in MUH from across the city of Seoul, Korea, was obtained through a web-based selection panel. The residents completed a questionnaire detailing socio-demographic factors, smoking status, frequency of SHS incursion, and built environmental factors. The presence of a personal smoke-free home rule was determined by residents declaring that no one smoked inside the home. Of the 2,600 participants, non-smoking residents who lived in homes with a personal smoke-free rule were selected for further analysis (n = 1,784). In the previous 12 months, 74.7% of residents had experienced SHS incursion ≥1 times. A multivariate ordinal logistic regression analysis indicated that residents who spent more time at home, lived with children, supported the implementation of smoke-free regulations in MUH, lived in small homes, lived in homes with natural ventilation provided by opening a front door or the windows and front door, and lived in homes with more frequent natural ventilation were more likely to report SHS incursion into their homes. The majority of the non-smoking residents experienced SHS incursion, even with a personal smoke-free rule in their homes.

Introduction

Secondhand smoke (SHS) exposure is causally linked to cardiovascular disease, respiratory effects, and lung cancer.[1-4] Exposure to SHS in children is associated with increased risks of asthma, middle ear infections, and sudden death syndrome in infancy.[4] SHS exposure caused 603,000 premature deaths in 2004, equivalent to 1.0% of worldwide mortality, based on data from 192 countries.[5] The US Surgeon General concluded that there is no risk-free level of SHS exposure and only the elimination of indoor smoking can protect non-smokers.[4]

The extensive evidence of adverse health effects associated with SHS exposure has led many countries to introduce smoke-free regulations in indoor public spaces and work places. The implementation of smoke-free regulations has resulted in an improvement in indoor air quality[6,7] and the health of non-smoking staff in hospitality venues.[8,9] However, there has been a limited implementation of similar regulations in personal living spaces. Although it might be difficult to pass legislation to restrict smoking in a private home, public housing could be smoke-free. Smoke-free public rule of the U.S. Department of Housing and Urban Development went into effects since February 3, 2017.[10] Public Housing Authorities were required to adopt and implement a smoke-free regulation in all of their public housing properties by August 2018.

Residents living in multi-unit housing (MUH) are particularly susceptible to SHS exposure because SHS can be transferred between units in MUH.[11] In 2009, 44.0–46.2% of Americans who lived in smoke-free MUH reported SHS incursion into their units.[12] In Denmark, 28.2% of MUH residents living in non-smoking homes reported that SHS from their neighbors had seeped into their homes.[13] In Hong Kong, 11.8% of students who lived in homes without smokers were experienced SHS in their homes that came from neighboring flats.[14] Because people spend the majority of their time in their homes, SHS exposure at home can be a significant contributor to their total SHS exposure.[4] The prevalence of SHS incursion in MUH in Korea has not been established. Furthermore, most of the studies that have been conducted have examined the relationship between SHS incursion into MUH living spaces and socio-demographic factors. Smoking status, the presence of children living in the home, and the type of MUH have been identified as predictors of SHS incursion.[13,15,16] A previous study reported that up to 65% of the air in a private unit could come from somewhere else in the building depending on the construction and age of building.[17] The aim of this study was to determine the prevalence of SHS incursion in MUH and to establish the relationship between SHS incursion into the homes of non-smoking residents and socio-demographic and built environmental factors.

Methods

Samples

The study was approved by Seoul Medical Center's institutional review board (IRB No. 2015-051). Because we used a web-based survey using internet panelists who voluntarily enrolled in the survey company, written informed consent of the panelists was not necessary. The study included internet panelists (≥19 years) who lived in MUH in Seoul, Korea. The MUH in the study included apartments and attached homes. In Korea, an apartment is defined as a unit in a building with five or more stories, similar to a high-rise condominium building in the US. An attached home is a unit in a multi-family building less than five stories tall. Data were collected from 21 August to 4 September 2015. Using August 2015 population statistics from the residential registry of the Ministry of the Interior (MI),[18] quotas were calculated for sex, age, and residential region that corresponded to the Seoul population. Although the proportion of residents in the various categories differed between apartments and attached homes, we considered that about 50% of each category was present in each type of residence, enabling us to determine whether housing type played an important role in SHS incursion. A flow chart describing the selection of final study sample is shown in Figure 3-1. Of the more than 300,000 panelists, 11,788 people were selected for the study because they had participated in web-based survey within the previous 12 months. Of these 4,578 accepted the invitation to participate, 3,762 began

the questionnaire. Of these 3,762 residents, 187 did not complete the questionnaire, and 547 were screened out because they did not live in MUH. Thus, a total of 3,028 residents completed the questionnaire and were evaluated further. Of these 3,028 residents, 351 answered the open-ended questions inadequately and were excluded, and a further 77 were screened out to meet the quotas. Ultimately, 2,600 residents were included in the final analysis.

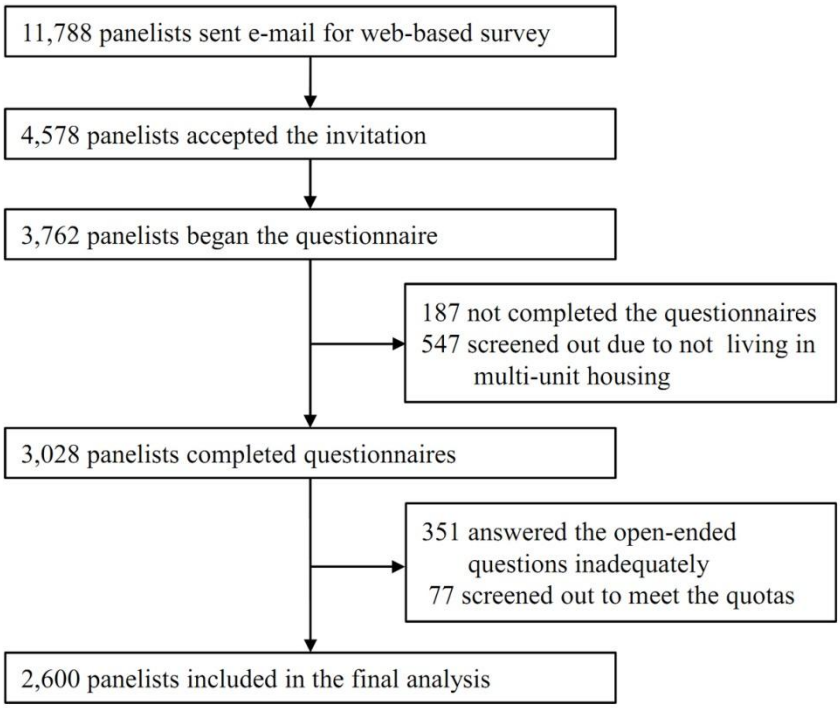

Figure 3-1. Flow chart toward the final study sample.

The initial sample size that needed to provide 95% confidence intervals (CI) with a margin of error of 0.03 was calculated to be 1,067. Because non-smoking homes accounted for 41–51% of all MUH units,[19] we collected more samples than our required initial sample size.

Socio-demographic factors

The self-reported socio-demographic factors investigated were sex, age, household income, education, housing type, time spent at home, number of residents, children aged ≤18 years living in home, type of ownership, duration in current residence, presence of other smokers inside home (i.e., family members or regular visitors), support for the implementation of smoke-free regulations in MUH, and living in a home with a personal smoke-free rule. Respondents were determined to be living in a home with a personal smoke-free rule if they indicated that they lived in a home in which no one smoked inside. Therefore, the homes with a personal smoke-free rule included homes without smokers or homes with smokers, but smokers were not allowed to smoke inside homes.

Smoking status

Residents were asked whether they were currently smoking "every day," "sometimes," "in the past but not currently," or "never." Residents were classified as non-smokers if they reported smoking "in the past but not currently" or "never."

Frequency of SHS incursion

Residents were asked how often they could smell tobacco smoke that entered their living space from somewhere else in or around their building during a 12-month period. The possible responses were “never,” “once a month or less,” “twice a month,” “four times a month,” “two to four times a week,” or “every day.” A similar question was used in a previous study.[15] When a resident indicated that they had experienced SHS incursion within the previous 12 months, we asked them where the SHS had entered and gave them the following options: “balcony,” “window,” “bathroom,” “front door,” or “other location.”

Built environmental factors

Residents were asked to identify various built environmental factors in the MUH. The environmental factors investigated were date of construction, type of corridor, home size, presence of balcony, presence of air conditioning, method of natural ventilation, and the frequency of natural ventilation. Date of construction might be associated with SHS incursion because air that contained SHS particles could be infiltrated from other unit or the building envelope.[17,20] Other factors might be associated with SHS incursion due to resident’s behavior at homes (e.g., method and frequency of natural ventilation).[21]

Statistical analysis

For the statistical analyses, the self-reported frequency of SHS incursion in MUH was classified into four ordinal categories (never or ≤1, 2–4, or >4 times/month); similar proportions were found in all categories. A chi-square test was used to compare residents who were smokers and non-smokers according to socio-demographic factors and the frequency of SHS incursion. The Cochran-Mantel-Haenszel test was used to select potential socio-demographic and built environmental factors on SHS incursion. Using the variables identified in the Cochran-Mantel-Haenszel test ($p<0.1$), ordinal logistic regression analysis was used to assess the relationships between SHS incursion and the variables. Because sex might be potential factors that affect the observed associations, sex was included in the multivariate ordinal logistic model. The score test for the proportional odds assumption in the ordinal regression models was conducted to confirm or reject the assumption. When the assumption was violated ($p<0.05$), partial proportional odds model was fit. Odds ratios (ORs) for the variables in the model were reported with a 95% CI. A p-value 0.05 was considered significant in all analyses. SAS 9.2 software (SAS Institute, Inc., Cary, NC, USA) was used for all statistical analyses.

Results

The distributions of sex, age, and residential region in the Seoul population obtained from the MI[18] and the population in this study are shown in Table 3-1. The distributions of sex and residential region in the study population were similar to those of the Seoul population. The study population was slightly younger, on average, than the Seoul population.

Table 3-1. Distributions of sex, age, and residential region in Seoul and study population

	Seoul population (%; *n* = 7,018,172)*	Study population (%; *n* = 2,600)
Sex		
Men	49.6	49.8
Women	50.4	50.2
Age (years)		
19–29	20.7	22.7
30–39	24.0	26.0
40–49	24.5	26.6
≥50	30.8	24.7
Region		
Urban areas	5.1	5.0
Northeast	31.2	31.4
Northwest	11.8	11.7
Southeast	30.5	30.4
Southwest	21.4	21.6

*The *Statistics* of the Registered Population in August, 2015.[18]

The relationship of socio-demographic factors with the frequency of SHS incursion for smoking and non-smoking residents is shown in Table 3-2. A total of 74.8% of the residents were non-smokers. Women were more likely than men to be non-smokers (62.1%, $p<0.001$). Non-smokers were older ($p<0.001$) and had lower household incomes ($p = 0.035$) compared with smokers. Non-smokers were more likely than smokers to live in an apartment (51.2%, $p<0.001$) and to spend more time at home ($p<0.001$), and were less likely to live with children (38.6%, $p = 0.018$). Non-smokers were likely to have been residents for a longer period ($p = 0.019$). Non-smokers were more likely than smokers to support the implementation of smoke-free regulations in MUH (89.9%, $p<0.001$), and to live in homes with a personal smoke-free rule (72.1%, $p<0.001$). Non-smokers were more likely than smokers to have reported an SHS incursion within the previous 12 months ($p<0.001$). However, level of educational attainment, number of residents, type of ownership, and presence of other smokers inside a home did not differ between smoking and non-smoking residents.

Table 3-2. Characteristics between smoking and non-smoking resident in multi-unit housing

	Total (%)	Smoker (%)	Non-smoker (%)	*p*-value
Overall	2600 (100.0)	654 (25.2)	1946 (74.8)	
Sex				
Men	1294 (49.8)	557 (85.2)	737 (37.9)	<0.001
Women	1306 (50.2)	97 (14.8)	1209 (62.1)	
Age (years)				
19–29	590 (22.7)	128 (19.6)	462 (23.7)	<0.001
30–39	677 (26.0)	187 (28.6)	490 (25.2)	
40–49	692 (26.6)	204 (31.2)	488 (25.1)	
≥50	641 (24.7)	135 (20.6)	506 (26.0)	
Household income (USD/month)				
<2,000	188 (7.2)	37 (5.7)	151 (7.8)	0.035
2,000–3,999	759 (29.2)	184 (28.1)	575 (29.5)	
4,000–5,999	963 (37.0)	235 (35.9)	728 (37.4)	
6,000–7,999	405 (15.6)	108 (16.5)	297 (15.3)	
≥8,000	285 (11.0)	90 (13.8)	195 (10.0)	
Education				
Less than university level	908 (34.9)	217 (33.2)	691 (35.5)	0.418
University level	1461 (56.2)	382 (58.4)	1079 (55.4)	
More than university level	231 (8.9)	55 (8.4)	176 (9.0)	
Housing type				<0.001
Apartment	1302 (50.1)	306 (46.8)	996 (51.2)	
Attached home	1298 (49.9)	348 (53.2)	950 (48.8)	
Time spent at home (hours/day)				
<5	716 (27.5)	233 (35.6)	483 (24.8)	<0.001
5–9	1346 (51.8)	338 (51.7)	1008 (51.8)	
≥10	538 (20.7)	83 (12.7)	455 (23.4)	
Number of residents (people)				
<4	1280 (49.2)	343 (52.4)	937 (48.2)	0.057
≥4	1320 (50.8)	311 (47.6)	1009 (51.8)	
Children living in home (aged ≤18 years)				
No	1561 (60.0)	367 (56.1)	1194 (61.4)	0.018
Yes	1039 (40.0)	287 (43.9)	752 (38.6)	
Type of ownership				
Owned	1475 (56.7)	354 (54.1)	1121 (57.6)	0.179
Leased based on deposit	772 (29.7)	199 (30.4)	573 (29.4)	
Monthly rent	353 (13.6)	101 (15.4)	252 (12.9)	
Duration of residence (years)				
<2	699 (26.9)	166 (25.4)	533 (27.4)	0.019
2–3	514 (19.8)	154 (23.5)	360 (18.5)	
≥4	1387 (53.3)	334 (51.1)	1053 (54.1)	
Presence of other smokers inside the home*				
No	1635 (62.9)	394 (60.2)	1241 (63.8)	0.819
Yes	965 (37.1)	260 (39.8)	705 (36.2)	

*Smokers among family members or regular visitors to the home.

Table 3-2. Characteristics between smoking and non-smoking resident in multi-unit housing (continued)

	Total (%)	Smoker (%)	Non-smoker (%)	*p*-value
Support for the implementation of smoke-free regulations in MUH				
No	438 (16.8)	242 (37.0)	196 (10.1)	<0.001
Yes	2162 (83.2)	412 (63.0)	1750 (89.9)	
Living in a home with a personal smoke-free rule				
No	976 (37.5)	433 (66.2)	543 (27.9)	<0.001
Yes	1624 (62.5)	221 (33.8)	1403 (72.1)	
Frequency of SHS incursion				
Never	743 (28.6)	243 (37.2)	500 (25.7)	<0.001
≤1 times/month	504 (19.4)	108 (16.5)	396 (20.3)	
2–4 times/month	657 (25.3)	165 (25.2)	492 (25.3)	
>4 times/month	696 (26.8)	138 (21.1)	558 (28.7)	

The proportions of general smoking locations in smokers' homes were estimated using data from residents who were either smokers or resided with smokers (n = 1,359). Among the residents who smoked at their homes (n = 560), the most common smoking location was the balcony (51.4%, n = 288), followed by the bathroom (20.2%, n = 113), main room (14.8%, n = 83), and outside the front door (13.6%, n = 76).

Although there was no difference in SHS incursion between the non-smoking residents who lived in homes with and without a personal smoke-free rule (p = 0.568), only non-smoking residents who lived in homes with a personal smoke-free rule were used for further analysis (n = 1,784). In total, 74.7% of these non-smoking residents (n = 1,333) reported that they had experienced SHS incursion into their home within the previous 12 months. In total, 9.9% of the residents (n = 176) reported that they had experienced SHS incursion every day, and 44.2% (n = 788) reported that they had experienced SHS incursion once a week or more. The residents who had experienced SHS

incursion reported the entry point of SHS into their homes (n = 1,333); the main source of SHS incursion was the balcony (45.7%, n = 609), followed by windows (28.4%, n = 378), bathroom (12.9%, n = 172), front door (11.7%, n = 156), and other locations (1.4%, n = 18).

Table 3-3 shows characteristics of the non-smoking residents living in home with a personal smoke-free rule by frequency of SHS incursion. Residents who were women (p = 0.020), spent more time at home (p<0.001), lived with children (p<0.001), and supported the implementation of smoke-free homes in MUH (p = 0.020) exhibited a positive trend across the categories of SHS incursion. Residents who lived in large homes exhibited a negative trend across the categories (p = 0.038). Method of natural ventilation at residents' homes was related to frequency of SHS incursion (p = 0.042). Residents who lived in homes with more frequent natural ventilation exhibited a positive trend across the categories (p<0.001). However, age, household income, level of educational attainment, housing type, number of residents, type of ownership, duration of residential period, presence of other smokers inside the home, date of construction, type of corridor, presence of a balcony, and air-conditioning were not significantly associated with frequency of SHS incursion.

Table 3-3. Characteristics of the non-smoking residents living in home with smoke-free rules by SHS incursion (n = 1784)

	Total (%)	Frequency of SHS incursion				
		Never (%)	≤1 times/month (%)	2-4 times/month (%)	>4 times/month (%)	*p*-value*
Socio-demographic factor						
Sex						
Men	687 (38.5)	180 (39.9)	156 (42.6)	180 (39.8)	171 (33.2)	0.020
Women	1097 (61.5)	271 (60.1)	210 (57.4)	272 (60.2)	344 (66.8)	
Age (years)						
19–29	404 (22.6)	110 (24.4)	74 (20.2)	107 (23.7)	113 (21.9)	0.079
30–39	464 (26.0)	104 (23.1)	86 (23.5)	132 (29.2)	142 (27.6)	
40–49	442 (24.8)	92 (20.4)	103 (28.1)	108 (23.9)	139 (27.0)	
≥50	474 (26.6)	145 (32.2)	103 (28.1)	105 (23.2)	121 (23.5)	
Household income (USD/month)						
<2,000	140 (7.8)	47 (10.4)	22 (6.0)	23 (5.1)	48 (9.3)	0.171
2,000–3,999	528 (29.6)	138 (30.6)	107 (29.2)	135 (29.9)	148 (28.7)	
4,000–5,999	655 (36.7)	169 (37.5)	133 (36.3)	171 (37.8)	182 (35.3)	
6,000–7,999	272 (15.2)	50 (11.1)	68 (18.6)	76 (16.8)	78 (15.1)	
≥8,000	189 (10.6)	47 (10.4)	36 (9.8)	47 (10.4)	59 (11.5)	
Education						
Less than university level	620 (34.8)	168 (37.3)	110 (30.1)	152 (33.6)	190 (36.9)	0.698
University level	991 (55.5)	246 (54.5)	212 (57.9)	247 (54.6)	286 (55.5)	
More than university level	173 (9.7)	37 (8.2)	44 (12.0)	53 (11.7)	39 (7.6)	
Housing type						
Apartment	921 (51.6)	226 (50.1)	212 (57.9)	243 (53.8)	240 (46.6)	0.147
Attached house	863 (48.4)	225 (49.9)	154 (42.1)	209 (46.2)	275 (53.4)	
Time spent at home (hours/day)						
<5	452 (25.3)	133 (29.5)	100 (27.3)	116 (25.7)	103 (20.0)	<0.001
5–9	923 (51.7)	236 (52.3)	183 (50.0)	236 (52.2)	268 (52.0)	
≥10	409 (22.9)	82 (18.2)	83 (22.7)	100 (22.1)	144 (28.0)	

*The Cochran-Mantel-Haenszel test.

Table 3-3. Characteristics of the non-smoking residents living in home with smoke-free rules by SHS incursion (n = 1784) (continued)

	Total (%)	Frequency of SHS incursion				
		Never (%)	≤1 times/month (%)	2-4 times/month (%)	>4 times/month (%)	*p*-value*
Number of residents (people)						
<4	875 (49.0)	234 (51.9)	182 (49.7)	209 (46.2)	250 (48.5)	0.207
≥4	909 (51.0)	217 (48.1)	184 (50.3)	243 (53.8)	265 (51.5)	
Children living in home (aged ≤18 years)						
No	1089 (61.0)	316 (70.1)	216 (59.0)	264 (58.4)	293 (56.9)	<0.001
Yes	695 (39.0)	135 (29.9)	150 (41.0)	188 (41.6)	222 (43.1)	
Type of ownership						
Owned	1024 (57.4)	255 (56.5)	216 (59.0)	268 (59.3)	285 (55.3)	0.830
Leased based on deposit	533 (29.9)	126 (27.9)	110 (30.1)	135 (29.9)	162 (31.5)	
Monthly rent	227 (12.7)	70 (15.5)	40 (10.9)	49 (10.8)	68 (13.2)	
Duration of residence (years)						
<2	496 (27.8)	137 (30.4)	90 (24.6)	109 (24.1)	160 (31.1)	0.495
2–3	334 (18.7)	80 (17.7)	62 (16.9)	93 (20.6)	99 (19.2)	
≥4	954 (53.5)	234 (51.9)	214 (58.5)	250 (55.3)	256 (49.7)	
Presence of other smokers inside home†						
No	1241 (69.6)	329 (72.9)	260 (71.0)	293 (64.8)	359 (69.7)	0.116
Yes	543 (30.4)	122 (27.1)	106 (29.0)	159 (35.2)	156 (30.3)	
Support for the implementation of smoke-free regulations in MUH						
No	160 (9.0)	56 (12.4)	25 (6.8)	42 (9.3)	37 (7.2)	0.020
Yes	1624 (91.0)	395 (87.6)	341 (93.2)	410 (90.7)	478 (92.8)	
Built environmental factor						
Date of construction (year)						
Before 1995	487 (27.3)	123 (27.3)	98 (26.8)	112 (24.8)	154 (29.9)	0.322
1995–1999	419 (23.5)	107 (23.7)	82 (22.4)	112 (24.8)	118 (22.9)	
2000–2004	393 (22.0)	97 (21.5)	82 (22.4)	105 (23.2)	109 (21.2)	
2005–2009	262 (14.7)	57 (12.6)	66 (18.0)	61 (13.5)	78 (15.1)	
2010 or later	223 (12.5)	67 (14.9)	38 (10.4)	62 (13.7)	56 (10.9)	

*The Cochran-Mantel-Haenszel test.

†Smokers among family members or regular visitors to the home.

Table 3-3. Characteristics of the non-smoking residents living in home with smoke-free rules by SHS incursion (n = 1784) (continued)

	Total (%)	Frequency of SHS incursion				
		Never (%)	≤1 times/month (%)	2-4 times/month (%)	>4 times/month (%)	p-value*
Type of corridor						
Stairwell	1376 (77.1)	355 (78.7)	287 (78.4)	339 (75.0)	395 (76.7)	0.142
Indoor corridor	250 (14.0)	64 (14.2)	49 (13.4)	68 (15.0)	69 (13.4)	
Outdoor corridor	158 (8.9)	32 (7.1)	30 (8.2)	45 (10.0)	51 (9.9)	
Home size (m^2)						
<66	450 (25.2)	121 (26.8)	83 (22.7)	100 (22.1)	146 (28.3)	0.038
66–98	639 (35.8)	147 (32.6)	119 (32.5)	172 (38.1)	201 (39.0)	
≥99	695 (39.0)	183 (40.6)	164 (44.8)	180 (39.8)	168 (32.6)	
Presence of balcony						
No	394 (22.1)	105 (23.3)	93 (25.4)	90 (19.9)	106 (20.6)	0.128
Yes	1390 (77.9)	346 (76.7)	273 (74.6)	362 (80.1)	409 (79.4)	
Presence of air–conditioning						
No	327 (18.3)	88 (19.5)	62 (16.9)	66 (14.6)	111 (21.6)	0.558
Yes	1457 (81.7)	363 (80.5)	304 (83.1)	386 (85.4)	404 (78.4)	
Method of natural ventilation						
Opening windows	941 (52.7)	250 (55.4)	214 (58.5)	226 (50.0)	251 (48.7)	0.042
Opening front doors	95 (5.3)	19 (4.2)	16 (4.4)	29 (6.4)	31 (6.0)	
Opening both windows and front doors	326 (18.3)	73 (16.2)	54 (14.8)	100 (22.1)	99 (19.2)	
Windows always slightly open	422 (23.7)	109 (24.2)	82 (22.4)	97 (21.5)	134 (26.0)	
Frequency of natural ventilation (times/week)						
<5	569 (31.9)	155 (34.4)	141 (38.5)	148 (32.7)	125 (24.3)	<0.001
≥5	1215 (68.1)	296 (65.6)	225 (61.5)	304 (67.3)	390 (75.7)	

*The Cochran-Mantel-Haenszel test.

The univariate and multivariate ordinal logistic regression model of SHS incursion are shown in Table 3-4. In the univariate analysis, the proportional odds assumption was violated for home size (p = 0.049) and frequency of natural ventilation (p = 0.019); thus, different effects in these variables were estimated for the different levels of frequency of SHS incursion. In the multivariate analysis, all variables except sex seemed consistent effects with univariate analysis on SHS incursion. Residents who spent 5–9 hours/day and those who spent ≥10 hours/day at home were more likely to report SHS incursion than were those who spent <5 hours/day at home. Residents who lived with children and those who supported the implementation of smoke-free regulations in MUH were more likely to report SHS incursion than were those who did not. Residents who lived in home sized ≥99 m^2 were less likely to report SHS incursion in the highest SHS incursion categories than were those who lived in home sized <66 m^2. Residents who lived in homes with natural ventilation provided by open front doors or both open windows and front doors were more likely to report SHS incursion than were those with only open windows. Residents who lived in homes with a natural ventilation frequency of ≥5 times/week were more likely to report SHS incursion in the 2 highest SHS categories and in the highest SHS incursion categories than were those who lived in homes with ventilation frequency of <5 times/week.

Table 3-4. Factors associated with SHS incursion among non-smoking residents living in home with smoke-free rules*

	Univariate		Multivariate	
	OR (95% CI)†	*p*-value	OR (95% CI)‡	*p*-value
Socio-demographic factor				
Sex				
Men	1.00		1.00	
Women	**1.23 (1.04-1.46)**	**0.018**	1.11 (0.93-1.32)	0.273
Age (years)				
19–29	1.00		1.00	
30–39	1.20 (0.95-1.53)	0.133	1.10 (0.86-1.41)	0.436
40–49	1.19 (0.94-1.52)	0.157	0.96 (0.73-1.26)	0.768
≥50	0.82 (0.65-1.04)	0.107	0.79 (0.62-1.01)	0.064
Time spent at home (hours/day)				
<5	1.00		1.00	
5–9	**1.29 (1.05-1.58)**	**0.014**	**1.31 (1.06-1.60)**	**0.011**
≥10	**1.68 (1.32-2.14)**	**<0.001**	**1.64 (1.28-2.12)**	**<0.001**
Children living in home (aged ≤18 years)				
No	1.00		1.00	
Yes	**1.41 (1.19-1.67)**	**<0.001**	**1.35 (1.11-1.65)**	**0.003**
Support for the implementation of smoke-free regulations in MUH				
No	1.00		1.	
Yes	**1.43 (1.07-1.91)**	**0.017**	**1.46 (1.09-1.97)**	**0.012**
Built environmental factor				
Home size (m^2)				
<66	1.00		1.00	
66-98				
OR 1§	1.23 (0.93-1.63)	0.730	1.19 (0.89-1.57)	0.240
OR 2‖	1.16 (0.91-1.48)	0.224	1.09 (0.85-1.39)	0.518
OR 3¶	0.96 (0.74-1.24)	0.143	0.90 (0.69-1.17)	0.412
≥99				
OR 1§	1.03 (0.79-1.35)	0.835	1.03 (0.78-1.37)	0.821
OR 2‖	0.83 (0.66-1.06)	0.129	0.80 (0.62-1.03)	0.080
OR 3¶	**0.66 (0.51-0.86)**	**0.002**	**0.64 (0.48-0.84)**	**0.001**
Method of natural ventilation				
Opening windows	1.00		1.00	
Opening front doors	1.46 (1.00-2.14)	0.051	**1.65 (1.12-2.43)**	**0.011**
Opening both windows and front doors	**1.32 (1.05-1.65)**	**0.017**	**1.29 (1.03-1.62)**	**0.028**
Windows always slightly open	1.17 (0.96-1.44)	0.128	1.18 (0.96-1.46)	0.120
Frequency of natural ventilation (times/week)				
<5	1.00		1.00	
≥5				
OR 1§	1.16 (0.93-1.46)	0.193	1.13 (0.90-1.43)	0.306
OR 2‖	**1.44 (1.18-1.76)**	**<0.001**	**1.44 (1.17-1.77)**	**<0.001**
OR 3¶	**1.68 (1.33-2.12)**	**<0.001**	**1.66 (1.31-2.10)**	**<0.001**

ORs with $p<0.05$ are in bold.

*Cumulative logistic models were used when the proportion odds assumption was retained and partial proportional odds models were used when the assumption was violated. Proportional odds assumption is violated for home size (p = 0.049) and frequency of natural ventilation (p = 0.019) but others were met the assumption (p >0.05).

†Unadjusted OR.

‡Adjusted OR: adjusted for all variables listed in the table.

§OR 1: >4, 2-4, or ≤1 times/month vs. never.

‖OR 2: >4 or 2-4 times/month vs. ≤1 times/month or never.

¶OR 3: >4 times/month vs. 2-4 or ≤1 times/month or never.

We further conducted an ordinal logistic regression analysis among smoking residents living in home with a smoke-free rule (n = 433). In the univariate analysis, the proportional odds assumption was violated for age (p = 0.029); thus, different effects in this variable were estimated for the different levels of frequency of SHS incursion. In the multivariate analysis, all variables except age seemed consistent effects with univariate analysis on SHS incursion. In the multivariate analysis, residents who lived with children (OR = 1.63, 95% CI = 1.09–2.44) and those who supported the implementation of smoke-free regulations in MUH (OR = 2.09, 95% CI = 1.35–3.25) were more likely to report SHS incursion than were those who did not. Residents who lived in homes with indoor corridor (OR = 1.69, 95% CI = 1.06–2.70) and those who lived in homes with natural ventilation provided by both windows and front doors (OR = 2.74, 95% CI = 1.73–4.34) or those with always slightly open windows (OR = 2.33, 95% CI = 1.47–3.71) were more likely to report SHS incursion than a reference value (Table 3-5). Other variables were not significantly associated with SHS incursion.

Table 3-5. Factors associated with SHS incursion among smoking residents living in home with smoke-free rules*

	Univariate		Multivariate	
	OR (95% CI) †	*p*-value	OR (95% CI) ‡	*p*-value
Socio-demographic factor				
Sex				
Men	1.00		1.00	
Women	1.16 (0.68-1.97)	0.596	1.04 (0.58-1.88)	0.885
Age (years)				
19–29	1.00			
30–39				
OR 1§	1.51 (0.83-2.75)	0.176	1.39 (0.72-2.69)	0.326
OR 2‖	**1.80 (1.02-3.17)**	**0.044**	1.64 (0.88-3.07)	0.123
OR 3¶	1.25 (0.64-2.43)	0.518	1.03 (0.50-2.12)	0.937
40–49				
OR 1§	1.39 (0.76-2.52)	0.283	0.97 (0.49-1.94)	0.933
OR 2‖	1.14 (0.65-2.00)	0.660	0.79 (0.41-1.55)	0.497
OR 3¶	0.79 (0.39-1.60)	0.519	0.53 (0.24-1.17)	0.115
≥50				
OR 1§	0.67 (0.37-1.22)	0.190	0.55 (0.28-1.00)	0.073
OR 2‖	0.66 (0.36-1.21)	0.182	0.54 (0.28-1.05)	0.069
OR 3¶	0.95 (0.46-1.96)	0.898	0.78 (0.37-1.67)	0.526
Children living in home (aged ≤18 years)				
No	1.00		1.00	
Yes	**1.42 (1.01-2.00)**	**0.043**	**1.63 (1.09-2.44)**	**0.017**
Support for the implementation of smoke-free regulations in MUH				
No	1.00		1.000	
Yes	**2.39 (1.57-3.64)**	**<0.001**	**2.09 (1.35-3.25)**	**0.001**
Built environmental factor				
Type of corridor				
Stairwell	1.00		1.00	
Indoor corridor	**2.04 (1.29-3.22)**	**0.002**	**1.69 (1.06-2.70)**	**0.027**
Outdoor corridor	**2.46 (1.31-4.59)**	**0.005**	1.83 (0.91-3.71)	0.092
Method of natural ventilation				
Opening windows	1.00		1.00	
Opening front doors	2.09 (0.93-4.70)	0.073	1.97 (0.90-4.30)	0.091
Opening both windows and front doors	**3.08 (2.01-4.72)**	**<0.001**	**2.74 (1.73-4.34)**	**<0.001**
Windows always slightly open	**2.23 (1.42-3.50)**	**<0.001**	**2.33 (1.47-3.71)**	**<0.001**

ORs with $p<0.05$ are in bold.

*Cumulative logistic models were used when the proportion odds assumption was retained and partial proportional odds models were used when the assumption was violated. Proportional odds assumption is violated for age ($p = 0.029$) but others were met the assumption ($p > 0.05$).

†Unadjusted OR.

‡Adjusted OR: adjusted for all variables listed in the table.

§OR 1: >4, 2-4, or ≤1 times/month vs. never.

‖OR 2: >4 or 2-4 times/month vs. ≤1 times/month or never.

¶OR 3: >4 times/month vs. 2-4 or ≤1 times/month or never.

Discussion

Among the non-smokers who lived in homes with a personal smoke-free rule, 74.7% had experienced SHS incursion within the previous 12 months. One in 10 residents reported that they experienced daily SHS incursion. The prevalence of SHS incursion in this study was higher than that reported in previous studies. In a 2010 study in the US, 44% of residents in MUH with a personal smoke-free home rule had experienced SHS incursion in their units within the previous 12 months.[22] In that study, the smoking rate of the residents was 21.1%. In a 2009 study in New York State, 46.2% of residents with a personal smoke-free home policy had experienced SHS incursion in their unit within the previous 12 months.[15] The smoking rate of the study population was 19.0%. A possible reason for the high prevalence of SHS incursion in the present study might be because smoking rate in this study was higher than that in previous studies conducted in the USA.

The majority of non-smoking residents who had experienced SHS incursion within the past 12 month reported that SHS entered their homes through the balcony or windows. The ingress route taken by SHS incursion was slightly higher in bathrooms than through the front door. SHS could migrate through the balcony,[15] hallway (similar to a corridor),[11] and bathroom ceiling exhaust fans.[23] In this study, it was suggested that SHS incursion into bathrooms might have been associated with migration of SHS through

bathroom ceiling exhaust fans in other units. A front door was associated with migration of SHS from the corridor outside a home.

In this study, the source of SHS incursion was consistent with the smoking locations used by smokers in their homes in MUH. The most common smoking location was the balcony, followed by the bathroom, main room, and outside the front door. This suggested that smoking in these locations might be associated with SHS incursion into other units. Therefore, limitations on smoking in these locations should be placed to reduce the SHS incursion into other units in MUH. Because it might be difficult to implement smoke-free regulations in MUH, offering educational information on how to implement smoke-free policy to building managers or owners could be the first step for smoke-free MUH.[24]

In the multivariate analysis, residents who spent more time at home were more likely to report SHS incursion. As the time spent at home increased, the ORs of SHS incursion also tended to increase. As residents spend more time in their home, they are more likely to be exposed to SHS incursion. Thus, MUH residents who spend long periods at home might be at risk of high SHS exposure from such incursion.

Residents who lived with children and who supported the implementation of smoke-free regulations in MUH were more likely to report SHS incursion. MUH residents who lived with children might be more sensitive to SHS incursion because their children are being exposed to SHS.[13] MUH residents who experienced a high level of SHS incursion might express more support

for smoke-free regulations in MUH so as to reduce their SHS exposure at home.

Among the built environmental factors investigated here, home size was significantly associated with SHS incursion. Overall, residents who lived in homes ≥99 m^2 in size were less likely to report SHS incursion than were those in homes of <66 m^2. This might be because home size was associated with housing type. In Korea, the average home size per person was larger in an apartment than in an attached home in 2010.[25] In the present study, residents who lived in an apartment were slightly less likely to report SHS incursion than were those in an attached home. Therefore, residents who lived in larger homes were more likely to live in an apartment and might therefore be less likely to experience SHS incursion.

Factors related to natural ventilation were associated with SHS incursion. Residents who lived in homes with natural venation provided by opening the front door or by opening both the front door and windows were more likely to report SHS incursion than were those with natural venation provided only by opening the windows. The ORs for providing natural ventilation with an open front door were higher than those where natural ventilation was provided by opening both windows and front doors. Furthermore, residents who frequently used natural ventilation were more likely to report SHS incursion. The results of the study indicate that residents who lived in homes where natural ventilation was provided by opening the front door and those who lived in

homes with frequent natural ventilation were more likely to be exposed to SHS incursion.

In this study, SHS incursion, a dependent variable, was assigned as an ordinal variable in a logistic regression analysis. Previous studies have used dichotomized dependent variables for SHS incursion to examine associated factors.[15,22] When we used SHS incursion as a dichotomized dependent variable (i.e., no = 0 vs. yes = 1), the factors associated with SHS incursion among non-smoking residents living in home with a smoke-free rule in the multivariate logistic regression analysis were household income, children living in the home, time spent at home, and support for the implementation of smoke-free regulations in MUH. Other variables were not significantly associated with SHS incursion. This indicated that using SHS incursion as an ordinal variable might be a more useful approach to examine predictors for SHS incursion in MUH.

The self-reported frequency of SHS incursion differed between smoking and non-smoking residents. In the present study, smokers were less likely to report SHS incursion. This might be explained by a difference in the perception of SHS exposure between smokers and non-smokers. Smokers could be habituated and less likely to be irritated by to the smell of SHS.[16] Similar findings have been reported that residents who were smokers were less likely to report SHS incursion in MUH than were non-smokers.[13,15,16]

The smoking rate of MUH residents in the study population was 25.2%, which was higher than that in the Seoul general population in 2014. Based on

statistical data from the Community Health Survey (CHS), a comprehensive health status survey program in Korea, the smoking rate in the Seoul population (≥19 years) in 2014 was 20.6%.[26] The results of the CHS indicate that the smoking rate increases with age from 19 to 49 years (20.3–25.8%), but then decreases sharply from 50 to 70 years or older (9.0–13.9%). One possible reason for the higher smoking rate in this study could be the low proportion of respondents older than 60 years, which might have led to an overestimation of the smoking rate.

To our knowledge, this is the first study to determine prevalence and predictors of SHS incursion among MUH residents in Korea. The present study included socio-demographic factors as well as built environmental factors to determine predictors on SHS incursion. The findings of the present study could be useful for targeted effort to promote smoke-free regulation in MUH and understanding SHS exposure of residents in homes due to SHS incursion.

This study has a few limitations. We used self-reported SHS incursion experienced by residents within the previous 12 months. The self-report measure might be subject to variations and recall-bias due to a respondent's sensitivity. Because SHS incursion was less likely to be reported by residents who were smokers, we used data from non-smoking residents to identify the factors associated with SHS incursion, which enabled better estimations. Another limitation was that SHS incursion was based on the detection of SHS by smell by MUH residents. Because we measured SHS incursion using a

self-reported questionnaire, we could not confirm or quantify each resident's exposure to SHS due to SHS incursion. Furthermore, self-report of SHS might partially be due to third-hand smoke particularly for the home with smokers in the past. Further study is needed using more specific SHS markers to provide a better understanding of SHS incursion in MUH.

Conclusions

A sample of 2,600 MUH residents in Seoul, Korea, was investigated. The majority of non-smoking respondents who lived in homes with a personal smoke-free rule experienced SHS incursion in their units within the previous 12 months. The high prevalence of SHS incursion suggests that most residents might be at risk from exposure due to SHS incursion. SHS incursion was associated with time spent at home, living with children, support for the implementation of smoke-free regulations in MUH, home size, and the method and frequency of natural ventilation used. Built environmental factors identified in the study could be useful to understand exposure due to SHS incursion at homes in MUH.

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Chapter 4.

Relationships between secondhand smoke incursion and wheeze, rhinitis, and eczema symptoms among children living in multi-unit homes without smokers

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Abstract

Secondhand smoke (SHS) incursion can occur in units of multi-unit housing (MUH). The purpose of this study was to determine the relationship between SHS incursion and allergic symptoms in children living in MUH without smokers. We conducted a cross-sectional study from May to September 2015 in Seoul, Korea. Children were recruited from elementary schools, kindergartens, and daycare centers. In total, 16,676 children between 1 and 13 years of age living in MUH without smokers were included in the analysis. Allergic symptoms during the previous 12 months (current wheeze, rhinitis, and eczema) and home environmental factors, including the frequency of SHS incursion during the previous 12 months, were examined using a questionnaire filled out by the parents or guardians of the children. The prevalence of current allergic symptoms in children was 4.9% for wheeze, 42.0% for rhinitis, and 28.1% for eczema. The prevalence of SHS incursion into the children's homes was 61.6%. In a multivariable logistic analysis adjusted for demographic and home environmental factors, children living in homes with SHS incursion (either no more than once a month or more than once a month) were more likely to have current wheeze, rhinitis, and eczema than were those with no SHS incursion. More than half of the children's homes in MUH without smokers had SHS incursion. SHS incursion into homes was associated with wheeze, rhinitis, and eczema symptoms in children.

Introduction

Secondhand smoke (SHS), also known as passive smoking and environmental tobacco smoke, contains a complex mixture of over 7,000 chemicals, including 69 known carcinogens.[1] It was estimated that more than 41,000 adult and approximately 900 infant deaths were attributed to SHS in the United States in 2006.[2] In the same year, the US Surgeon General concluded that there are no risk-free levels of SHS exposure and that complete elimination of indoor smoking is the only way to protect non-smokers from SHS exposure.[3]

Many countries have implemented smoke-free regulations in enclosed public places to reduce SHS exposure. However, implementation of smoke-free regulations has not been applied to personal living spaces. Children are particularly at risk of SHS exposure at home because they spend a large proportion of their time in the home environment. A previous study reported that nearly half of children (42.5%) worldwide were exposed to SHS in their homes.[4] The two main factors for SHS exposure in children at home were smoking by parents or caregivers and smoking inside the home.[5]

SHS exposure in children has been associated with allergic diseases. Epidemiological studies showed that SHS exposure during pregnancy and early childhood was associated with increased risk of asthma and other respiratory symptoms including wheeze, cough, and respiratory infections.[6]

Data from phase three of the International Study of Asthma and Allergies in Childhood (ISAAC) showed an association between parental smoking and asthma symptoms in children.[7] SHS was also associated with rhinitis[8,9] and eczema symptoms.[7,10,11] A recent systematic review showed that allergic rhinitis (pooled relative risk [RR] = 1.40, 95% confidence interval [CI] = 1.24 to 1.59) and eczema (pooled RR = 1.06, 95% CI = 1.01 to 1.11) were associated with SHS exposure in children and adolescents.[12]

Even in smoke-free homes, children living in multi-unit housing (MUH) could be exposed to SHS from smoke transferred from neighboring units with smokers,[13] known as "SHS incursion." A total of 44.0%–46.2% of American[14] and 28.2% of Danish adults[15] living in non-smoking homes of MUH experienced SHS incursion into their home from another unit. In Seoul, Korea, 74.7% of MUH residents living in smoke-free homes experienced SHS incursion.[16] Blood serum cotinine concentrations measured in American children living in homes in which no one smoked were higher in children living in apartments than in those living in detached houses.[17]

The relationship between SHS exposure from SHS incursion into homes and health effects in children has not been well established. Only one study in Hong Kong has reported that SHS incursion into homes without smokers was significantly associated with respiratory symptoms in adolescents.[18] The present study investigated the relationship between SHS incursion and allergic symptoms in children living in MUH homes without smokers.

Methods

Study design and participants

The results were based on the Atopy Free School in collaboration with the Seoul Atopy Asthma Information Center in Korean Center for Disease Control and Prevention and in Seoul Metropolitan Government. The cross-sectional study was conducted from May to September 2015 in Seoul, Korea. The target population consisted of children who were attending elementary schools, kindergartens, and daycare centers. The total number of eligible participants was 56,336 children in 492 facilities. A newsletter containing a written consent form and questionnaires was delivered by the children to their parents or guardians asking if they wished to voluntarily participate in the study. Among these participants, filled-out questionnaires from 38,150 children in 430 facilities were submitted (response rate of 67.7%). The study was approved by Seoul Medical Center's institutional review board (IRB no. 2015–052).

The study included children living in MUH, including attached housing and apartments. Attached housing was defined as multi-family housing including multi-household housing or row/multiplex housing of fewer than five stories. Apartments were defined as high-rise, multi-family buildings of five or more stories. Children living in detached housing and non-residential buildings were not included because of logistical reasons as well as small sample size.

Among 38,150 children, those who answered questions related to individual factors such as sex, age, parental history of allergic diseases, breastfeeding, duration of residency, housing type, and allergic symptoms and those related to home environmental factors including SHS incursion were included for further analysis (n = 35,831). The children were categorized using the following criteria: those living in (1) attached housing or apartments (n = 33,174); (2) homes without smokers (n = 20,481); and (3) homes for longer than 1 year (n = 16,676). Children living in homes without smokers were identified by the response "no" to the following question: "Are there any current smokers in your home, including yourself?" Data from 16,676 children between 1 and 13 years of age living in homes without smokers in MUH were included in the final analysis.

Measurements

The ISAAC core module was used to assess allergic symptoms in the children.[19] Similar questions have been used in previous studies.[20] Prevalence of wheeze, rhinitis, and eczema during the previous 12 months (current wheeze, rhinitis, and eczema) was determined by the official Korean version of the ISAAC questionnaires. A child with current wheeze was identified by the response "yes" to the question "Has your child had wheezing or whistling in the chest in the last 12 months?" A child with current rhinitis was identified by the response "yes" to the question "Has your child had a problem with sneezing or a runny or blocked nose when he/she did not have a cold or the flu

in the past 12 months?" A child with current eczema was identified by the response "yes" to the question "Has your child had an itchy rash at any time in the last 12 months?"

To examine the prevalence of SHS incursion into the children's homes, respondents were asked how often they could smell tobacco smoke that had entered their living space from somewhere else in or around their building during the previous 12-month period. This question was based on a similar one used in a previous study.[21] Respondents chose one of the following answer: "Never," "Once a month or less," "Twice a month," "Four times a month," "Two to four times a week," or "Every day."

We also asked questions, obtained from previous studies, regarding several associated factors for allergic disease.[11,22,23] We obtained information on the children's demographic factors, including age, sex, parental history of allergic disease (any diagnoses of allergic disease by a doctor in either vs. neither parent), breastfeeding, and duration of residency, and on the children's home environmental factors, including housing type, whether any remodeling or purchase of new furniture had taken place in the previous 12 months in the home, presence of any mold spots inside the home, and traffic density near the home.

Statistical analysis

The frequency of SHS incursion into homes was classified into three categories (none, SHS incursion no more than once a month, and SHS

incursion more than once a month) with similar proportions before statistical analysis. A chi-square test was used to compare the prevalence of current wheeze, rhinitis, or eczema in the children with demographic and home environmental factors, and to compare the frequency of SHS incursion with the housing type. Univariable logistic regression analysis was used to identify individual associations of current wheeze, rhinitis, or eczema in the children with demographic and home environmental factors. The relationships between current wheeze/rhinitis/eczema and the demographic/home variables were presented as odds ratios (ORs) with 95% CIs. Multivariable logistic regression models were used to determine the effects of the frequency of SHS incursion into homes on the prevalence of current wheeze, rhinitis, and eczema after controlling for independent variables identified in the chi-squared test. We included sex and housing type in the multivariable model because they were important potential factors that affect the observed associations. Linear trend tests were performed by including frequency of SHS incursion as continuous variables in the univariable and multivariable models. A p value <0.05 was deemed as significant in all analyses. SAS software (ver. 9.2; SAS Institute, Inc., Cary, NC) was used for all statistical analyses.

Results

The average age of all 16,676 children residing in homes without smokers in MUH was 5.6 ± 2.8 years. The prevalence of current allergic symptoms in the children was 4.9% for wheeze, 42.0% for rhinitis, and 28.1% for current eczema. The prevalence of current wheeze, rhinitis, and eczema significantly differed according to SHS incursion ($p<0.001$) (Table 4-1).

Table 4-1. Prevalence of current wheeze, rhinitis, and eczema in children living in homes without smokers according to demographic and home environmental factors

	Total	Current wheeze		Current rhinitis		Current eczema	
		Yes (%)	*p*-value	Yes (%)	*p*-value	Yes (%)	*p*-value
Demographic factor							
Age (years)							
1-3	4522	388 (8.6)	**<0.001**	1599 (35.4)	**<0.001**	1490 (33.0)	**<0.001**
4-6	6779	281 (4.1)		2965 (43.7)		1976 (29.1)	
7-9	3131	101 (3.2)		1427 (45.6)		754 (24.1)	
10-13	2244	50 (2.2)		1007 (44.9)		459 (20.5)	
Sex							
Female	8179	332 (4.1)	**<0.001**	3079 (37.6)	**<0.001**	2341 (28.6)	0.112
Male	8497	488 (5.7)		3919 (46.1)		2338 (27.5)	
Parental history of allergic disease							
No	8492	289 (3.4)	**<0.001**	2411 (28.4)	**<0.001**	1785 (21.0)	**<0.001**
Yes	8184	531 (6.5)		4587 (56.0)		2894 (35.4)	
Breastfeeding							
No	3424	165 (4.8)	0.765	1448 (42.3)	0.665	848 (24.8)	**<0.001**
Yes	13252	655 (4.9)		5550 (41.9)		3831 (28.9)	
Duration of residency (years)							
<2	3413	198 (5.8)	**0.002**	1429 (41.9)	0.467	965 (28.3)	**0.003**
2-4	6337	336 (5.3)		2626 (41.4)		1862 (29.4)	
≥5	6926	286 (4.1)		2943 (42.5)		1852 (26.7)	
Home environmental factor							
Housing type							
Attached housing	4985	266 (5.3)	0.103	1950 (39.1)	**<0.001**	1399 (28.1)	0.991
Apartment	11691	554 (4.7)		5048 (43.2)		3280 (28.1)	
Remodeling							
No	14734	707 (4.8)	0.051	6136 (41.6)	**0.021**	4094 (27.8)	**0.031**
Yes	1942	113 (5.8)		862 (44.4)		585 (30.1)	
New furniture							
No	12249	595 (4.9)	0.553	5028 (41.0)	**<0.001**	3284 (26.8)	**<0.001**
Yes	4427	225 (5.1)		1970 (44.5)		1395 (31.5)	
Mold spots							
No	8907	361 (4.1)	**<0.001**	3475 (39.0)	**<0.001**	2226 (25.0)	**<0.001**
Yes	7769	459 (5.9)		3523 (45.3)		2453 (31.6)	
Traffic density							
Light	3143	139 (4.4)	0.141	1219 (38.8)	**<0.001**	796 (25.3)	**<0.001**
Moderate	7476	359 (4.8)		3045 (40.7)		2042 (27.3)	
Heavy	6057	322 (5.3)		2734 (45.1)		1841 (30.4)	
SHS incursion (times/month)							
None	6401	253 (4.0)	**<0.001**	2328 (36.4)	**<0.001**	1512 (23.6)	**<0.001**
≤1	4565	229 (5.0)		1979 (43.4)		1336 (29.3)	
>1	5710	338 (5.9)		2691 (47.1)		1831 (32.1)	

Bold estimates are statistically significant at $p<0.05$.

Overall, 61.6% of the parents or guardians of the children living in homes without smokers reported that they had experienced SHS incursion more than once in the past 12 months. SHS incursion was higher among children living in attached housing (62.4%) than among those in apartments (61.3%). The prevalence of SHS incursion differed significantly depending on the housing type ($p<0.001$).

Table 4-2 shows the results of univariable logistic regression analyses of current wheeze, rhinitis, and eczema in children by demographic and home environmental factors. Several demographic and home environmental factors were significantly associated with current wheeze, rhinitis, and eczema. In particular, children living in homes with SHS incursion (either no more than once a month or more than once a month) were more likely to have current wheeze (OR = 1.29, 95% CI = 1.07–1.55; OR = 1.52, 95% CI = 1.29–1.80, p for trend <0.001), rhinitis (OR = 1.34, 95% CI = 1.24–1.45; OR = 1.55, 95% CI = 1.44–1.67, p for trend <0.001), and eczema (OR = 1.34, 95% CI = 1.23–1.46; OR = 1.52, 95% CI = 1.41–1.65, p for trend <0.001) than those with no SHS incursion.

Table 4-2. Univariable logistic analysis of current wheeze, rhinitis, and eczema in children living in homes without smokers

	Current wheeze		Current rhinitis		Current eczema	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Demographic factor						
Age (years) (continuous)	**0.83 (0.80-0.85)**	**<0.001**	**1.05 (1.04-1.06)**	**<0.001**	**0.92 (0.91-0.93)**	**<0.001**
Sex						
Female	1.00		1.00		1.00	
Male	**1.44 (1.24-1.66)**	**<0.001**	**1.42 (1.33-1.51)**	**<0.001**	0.95 (0.88-1.01)	0.107
Parental history of allergic disease						
No	1.00		1.00		1.00	
Yes	**1.95 (1.69-2.26)**	**<0.001**	**3.21 (3.01-3.42)**	**<0.001**	**2.05 (1.92-2.2)**	**<0.001**
Breastfeeding						
No	1.00		1.00		1.00	
Yes	1.02 (0.86-1.22)	0.830	0.99 (0.91-1.06)	0.690	**1.23 (1.13-1.34)**	**<0.001**
Duration of residency (years)						
<2	1.00		1.00		1.00	
2-4	0.91 (0.76-1.09)	0.302	0.98 (0.90-1.07)	0.682	1.06 (0.96-1.16)	0.257
≥5	**0.70 (0.58-0.84)**	**<0.001**	1.03 (0.95-1.12)	0.494	0.93 (0.84-1.01)	0.095
Home environmental factor						
Housing type						
Attached housing	1.00		1.00		1.00	
Apartment	0.88 (0.76-1.02)	0.095	**1.18 (1.11-1.27)**	**0.003**	1.00 (0.93-1.08)	0.968
Remodeling						
No	1.00		1.00		1.00	
Yes	1.21 (0.98-1.48)	0.077	**1.11 (1.01-1.22)**	**0.028**	**1.12 (1.01-1.24)**	**0.039**
New furniture						
No	1.00		1.00		1.00	
Yes	1.06 (0.90-1.24)	0.496	**1.15 (1.08-1.24)**	**<0.001**	**1.26 (1.17-1.36)**	**<0.001**
Mold spots						
No	1.00		1.00		1.00	
Yes	**1.47 (1.28-1.70)**	**<0.001**	**1.29 (1.22-1.38)**	**<0.001**	**1.38 (1.29-1.48)**	**<0.001**
Traffic density						
Light	1.00		1.00		1.00	
Moderate	1.08 (0.89-1.32)	0.441	1.09 (1.00-1.18)	0.060	**1.11 (1.01-1.22)**	**0.038**
Heavy	1.20 (0.98-1.48)	0.076	**1.30 (1.19-1.42)**	**<0.001**	**1.29 (1.17-1.42)**	**<0.001**
SHS incursion (times/month)						
None	1.00		1.00		1.00	
≤1	**1.29 (1.07-1.55)**	**0.007**	**1.34 (1.24-1.45)**	**<0.001**	**1.34 (1.23-1.46)**	**<0.001**
>1	**1.52 (1.29-1.80)**	**<0.001**	**1.55 (1.44-1.67)**	**<0.001**	**1.52 (1.41-1.65)**	**<0.001**
p for trend	***p*<0.001**		***p*<0.001**		***p*<0.001**	

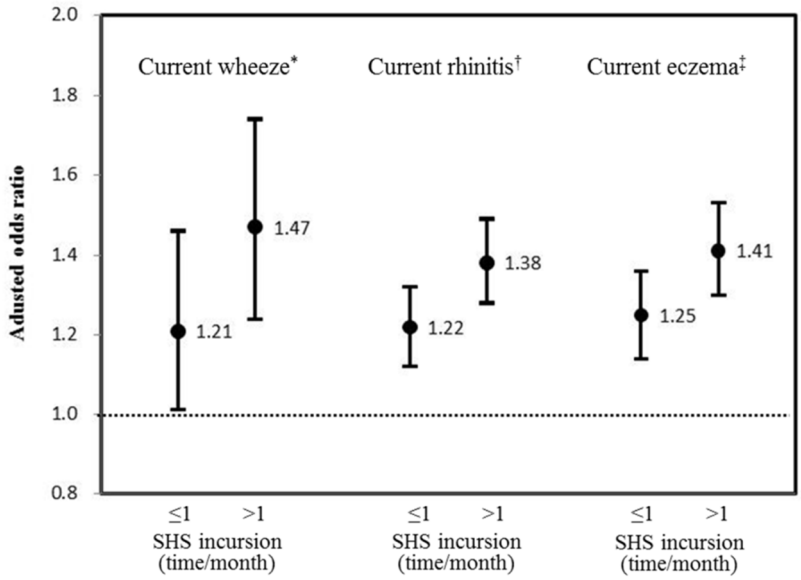
Bold estimates are statistically significant at $p<0.05$.
OR = unadjusted odds ratio; CI = confidence interval; SHS = secondhand smoke.

The demographic factors associated with allergic symptoms in the univariable analysis were consistent with the results from the multivariable analysis (Table 4-3). Furthermore, all of the home environmental factors that showed significant associations in the univariable analysis remained significant in the multivariable analysis, except the remodeling status. In the home environmental factors, children living in homes with SHS incursion (either no more than once a month or more than once a month) were more likely to have current wheeze (adjusted odds ratio, aOR = 1.21, 95% CI = 1.01–1.46; aOR = 1.47, 95% CI = 1.24–1.74, p for trend <0.001), rhinitis (aOR = 1.22, 95% CI = 1.12–1.32; aOR = 1.38, 95% CI = 1.28–1.49, p for trend <0.001), and eczema (aOR = 1.25, 95% CI = 1.14–1.36; aOR = 1.41, 95% CI = 1.30–1.53, p for trend <0.001) than were those with no SHS incursion (Figure 4-1).

Table 4-3. Multivariable logistic analysis of current wheeze, rhinitis, and eczema in children living in homes without smokers

	Current wheeze		Current rhinitis		Current eczema	
	aOR (95% CI)	*p*-value	aOR (95% CI)	*p*-value	aOR (95% CI)	*p*-value
Demographic factor						
Age (years) (continuous)	**0.83 (0.81-0.86)**	**<0.001**	**1.07 (1.05-1.08)**	**<0.001**	**0.93 (0.92-0.94)**	**<0.001**
Sex						
Female	1.00		1.00		1.00	
Male	**1.44 (1.24-1.66)**	**<0.001**	**1.45 (1.36-1.55)**	**<0.001**	0.94 (0.88-1.01)	0.070
Parental history of allergic disease						
No	1.00		1.00		1.00	
Yes	**1.81 (1.56-2.10)**	**<0.001**	**3.17 (2.97-3.38)**	**<0.001**	**1.92 (1.79-2.06)**	**<0.001**
Breastfeeding						
No					1.00	
Yes					**1.14 (1.05-1.25)**	**0.003**
Duration of residency (years)						
<2	1.00				1.00	
2-4	0.87 (0.72-1.04)	0.128			1.03 (0.94-1.14)	0.513
≥5	**0.79 (0.66-0.96)**	**0.017**			0.96 (0.88-1.06)	0.432
Home environmental factor						
Housing type						
Attached housing	1.00		1.00		1.00	
Apartment	1.01 (0.86-1.18)	0.913	**1.17 (1.09-1.26)**	**<0.001**	1.07 (0.99-1.16)	0.098
Remodeling						
No			1.00		1.00	
Yes			1.01 (0.91-1.12)	0.832	1.03 (0.92-1.14)	0.638
New furniture						
No			1.00		1.00	
Yes			**1.10 (1.02-1.18)**	**0.017**	**1.18 (1.09-1.28)**	**<0.001**
Mold spots						
No	1.00		1.00		1.00	
Yes	**1.32 (1.14-1.54)**	**<0.001**	**1.23 (1.15-1.31)**	**<0.001**	**1.27 (1.19-1.37)**	**<0.001**
Traffic density						
Light			1.00		1.00	
Moderate			1.06 (0.97-1.16)	0.240	1.09 (0.99-1.20)	0.081
Heavy			**1.18 (1.07-1.29)**	**<0.001**	**1.19 (1.07-1.31)**	**<0.001**
SHS incursion (times/month)						
None	1.00		1.00		1.00	
≤1	**1.21 (1.01-1.46)**	**0.043**	**1.22 (1.12-1.32)**	**<0.001**	**1.25 (1.14-1.36)**	**<0.001**
>1	**1.47 (1.24-1.74)**	**<0.001**	**1.38 (1.28-1.49)**	**<0.001**	**1.41 (1.30-1.53)**	**<0.001**
p for trend	**<0.001**		**<0.001**		**<0.001**	

Variables identified in the chi-square test ($p<0.05$) and sex and housing type were included in the multivariable analysis. Bold estimates are statistically significant at $p<0.05$.
aOR = adjusted odds ratio; CI = confidence interval; SHS = secondhand smoke.



*Adjusted for age, sex, parental history of allergic disease, duration of residency, housing type, mold spot
†Adjusted for age, sex, parental history of allergic disease, housing type, remodeling, new furniture, mold spot, traffic density
‡Adjusted for age, sex, parental history of allergic disease, breastfeeding, duration of residency, housing type, remodeling, new furniture, mold spot, traffic density

Figure 4-1. Adjusted odds ratios and 95% confidence interval for current wheeze, rhinitis, and eczema in children by frequency of SHS incursion.

Discussion

The prevalence of current wheeze, rhinitis, and eczema among children was slightly different compared with that in a previous study conducted in Seoul in 2012.[20] The prevalence of current wheeze was slightly lower, but that of current rhinitis and eczema was higher, compared with the earlier study in Seoul,[20] which reported prevalence rates of 5.6%, 32.5%, and 17.7% for current wheeze, rhinitis, and eczema, respectively, in children aged 0–13 years. Recent studies of allergic disease based on nationwide populations from 2009 to 2014 in Korea reported a decreasing trend for the prevalence of asthma and eczema but an increasing trend for the prevalence of allergic rhinitis in children aged <10 years.[24] Thus, the higher prevalence of current rhinitis in the present study might be because of this increasing trend in allergic rhinitis among Korean children. In 2016, similar prevalence of current rhinitis (39.7%) of elementary school children in Yongin City had been reported.[25] The higher prevalence of eczema in the present study could be explained by the definition of eczema used in the questionnaires in our study. In the 2012 study in Seoul,[20] for the diagnosis of eczema, patients were asked if they ever had an itchy rash intermittently for at least 6 months and then were further asked if they had experienced an itchy rash at any time during the previous 12 months. In the present study, the children were asked only if they had experienced an itchy rash at any time during the previous 12 months. Thus, the prevalence of

eczema, as defined by the response to this question, in the present study may be higher than that in the 2012 study.

More than half of children's homes had SHS incursion. The prevalence of SHS incursion was associated with housing type and was higher among children living in attached housing than among those in apartments. This indicates that more than half of the children living in MUH homes without smokers were at risk of SHS exposure due to incursion. The findings suggest that smoking in MUH should be restricted to reduce SHS incursion into homes. Because implementation of smoke-free regulations in MUH might be difficult, providing educational material to building owners and managers on the importance of implementing smoke-free policies may be the first step to smoke-free MUH.[26]

The prevalence of SHS incursion into homes in the present study was slightly lower than that in a previous study in Seoul among people living in MUH, and higher than that in a previous study in the United States. A population-based study in Seoul in 2015 showed that the prevalence of SHS incursion into homes of MUH with smoke-free rules was 74.7%, and the smoking rate of the respondents was 25.2%.[16] In the United States in 2010, 44% of residents in MUH living in homes with personal smoke-free rules experienced SHS incursion, and the smoking rate of the respondents was 21.1%.[27] The present study asked whether the children were living with smokers in the home, including the respondents. Thus, we were unable to estimate the smoking rate of the respondents in the study. The higher

prevalence of SHS incursion in the present study might be because of the higher smoking rate in Seoul than in the United States; thus, more smokers may live in MUH homes, leading to the higher prevalence of SHS incursion in Seoul.

In the multivariable analysis, several demographic and home environmental factors were significantly associated with allergic symptoms in the children. In particular, SHS incursion into homes was strongly associated with allergic symptoms in children. The relationship between SHS incursion and current wheeze, rhinitis, and eczema among children was dose-dependent even after adjustment for demographic and home environmental factors. The aORs of SHS incursion experienced more than once a month was greater for current wheeze than that for current rhinitis and eczema in children. This finding indicates that SHS incursion into homes was associated with current wheeze, rhinitis, and eczema among children living in MUH homes without smokers.

SHS incursion into homes was classified into three categories to examine the dose-dependent relationships between allergic symptoms in children and SHS incursion. In previous studies, SHS incursion was used as a dichotomous dependent variable.[21,27] When we evaluated SHS incursion as a dichotomized variable (Yes = 1 vs. No = 0), we found that children living in homes with SHS incursion were more likely to have current wheeze (aOR = 1.35, 95% CI = 1.16 to 1.57), rhinitis (aOR = 1.30, 95% CI = 1.22 to 1.40), and eczema (aOR = 1.34, 95% CI = 1.24 to 1.44) in the multivariable analysis. Our

findings were similar to those obtained using three categories for SHS incursion in the multivariable analysis.

SHS exposure has been reported as a risk factor for allergic disease in children.[7,8,10-12] Although we found significant associations between allergic symptoms in children and SHS incursion, we were unable to confirm that allergic symptoms were associated only with SHS exposure. Frequent SHS incursion might be associated with the exposure of children to SHS, but could also be associated with exposure to residual tobacco pollutants in homes. Infiltrated SHS pollutants can remain on surfaces and dust particles in homes and can be re-emitted and/or re-suspended into the air, which is referred to as third-hand smoke (THS).[28] A previous study showed that indoor surface nicotine concentrations were higher in homes of MUH whose non-smoking residents reported frequent SHS incursion than in those whose non-smoking residents reported no or infrequent SHS incursion.[29] Frequent SHS incursion may be associated with an increased risk of THS exposure, and this could have resulted in the associations with allergic symptoms observed among the children in our study. Furthermore, children may have been exposed to THS, because they lived in homes with smokers in the past. Further research is needed to evaluate the relationships of SHS and THS with allergic disease among children to distinguish the effects of SHS and THS exposure.

This is the first study to examine the relationship between SHS incursion and allergic symptoms among children in MUH. We included several demographic and home environmental factors in the multivariable logistic

model. The study included children of a wide range of ages (1–13 years) among a large population in Seoul.

This study has several limitations. Because the study design was cross-sectional, we could not infer that SHS incursion were causally associated with allergic symptoms in children. It might not be representative of Seoul's population, because recruitment of the study subjects did not involve a random sampling process. Because samples size was large, the null hypothesis might be more likely to be rejected. Although ISAAC core module was used to assess the allergic symptoms in children, misclassification may have occurred because it was developed for children 6 years or older. However, epidemiology studies have used ISAAC core questions to assess the risk of allergic disease in infants.[30-32] Another limitation was that there might be residual confounding by unknown or unmeasured confounders although we included several demographic and home environmental factors. Data on the children, including demographic and home environmental factors were obtained from parents or guardians of the children. This reporting may have produced variation and recall-bias depending on the sensitivity of the respondents. In particular, we measured SHS incursion based on the detection of tobacco smoke odor by the children's parents or guardians. Thus, we did not confirm or quantify the exposure to tobacco pollutants caused by SHS incursion. Although the use of specific biomarkers for SHS exposure (e.g., cotinine) can give objective measurement, it cannot distinguish the sources and locations of exposure. The self-reported SHS incursion by MUH residents

in smoke-free homes has been used as objective measurements in other studies.[16,21] Before and after study using specific markers for SHS exposure might give a better understanding of the associations between SHS incursion and allergic disease among children.

Conclusions

We evaluated 16,676 children living in MUH homes without smokers. More than half of the children's homes had experienced SHS incursion in the previous 12 months. The high prevalence of SHS incursion into homes suggests that most children living in MUH may be at risk of pollutant exposure due to SHS incursion. Several demographic and home environmental factors were associated with current wheeze, rhinitis, and eczema among the children. In particular, the frequency of SHS incursion displayed a dose-dependent relationship with current wheeze, rhinitis, and eczema in children even after adjustment for demographic and home environmental factors. Our findings suggest that SHS incursion was associated with these allergic symptoms in children living in MUH homes without smokers.

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Chapter 5.

Characterization of urinary cotinine of non-smoking residents in smoke-free homes: Korean National Environmental Health Survey (KoNEHS) 2009–2011

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Abstract

The objectives of this study were to determine urinary cotinine concentrations in non-smoking residents of smoke-free homes and to establish the relationship of urinary cotinine with housing type and socio-demographic and secondhand smoke (SHS) exposure factors. We used data from the Korean National Environmental Health Survey I (2009–2011). The study included 814 non-smoking adult residents living in apartments, attached, and detached housing. Residents who lived with smokers were excluded. Urinary cotinine was detected in 88% of the 814 non-smoking residents of smoke-free homes. The urinary cotinine concentrations of residents living in attached [geometric mean (GM): 1.25 ng/ml, 95% confidence interval (CI): 1.00–1.55] and detached housing (GM: 1.37 ng/ml; 95% CI: 1.09–1.73) were significantly higher than those of residents who lived in apartments (GM: 0.81 ng/ml; 95% CI: 0.64–1.01). Urinary cotinine concentrations were significantly higher in residents who were men, those with a household income ≤1000 USD/month, those who were former smokers with >1 year and ≤1 year of not smoking, and those who experienced tobacco smoke odor every day. In the multivariate regression analysis, housing type, former smoking status, and frequency of experiencing tobacco smoke odor were significantly associated with urinary cotinine concentrations (R^2 = 0.15). The associations in the multivariate analysis were similar to creatinine corrected urinary cotinine concentration although creatinine corrected urinary cotinine concentrations

were marginally associated with housing type. The majority of non-smoking residents of smoke-free homes had detectable urinary cotinine. Housing type, former smoking status, and frequency of experiencing tobacco smoke odor were predictors for urinary cotinine concentrations in the study participants.

Introduction

Secondhand smoke (SHS) contains more than 7,000 chemicals, including more than 69 known carcinogens.[1] It is associated with cardiovascular disease, coronary heart disease, asthma, other respiratory symptoms, and lung cancer.[2-5] Epidemiological studies have reported that SHS exposure is causally linked with increasing morbidity and mortality.[4] SHS was estimated to have caused 603,000 premature deaths in 192 countries in 2004, corresponding to about 1% of worldwide mortality.[6]

Based on mounting scientific evidence of the adverse health effects of SHS exposure, many countries have implemented smoke-free regulations in indoor public areas and workplaces, which have led to significant reductions in SHS exposure and positive health effects.[7-9] However, the home environment has remained a significant source of SHS exposure.[4,10]

Many studies that related to SHS exposure at home have focused on non-smoking residents who were living with smokers. Recently, SHS exposure in smoke-free multi-unit housing (MUH) has increased attention. The residents could be exposed to SHS because SHS from MUH with residents who smoke could be transferred to neighboring units.[11,12] In a 2009 survey in the US, 25.8% (79.2 million) lived in MUH, and 62.7 million MUH residents followed smoke-free home rules.[13] Of those residents, SHS incursions were reported in 44.0–46.2% of the residences. In Denmark, 28.2% of MUH residents who lived in homes where no one smoked inside were reported that neighbor

smoke seeped into their homes from other places (e.g., other unit, stairway, etc.).[14] Because people tend to spend a large proportion of their time indoors in their homes, SHS exposure in residences can be a significant contributor to their total exposure. In the US, people spend about 69% of their time in their home.[15] This compares with a daily mean of 59% in Korea.[16]

Limited studies assessed the SHS biomarker levels of non-smokers living in smoke-free homes. Cotinine, a metabolite of nicotine, is a specific and sensitive biomarker of SHS exposure.[17] It can be measured in urine, whole blood, serum, plasma, and saliva, and has an average half-life of 16 hours. One study that assessed the blood serum cotinine levels of US children who lived in homes where no one smoked indoors reported higher serum cotinine concentrations in children who lived in apartments than in those living in detached residences.[18]

In the present study, urinary cotinine concentration data from the Korean National Environmental Health Survey (KoNEHS) I, conducted by the National Institute of Environmental Research and the Ministry of Environment as a national bio-monitoring program, were used. The objectives of this study were to determine the urinary cotinine concentrations of non-smoking residents living in smoke-free homes and to establish the relationship of urinary cotinine concentration with housing type and other socio-demographic and SHS exposure factors.

Methods

Selection of data and study variables

KoNEHS I (2009–2011) used a stratified cluster sampling design that took into consideration geographic and socio-economic factors based on the household surveys of the 2005 Population and Housing Census. Overall, 6,311 individuals who were older than 19 years of age participated in KoNEHS I. The survey collected participants' questionnaires and blood and urine samples.

The 2009 data from KoNEHS (n = 2,101) were excluded because the questionnaires in that year did not ask whether the subjects resided with smokers in their homes. The study included subjects who lived in apartments, attached, and detached house. An apartment was defined as a high-rise multifamily building (≥5 stories) which was often owned by occupants. An attached house was a multi-family house (≤4 stories) with multiple owners. A detached house included single-family and multifamily house (≤3-story) with one owner. Other types of housing, such as non-residential buildings, were excluded due to their small numbers.

Data from the questionnaires were restricted to the following respondents: those who (1) lived in apartments, attached, or detached housing (n = 4,122); (2) had never smoked or were former smokers (n = 3,161); (3) were not living with smokers in their homes (n = 2,026); (4) spent their time mainly at home indoors (n = 933); (5) reported household income (n = 923); and (6) reported

the frequency or duration of tobacco smoke odor in their homes (n = 922). Of these 922 residents, those who were suspected to be smokers (n = 26) based on their urinary cotinine concentrations (≥100 ng/ml)[19] and those whose creatinine concentrations were not estimated were excluded (n = 82). Ultimately, samples from 814 individuals were included for further analysis. This study was approved by the Institutional Review Board of the National Institute of Environmental Research and the Ministry of the Environment (IRB number: EED-354).

Other socio-demographic data, such as sex (men/women), age (19–39 years, 40–59 years, and ≥60 years), education (middle school or less, high school, and college or higher), household income (≤1000 USD/month, 1001–2000 USD/month, 2001–3000 USD/month, and >3000 USD/month), and former smoking status (never smoked, >1 year not smoking, and ≤1 year not smoking) were included. Household income was classified based on 0–25, 26–50, 51–75, and 76–100 percentiles of the 814 subjects. SHS exposure factors, such as frequency of experiencing tobacco smoke odor (none, 1–2 times/week, 3–6 times/week, and every day) and duration of experiencing tobacco smoke odor (none, 1–5 min/day, 6–30 min/day, and ≥31 min/day) were included.

Urinary cotinine

Spot urine samples (80 ml) were collected mid-urination and frozen at –20°C until analysis. All urinary cotinine analyses were carried out at an

analytical laboratory certified by the Korean Ministry of Health and Welfare. For urinary cotinine analysis, internal standard (diphenylamine), 50 μL of 0.1 M sodium hydroxide, and 500 μL of chloroform was added to 1 mL of urine samples. After the solution was centrifuged, sodium sulfate was added to remove waters. The specific method of urinary cotinine analysis has been described in a previous study.[20] The urinary cotinine concentrations were measured using gas chromatograph-mass spectrometry with a Clarus 600T (PerkinElmer, Turku, Finland). The method detection limit (MDL) for urinary cotinine was estimated as 3.14 times the standard deviations of seven replicate measurements of the lowest standard. The MDL for urinary cotinine was 0.27 ng/ml. Urinary cotinine concentrations below the MDL were set to MDL/2 (i.e., 0.14 ng/ml) because data distribution were highly skewed (geometric standard deviation = 3.1 for the 814 subjects).[21] Urinary creatinine was determined with an alkaline picrate kinetic (Jaffe) method using an Adiva 2400 Chemistry System (Siemens Healthcare Diagnostics).

Statistical analysis

Statistical analyses included uncorrected urinary cotinine and creatinine corrected urinary cotinine concentrations. SAS 9.2 (SAS Institute, Inc., Cary, NC, USA) was used for all statistical analyses and calculations. The sample weights were used in all analyses in a stratified cluster sampling design. The proportions of variables by housing type were calculated with SAS PROC SURVEYFREQ. Because the urinary cotinine and creatinine corrected urinary

cotinine concentrations were skewed, the natural log (ln)-transformations of urinary cotinine and creatinine corrected urinary cotinine concentrations were used for all analyses. The geometric mean (GM) and 95% confidence interval (CI) of the urinary cotinine and creatinine corrected urinary cotinine concentrations by variable were calculated with SAS PROC SURVEYMEANS. SAS PROC SURVEYREG was used to perform a univariate and multivariate linear regression to assess the associations between urinary cotinine and creatinine corrected urinary cotinine concentrations and variables. Variables with p-values <0.1 in the univariate analysis were included in the multivariate regression analysis.

Although education and sex were not associated with urinary cotinine and creatinine corrected urinary cotinine concentrations, respectively, in the univariate analysis, we included these variables in the multivariate analysis because these variables might be important potential predictors for urinary cotinine and creatinine corrected urinary cotinine levels. Duration of tobacco smoke odor was not included in the multivariate analysis because this variable was correlated with frequency of tobacco smoke odor. A p-value of 0.05 was considered significant in all analyses.

Results

The characteristics of the study population by housing type are shown in Table 5-1. Non-smoking residents of detached housing represented slightly less than half (43.8%) of the overall study population, and residents of apartments and attached housing comprised 36.9% and 19.2% of the study subjects, respectively. The majority of the participants was women (64.0%), 40–59 years of age (34.1%), had middle school or less education (40.5%), and had household incomes of 1001–2000 USD/month (28.1%). Former smokers with >1 year of not smoking and ≤1 year of not smoking comprised 19.5% and 4.2% of the subjects, respectively. The percentages of residents who experienced tobacco smoke odor ≥1 time/week or ≥1 min/day in apartments, attached, and detached housing, were 20.7%, 16.1%, and 14.6%, respectively.

Table 5-1. Socio-demographic and SHS exposure factors among non-smoking residents of smoke-free homes by housing type*

	Apartment (%)	Attached housing (%)	Detached housing (%)	Total (%)
Sex				
Men	83 (28.1)	49 (36.8)	183 (42.4)	315 (36.0)
Women	204 (71.9)	82 (63.2)	213 (57.6)	499 (64.0)
Age (year)				
19–39	79 (34.3)	32 (27.0)	33 (12.7)	144 (23.5)
40–59	135 (41.7)	50 (37.1)	159 (38.0)	344 (39.2)
≥60	73 (24.0)	49 (35.8)	204 (49.3)	326 (37.3)
Education				
Middle school or less	69 (21.2)	54 (33.7)	254 (59.7)	377 (40.5)
High school	117 (43.8)	41 (34.9)	100 (25.5)	258 (34.1)
College or higher	101 (35.0)	36 (31.4)	42 (14.8)	179 (25.4)
Household income (USD/month)				
≤1000	27 (7.3)	36 (25.9)	157 (38.7)	220 (24.6)
1001–2000	66 (24.6)	40 (29.7)	113 (30.4)	219 (28.1)
2001–3000	91 (31.3)	29 (21.4)	75 (17.5)	195 (23.4)
>3000	103 (36.7)	26 (23.0)	51 (13.4)	180 (23.8)
Former smoker				
Never smoked	241 (83.4)	95 (71.8)	275 (72.4)	611 (76.3)
>1 year not smoking	43 (15.0)	26 (19.7)	102 (23.2)	171 (19.5)
≤1 year not smoking	3 (1.6)	10 (8.6)	19 (4.4)	32 (4.2)
Frequency of tobacco smoke odor (times/week)				
None	231 (79.3)	106 (83.9)	333 (85.4)	670 (82.8)
1–2	35 (10.5)	14 (8.6)	30 (7.2)	79 (8.7)
3–6	11 (6.2)	5 (4.2)	14 (3.6)	30 (4.7)
Every day	10 (4.0)	6 (3.3)	19 (3.8)	35 (3.8)
Duration of tobacco smoke odor (min/day)				
None	231 (79.3)	106 (83.9)	333 (85.4)	670 (82.8)
1–5	28 (9.9)	10 (5.8)	18 (3.7)	56 (6.4)
6–30	12 (4.6)	12 (9.5)	22 (5.5)	46 (6.0)
≥31	16 (6.2)	3 (0.8)	23 (5.4)	42 (4.8)

*All estimated data are based on weighted analyses.

Urinary cotinine was detected in 88% of the 814 residents. The GM of the urinary cotinine concentrations was 1.11 ng/ml (95% CI: 0.96–1.28) and that of the creatinine corrected urinary cotinine concentrations was 1.31 ng/mg creatinine (Cr) (95% CI: 1.13–1.51). In the detected urinary cotinine samples, cumulative frequency distributions of urinary cotinine and creatinine corrected urinary cotinine concentrations are shown in Figure 5-1.

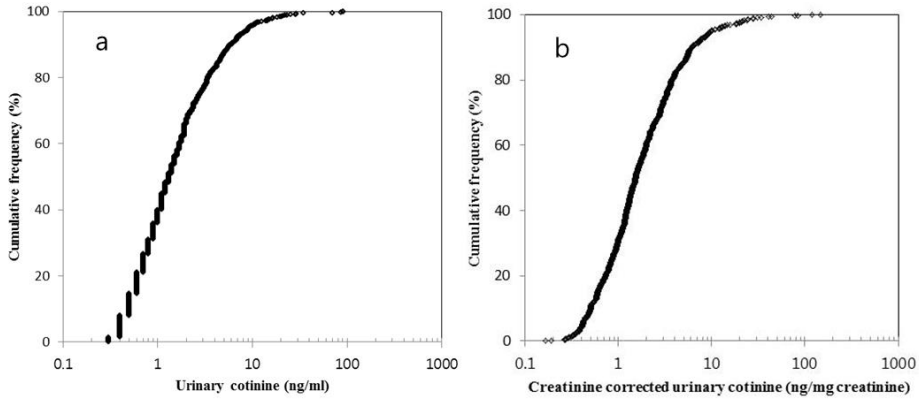

Figure 5-1. Cumulative frequency of (a) urinary cotinine and (b) creatinine corrected urinary cotinine concentrations of non-smoking residents of smoke-free homes. This graph included only the detected urinary samples (n = 719) from 814 samples.

In the univariate analysis, several variables were significantly associated with urinary cotinine and creatinine corrected urinary cotinine concentrations (Table 5-2). In the urinary cotinine, the concentrations of residents who lived in attached (GM: 1.25 ng/ml; 95% CI: 1.00–1.55) and detached (GM: 1.37 ng/ml; 95% CI: 1.09–1.73) housing were significantly higher than those of residents who lived in apartments (GM: 0.81 ng/ml; 95% CI: 0.64–1.01). The GM of the urinary cotinine concentrations of residents were 1.47 ng/ml (95% CI: 1.19–1.82) for men and 0.94 ng/ml (95% CI: 0.80–1.12) for women; these values were significantly different. The urinary cotinine concentrations of residents who had a household income of ≤1000 USD/month (GM: 1.41 ng/ml; 95% CI: 1.09–1.82) were significantly higher than those of residents with a household income of 2001–3000 USD/month (GM: 0.94 ng/ml; 95% CI: 0.73–1.21). The urinary cotinine concentrations of former smokers with >1 year of not smoking (GM: 1.65 ng/ml; 95% CI: 1.29–2.11) and ≤1 year of not smoking (GM: 3.64 ng/ml; 95% CI: 2.06–6.41) were significantly higher than the concentrations of residents who had never been smokers (GM: 0.94 ng/ml; 95% CI: 0.80–1.10). The urinary cotinine concentrations of residents who experienced tobacco smoke odor every day (GM: 2.26 ng/ml; 95% CI: 1.29–3.97) were significantly higher than those of residents who never experienced tobacco smoke odor (GM: 1.06 ng/ml; 95% CI: 0.91–1.24). However, urinary cotinine concentrations were not significantly associated with age, education, or duration of experiencing tobacco smoke odor. GMs and 95% CI of the urinary cotinine concentrations by significant variables are

shown in Figure 5-2. Similar associations were observed in creatinine corrected urinary cotinine concentrations except for sex and education.

Table 5-2. Univariate analysis results for natural log-transformed urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents of smoke-free homes*

	Urinary cotinine (ng/ml)				Creatinine corrected urinary cotinine (ng/mg Cr)			
	GM (95% CI)	β	SE	*p*-value	GM (95% CI)	β	SE	*p*-value
Type of housing								
Apartment	0.81 (0.64-1.01)	Reference			1.02 (0.81-1.30)	Reference		
Attached housing	1.25 (1.00-1.55)	0.43	0.16	**0.008**	1.37 (1.10-1.70)	0.29	0.16	0.077
Detached housing	1.37 (1.09-1.73)	0.53	0.16	**0.002**	1.57 (1.26-1.95)	0.43	0.16	**0.009**
Sex								
Men	1.47 (1.19-1.82)	Reference			1.43 (1.16-1.76)	Reference		
Women	0.94 (0.80-1.12)	-0.44	0.12	**<0.001**	1.24 (1.05-1.47)	-0.14	0.12	0.233
Age (year)								
19–39	0.89 (0.68-1.17)	Reference			1.07 (0.81-1.40)	Reference		
40–59	1.19 (1.00-1.42)	0.29	0.16	0.070	1.36 (1.16-1.59)	0.24	0.16	0.123
≥60	1.17 (0.93-1.47)	0.27	0.18	0.131	1.42 (1.13-1.79)	0.29	0.18	0.099
Education								
Middle school or less	1.27 (1.02-1.59)	Reference			1.54 (1.24-1.92)	Reference		
High school	1.04 (0.84-1.29)	-0.20	0.14	0.172	1.28 (1.05-1.55)	-0.19	0.13	0.160
College or higher	0.96 (0.73-1.26)	-0.28	0.17	0.105	1.04 (0.82-1.32)	-0.39	0.16	**0.012**
Household income (USD/month)								
≤1000	1.41 (1.09-1.82)	Reference			1.71 (1.33-2.18)	Reference		
1001–2000	1.07 (0.85-1.36)	-0.27	0.15	0.060	1.18 (0.93-1.49)	-0.37	0.15	**0.015**
2001–3000	0.94 (0.73-1.21)	-0.40	0.19	**0.032**	1.15 (0.90-1.47)	-0.39	0.18	**0.033**
>3000	1.05 (0.81-1.35)	-0.29	0.19	0.116	1.27 (0.99-1.62)	-0.30	0.18	0.097

*All estimated data are based on weighted analyses.
SE = standard error.
$p<0.05$ are in bold.

Table 5-2. Univariate analysis results for natural log-transformed urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents of smoke-free homes (continued)*

	Urinary cotinine (ng/ml)				Creatinine corrected urinary cotinine (ng/mg Cr)			
	GM (95% CI)	β	SE	*p*-value	GM (95% CI)	β	SE	*p*-value
Former smoker								
Never smoked	0.94 (0.80-1.10)	Reference			1.17 (1.00-1.37)	Reference		
>1 year not smoking	1.65 (1.29-2.11)	0.57	0.13	**<0.001**	1.64 (1.30-2.07)	0.34	0.13	**0.008**
≤1 year not smoking	3.64 (2.06-6.41)	1.36	0.30	**<0.001**	3.39 (1.77-6.49)	1.06	0.34	**0.002**
Frequency of tobacco smoke odor (times/week)								
None	1.06 (0.91-1.24)	Reference			1.27 (1.08-1.48)	Reference		
1–2	1.11 (0.83-1.48)	0.04	0.15	0.775	1.28 (0.93-1.76)	0.01	0.17	0.947
3–6	1.32 (0.71-2.44)	0.21	0.31	0.488	1.36 (0.61-3.03)	0.07	0.41	0.857
Every day	2.26 (1.29-3.97)	0.76	0.30	**0.014**	2.52 (1.44-4.41)	0.69	0.31	**0.026**
Duration of tobacco smoke odor (min/day)								
None	1.06 (0.91-1.24)	Reference			1.27 (1.08-1.48)	Reference		
1–5	1.54 (0.97-2.43)	0.37	0.25	0.145	2.06 (1.27-3.33)	0.49	0.26	0.062
6–30	1.25 (0.91-1.73)	0.17	0.17	0.334	1.22 (0.86-1.72)	-0.04	0.18	0.829
≥31	1.28 (0.71-2.30)	0.18	0.29	0.525	1.31 (0.65-2.62)	0.03	0.36	0.926

*All estimated data are based on weighted analyses.
SE: standard error.
$p<0.05$ are in bold.

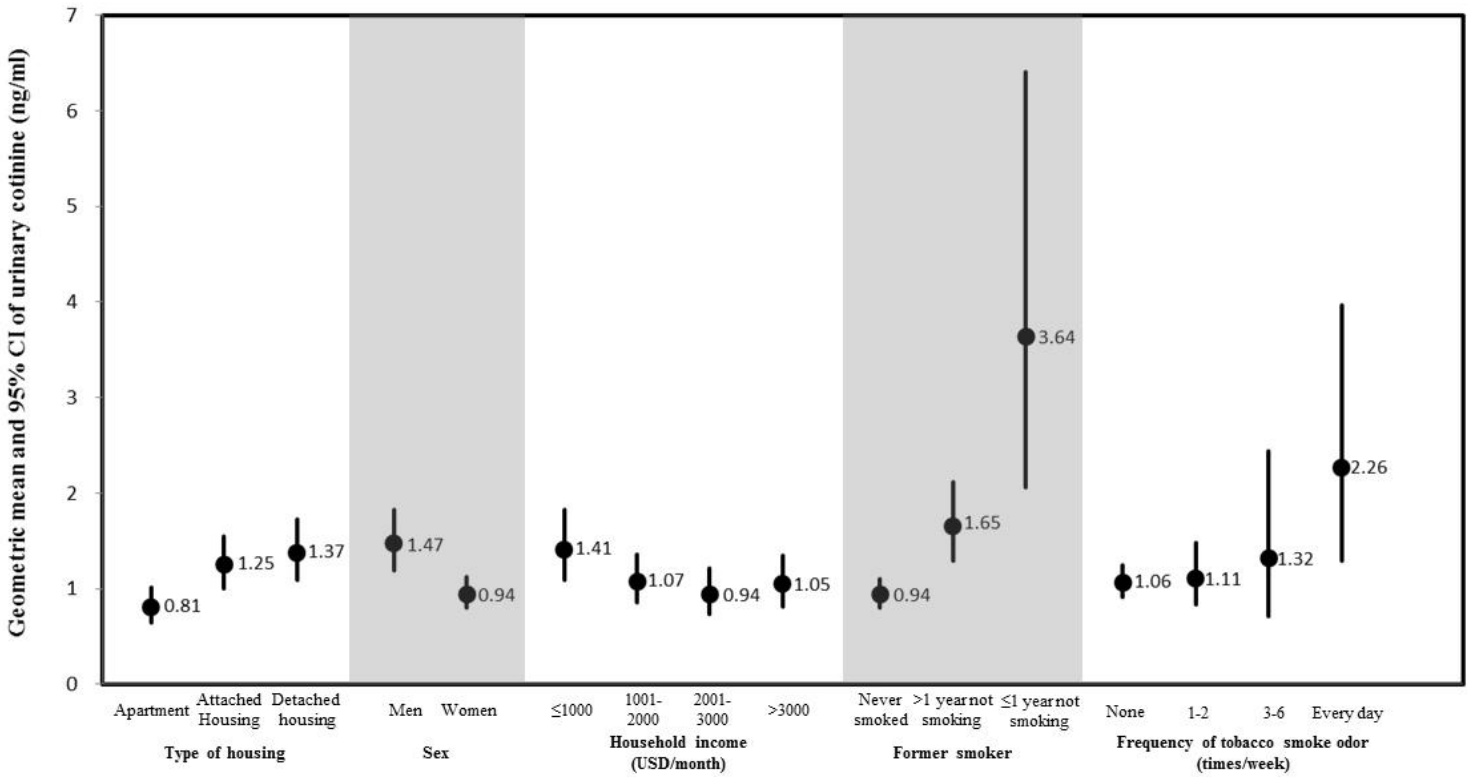

Figure 5-2. Geometric means and 95% confidence interval of urinary cotinine concentrations type of housing, sex, household income, former smoking status, and frequency of tobacco smoke odor.

Several variables were significantly associated with urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents in the multivariate analysis (Table 5-3). The urinary cotinine concentrations of residents who lived in attached (p = 0.062) and detached housing (p = 0.017) were higher than the concentrations of those living in apartments. The urinary cotinine concentrations of former smokers with >1 year of not smoking (p = 0.002) and ≤1 year of not smoking (p<0.001) were significantly higher than those of subjects who had never smoked. The urinary cotinine concentrations of residents who experienced tobacco smoke odor every day were significantly higher (p = 0.022) than the concentrations of those who never experienced tobacco smoke odor. However, urinary cotinine concentrations were not associated with sex, age, education, and household income. Similar results were observed in creatinine corrected urinary cotinine concentrations although relationships between creatinine corrected urinary cotinine concentrations and housing type were marginally associated.

Table 5-3. Multivariate analysis for natural log-transformed urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents of smoke-free homes*

	Urinary cotinine			Creatinine corrected urinary cotinine		
	β	SE	*p*-value	*β*	SE	*p*-value
Type of housing						
Apartment	Reference			Reference		
Attached housing	0.29	0.16	0.062	0.19	0.16	0.250
Detached housing	0.38	0.16	**0.017**	0.31	0.16	0.062
Sex						
Men	Reference			Reference		
Women	-0.02	0.15	0.898	0.26	0.16	0.101
Age (year)						
19–39	Reference			Reference		
40–59	0.12	0.15	0.432	0.04	0.16	0.817
≥60	-0.11	0.18	0.568	-0.07	0.18	0.711
Education						
Middle school or less	Reference			Reference		
High school	-0.11	0.15	0.466	-0.10	0.13	0.463
College or higher	-0.09	0.17	0.616	-0.24	0.17	0.158
Household income (USD/month)						
≤1000	Reference			Reference		
1001–2000	-0.20	0.16	0.208	-0.26	0.16	0.106
2001–3000	-0.25	0.20	0.205	-0.23	0.19	0.225
>3000	-0.17	0.20	0.391	-0.13	0.19	0.484
Former smoker						
Never smoked	Reference			Reference		
>1 year not smoking	0.50	0.16	**0.002**	0.47	0.17	**0.007**
≤1 year not smoking	1.22	0.31	**<0.001**	1.13	0.36	**0.002**
Frequency of tobacco smoke odor (times/week)						
None	Reference			Reference		
1–2	-0.06	0.14	0.687	-0.01	0.15	0.941
3–6	0.09	0.28	0.738	0.11	0.36	0.759
Every day	0.63	0.27	**0.022**	0.64	0.26	**0.013**

*All estimated data are based on weighted analyses.
SE = standard error.
$p<0.05$ are in bold.
R-squared values from the multivariate regression model were 0.15 for urinary cotinine and 0.09 for creatinine corrected urinary cotinine after adjusting for all factors listed in the table.

Discussion

The urinary cotinine concentrations and creatinine corrected urinary cotinine concentrations of the non-smoking residents living in smoke-free homes were associated with housing type in the univariate analysis. The urinary cotinine concentrations of non-smoking subjects living in attached and detached housing were 1.5- and 1.7-fold higher, respectively, than the concentrations of those living in apartments. The creatinine corrected urinary cotinine concentrations of non-smoking subjects living in detached housing were 1.5-fold higher than the concentrations of those living in apartments. While the urinary cotinine concentrations of non-smoking residents were significantly associated with housing type, the creatinine corrected urinary cotinine concentrations of residents were marginally associated with housing type in the multivariate analysis. Overall, urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents living in detached housing were higher than these concentrations of those living in apartment. This indicated that non-smoking residents living in detached housing were more likely to be exposed to tobacco smoke pollutants than were those living in apartments.

Although housing type was significantly associated with urinary cotinine concentrations, housing type was marginally associated with creatinine corrected urinary cotinine concentrations. This is might be because urinary creatinine concentrations were associated with housing type. The creatinine

concentrations of residents who lived in attached and detached housing were higher than those of residents who lived in apartments. In the present study, the residents who were men were more likely to live in detached housing than those who were women. The creatinine concentrations of residents who were men were significantly higher than those of residents who were women, similar to previous studies.[22,23] Therefore, resident who were men were more likely to live in detached housing, which might lead to higher urinary creatinine concentrations in detached housing. Because creatinine corrected urinary cotinine concentrations were estimated by urinary cotinine concentrations to urinary creatinine concentrations ratios, the findings of relationships between housing type and urinary cotinine and creatinine corrected urinary cotinine concentrations might be yielded differently.

Our findings on housing type differ from those of a study of serum cotinine concentrations in non-smoking children in the US living in homes where no one smoked inside the home.[18] The serum cotinine concentrations of non-smoking children who lived in apartments (0.075 ng/ml) were significantly higher than the concentrations of those living in attached (0.053 ng/ml) and detached housing (0.031 ng/ml). These differences are likely due to the different rates of residents who resided with smokers by housing type between Korea and America. The US children who resided with smokers inside the home were more likely to live in apartments than in detached housing.[18] In Korea, data from KoNEHS I showed that the non-smokers residing with smokers were less likely to live in apartments (49.2%) than in

attached (59.1%) and detached housing (53.1%).

The housing type reflected the socio-economic status of the residents. A previous study reported that socio-economic status was associated with SHS exposure among non-smoking residents.[10] Plasma cotinine concentrations among non-smoking adults were higher in those who were living in more socio-economically disadvantaged circumstances, suggesting that non-smoking residents of higher socio-economic level have lower SHS exposure. This trend was similar to our findings of lower urinary cotinine and creatinine corrected urinary cotinine concentrations in non-smoking residents living in apartments. In Korea, apartments are high-rise multifamily buildings that are similar to high-rise condominium buildings in the USA. Residents living in apartments tended to be of a higher socio-economic level than those living in other housing types.

Possible sources of SHS in smoke-free homes include SHS incursion from neighboring units and from outside the building. Evidence of SHS incursion from smoking units was reported in 2 of 14 smoke-free units and 6 of 8 hallways inside 11 MUH buildings.[11] Temporal profiles of concentrations of particulate matter smaller than 2.5 μm in diameter ($PM_{2.5}$), an airborne marker for SHS exposure,[24] demonstrated that $PM_{2.5}$ concentrations in hallways increased instantly when the front door of a smoking unit was opened, and later the $PM_{2.5}$ concentrations in nearby smoke-free units increased. Outdoor tobacco smoke near building entrances has been shown to drift into indoor spaces.[25] Median $PM_{2.5}$ concentrations were 17.2 $\mu g/m^3$ in outdoor main

entrances where smoking occurred and 18.2 μg/m^3 in halls adjacent to outdoor areas. However, the median $PM_{2.5}$ concentrations in outdoor and indoor areas with no presence of specific $PM_{2.5}$ sources (controls) were 13.0 μg/m^3 and 10.4 μg/m^3, respectively.

Non-smoking residents in smoke-free homes may also be exposed to residual tobacco smoke pollution. The SHS pollutant can remain on dust and surfaces in the indoor environment and be re-suspended and/or re-emitted into the air or react with other compounds to produce secondary pollutants, which are referred to as third-hand smoke (THS).[26] Dust and surface nicotine concentrations in homes previously occupied by smokers decreased after non-smoking residents moved in, but were still seven- to eight-fold higher than in previously smoke-free homes.[27] Average urinary cotinine concentrations of the youngest residents from former smokers' homes (n = 5; 0.61 ng/ml) were higher than the concentrations of those from former smoke-free homes (n = 13; 0.13 ng/ml, p = 0.12).

Former smoking was significantly associated with higher urinary cotinine concentrations and creatinine corrected urinary cotinine in the multivariate analysis. Former smokers of >1 year of not smoking and ≤1 year of not smoking had 1.8- and 3.9-fold higher urinary cotinine concentrations, respectively, and 1.4- and 2.9-fold higher creatinine corrected urinary cotinine concentrations, respectively, than did subjects who had never smoked. The higher urinary cotinine concentrations in former smokers may be because former smokers who quit smoking recently might be smoked occasional

cigarettes. The findings of this study are similar to those of a study that measured saliva cotinine concentrations in 97 non-smoking adults.[28] In that study, although the saliva cotinine concentrations of former smokers and adults who had never smoked were not significantly different, the median concentrations of salivary cotinine were slightly higher in former smokers (2.8 ng/ml) than in subjects who had never smoked (2.4 ng/ml; $p = 0.87$). The median cotinine concentrations of former smokers of ≤6 month of not smoking (9.5 ng/ml) were significantly higher than the concentrations of former smokers of >6 month of not smoking (2.2 ng/ml; $p = 0.02$).

Daily experience of tobacco smoke odor in the homes was significantly associated with increased urinary cotinine and creatinine corrected urinary cotinine concentrations in the multivariate analysis. Although some categories of frequency of tobacco smoke odor were not statistically significant, urinary cotinine and creatinine corrected urinary cotinine concentrations increased with increasing frequency of experiencing tobacco smoke odor.

The relationship between urinary cotinine levels and frequency of experiencing tobacco smoke odor differed by housing type. The urinary cotinine concentrations of non-smoking residents in detached housing were highest, followed by those of non-smoking residents in attached housing and apartments. However, non-smoking residents who experienced tobacco smoke odor ≥1 time/week were lowest in detached housing followed by attached housing and apartments. The self-reported perception of SHS exposure may have differed according to the social tolerance level. When smoking

prevalence rates are high, SHS exposure may be less likely to be perceived. Data from KoNEHS I showed that rates of non-smokers residing with smokers were more likely to live in attached and detached housing than in apartment. These findings indicate that non-smoking residents of detached housing may have higher tolerance regarding SHS exposure than the residents of other housing types.

Urinary cotinine concentrations were usually adjusted to urinary creatinine concentrations to correct urinary dilutions among spot urine samples. However, previous study showed that uncorrected urinary cotinine concentrations showed higher correlations with the serum cotinine concentrations than did creatinine corrected urinary cotinine concentrations.[29,30] In the present study, uncorrected and creatinine corrected urinary cotinine concentrations were used. Similar findings in multivariate analysis on urinary cotinine and creatinine corrected urinary cotinine concentrations were observed.

This study has several limitations. Because detached houses in the present study included single-family and multifamily houses (e.g., single room studio), urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents in smoke-free home were not separately assessed these types of houses. The KoNEHS I applied the housing types because it was legal classification. In future, it is needed to precise classification of housing types to assess urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents who lived in smoke-free single

family and multifamily houses.

Although we selected non-smoking residents not living with smokers in their homes, they might have been exposed to SHS in their homes from non-resident smokers. Although this study selected residents who spent the majority of their time at home indoors, they were possibly exposed to SHS outside their homes, such as outdoors, in transportation, and at social venues. This could not be assessed in this study, since the questionnaire did not ask whether regular guests or visitors smoked in the homes or residents were exposed to SHS exposure outside homes. The study could not account for nicotine containing products, including e-cigarettes and nicotine replacement therapies (e.g., nicotine patch) among former smokers. These unmeasured factors may have contributed to the urinary cotinine concentrations.

Previous studies have quantified SHS exposure in smoke-free homes using environmental markers (e.g., particulate matter, volatile organic compounds, etc.).[11,12] The present study measured urinary cotinine and creatinine corrected urinary cotinine concentrations in non-smoking residents living in smoke-free homes to determine SHS exposure and associated factors. The findings are based on nationwide survey data. Although the frequency of tobacco smoke odor in smoke-free homes was positively associated with the urinary cotinine and creatinine corrected urinary cotinine concentrations of non-smoking residents, the sources of tobacco smoke pollutants were not identified. Further study is needed to identify the sources of SHS in smoke-free homes in Korea.

Conclusions

Data from KoNEHS I were used to determine the urinary cotinine concentrations of 814 non-smoking residents of smoke-free homes. High detection rate of urinary cotinine in the non-smoking residents suggested that the most non-smoking residents in Korea might be exposed to SHS. The urinary cotinine concentration was associated with housing type, former smoking status, and frequency of experiencing tobacco smoke odor in the home. The associations were similar to creatinine corrected urinary cotinine concentration although creatinine corrected urinary cotinine concentrations were marginally associated with housing type. The findings suggested that residents in smoke-free homes might be exposed to tobacco smoke pollutants from SHS incursion from neighboring units and from outside the building, as well as from THS in the homes.

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Chapter 6.

Summary and conclusions

The Korean government implemented smoke-free regulations in indoor public places. However, the effects of SHS exposure reduction due to these regulations have not been evaluated. Although smoke-free regulations have been implemented in indoor public places, such regulations might not be appropriate for residences. Even in smoke-free homes, residents of MUH could be exposed to SHS due to incursion from neighboring units. The main objectives of this study were to determine the effects of smoke-free regulations in indoor public places and to establish scientific evidence of the risks of exposure and the health effects due to SHS incursion.

The first study included 121 hospitality venues and 95 non-smoking staff members in 71 venues to evaluate the effects of the Korean smoke-free regulations in venues with a net indoor area ≥150 m^2 in terms of air quality, biomarker levels, and health effects. Smoke-free regulations significantly reduced the levels of indoor fine particles (i.e., $PM_{2.5}$) and urinary total 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol in staff in bars ≥150 m^2. Staff at venues ≥150 m^2 experienced significantly improved sensory health. However, no improvement was observed in the measured data or health effects at venues <150 m^2, where indoor smoke-free regulations were not applied. The findings of this study support the expansion of smoke-free regulations to all indoor public places, including restaurants and bars <150 m^2. Further study is needed to determine the effect of ongoing regulations, their longer-term health effects, and the possible social determinants of change over time.

In the second study, 1,784 non-smoking residents of MUH who lived in homes with a personal smoke-free rule were surveyed to determine the prevalence of SHS incursion and to establish the relationship between SHS incursion and socio-demographic and built environmental factors. In total, 74.7% of these non-smoking residents had experienced SHS incursion ≥1 times during the previous 12 months. SHS incursion was associated with time spent at home, living with children, support for the implementation of smoke-free regulations in MUH, home size, and the method and frequency of natural ventilation used. The high prevalence of SHS incursion implied that most residents could be at risk of SHS exposure due to incursion.

In the third study, 16,676 children living in homes without smokers in MUHs were recruited from elementary schools, kindergartens, and daycare centers to determine the relationships between SHS incursion and allergic symptoms in children. The prevalence of current allergic symptoms in children was 4.9% for wheeze, 42.0% for rhinitis, and 28.1% for eczema. The prevalence of SHS incursion into children's homes was 61.6%. Thus, more than half the children surveyed in this study were at risk of SHS exposure due to incursion into smoke-free homes in MUH. In a multivariate logistic regression analysis adjusted for demographic and home environmental factors, children living in homes with SHS incursion (either no more than once a month or more than once a month) were found to be more likely to have current wheeze, rhinitis, and eczema than were those with no SHS incursion. Dose-dependent relationships between SHS incursion and current wheeze,

rhinitis, and eczema indicated that SHS incursion into homes without smokers in MUH was associated with these allergic symptoms in children.

In the final study, data from Korean National Environmental Health Survey I were used to determine urinary cotinine concentrations by housing type and socio-demographic and SHS exposure factors. Among 814 non-smoking residents in smoke-free homes, urinary cotinine was detected in 88%. The findings indicate that most non-smoking residents in smoke-free homes were at risk of SHS exposure in their homes. Urinary cotinine concentrations were associated with housing type, former smoking status, and frequency of experiencing tobacco smoke odor. The relationship between urinary cotinine concentrations and the frequency of experiencing tobacco smoke odor indicated that residents in smoke-free homes might be exposed to tobacco smoke pollutants from SHS incursion and from third-hand smoke in the home.

There are no risk-free levels of SHS exposure, and only the elimination of smoking in indoor spaces can protect non-smokers from SHS exposure. The present study demonstrated that the implementation of smoke-free regulations in restaurants and bars $\geq$150 m^2 has been effective in reducing levels of $PM_{2.5}$ and urinary total NNAL concentrations in non-smoking staff in bars of this size and in decreasing sensory symptoms in non-smoking staff in venues of this size. Although smoke-free regulations have been implemented in restaurants and bars, it may be difficult to apply such regulations to residences. The present studies showed that, even in smoke-free homes, most non-smoking residents were at risk of exposure and the health effects due to SHS

incursion. As of February 10, 2018, the Korean government allowed management authorities (e.g., apartment building managers) to recommend or educate smokers to stop smoking in MUH units. However, there are many limitations to regulating private spaces. Furthermore, many MUH structures, such as small apartments or attached houses, did not have management authorities. The final study showed that the urinary cotinine concentrations of non-smoking residents who lived in attached and detached housing were higher than those of apartment-dwelling residents. Detached housing in Korea includes single-family and multifamily houses (e.g., single room studios). Because urinary cotinine concentrations were associated with housing type, housing type-specific approaches to the creation of smoke-free environments (e.g., the development of smoke-free policies, education, or promotional materials) are needed to protect non-smoking residents from exposure due to SHS incursion into their homes; this may be especially important for attached and detached housing. The findings of these studies might be used to support the expansion of smoke-free policies to indoor public places and to inform the public about the need for smoke-free environments in all indoor living spaces.

국문초록

한국의 생활환경에서 간접흡연: 노출, 건강 위해 및 실내 금연정책의 효과

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간접흡연(secondhand smoke, SHS)의 노출은 성인과 아이들의 건강에 유해한 영향을 줄 수 있다. 간접흡연의 노출에는 안전한 수준은 없다. 이에 많은 국가들은 간접흡연의 노출을 줄이기 위해서 실내 공공장소에서 금연정책(smoke-free regulation)을 시행하였다. 우리나라의 경우 식당이나 술집과 같은 접객업소(hospitality venue)는 실내 면적에 따라 점진적으로 확대하는 금연정책을 시행하였다. 2013 년 7 월 1 일부터 실내 면적이 ≥150 m^2 인 식당과 술집에서 금연정책을 시행되었다.

하지만 아직 우리나라의 식당과 술집에서 금연정책의 시행으로 인한 간접흡연의 감소효과에 대해서 평가한 바가 없었다. 금연정책의 효과에 대한 과학적인 근거는 다른 실내 공공장소에서 금연정책의 확대를 지원하는데 중요하다.

실내 공공장소에서 금연정책을 시행하였지만 주거지에서는 금연정책의 시행이 제한적일 수 있다. 주거 환경은 간접흡연의 주요 노출 장소로 알려져 있다. 특히 공동주택(multi-unit housing)의 경우 집안에 흡연자가 거주하지 않는 비흡연 가구(smoke-free home)일지라도 이웃집의 흡연으로 인한 담배연기가 집안으로 흘러 들어오는 "간접흡연 침투(SHS incursion)"가 발생하여 거주자가 간접흡연에 노출될 수 있다. 최근 공동주택 내 비흡연 가구에서 간접흡연의 노출에 대한 우려가 증가하고 있지만, 간접흡연 침투로 인한 거주자의 노출과 건강영향에 대한 과학적인 정보는 제한적이다. 본 연구의 주 목적은 실내 공공장소에서의 금연정책에 대한 효과를 평가하고 주거장소 내 비흡연 가구에서 간접흡연 침투로 인한 노출과 건강영향의 위험에 대한 과학적인 근거를 마련하는 것이다.

첫 번째 연구는 실내 금연정책 시행에 따른 식당과 술집의 실내 공기 질, 종사자들의 생체시료 수준과 건강영향을 평가하였다. 실내 금연정책 시행 전과 후 146 개 접객업소에서 직경이 2.5 μm 이하인 입자인 $PM_{2.5}$ 와 77 개 접객업소 내 101 명의 비흡연

종사자를 대상으로 요 중 cotinine 과 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol(NNAL)을 반복 측정하였다. 또한, 종사자의 자기보고를 통한 호흡기 및 감각(즉, 눈/코/목 자극) 증상을 두 기간 동안 평가하였다. 식당과 술집 총 121 개소와 71 개소 내 95 명의 비흡연 종사자가 최종 분석에 포함되었다. 실내 $PM_{2.5}$ 의 기하평균 농도는 ≥150 m^2 술집에서 금연정책 시행 전 93.2 $\mu g/m^3$(기하표준편차 = 2.2)이었고 금연정책 시행 후 55.3 $\mu g/m^3$(기하표준편차 = 2.2)로 통계적으로 유의하게 감소하였다($p<0.05$). 비록 종사자의 요 중 cotinine 농도는 변화가 없었지만 요 중 총 NNAL 의 기하평균 농도는 ≥150 m^2 술집에서 금연정책 시행 전 12.1 pg/mg creatinine(Cr)(기하표준편차 = 2.0)이었고 금연정책 시행 후 7.3 pg/mg Cr(기하표준편차 = 1.7)로 유의하게 감소하였다($p<0.05$). ≥150 m^2 식당의 경우 금연정책 시행 전 흡연자 수가 적게 관측되었기 때문에 $PM_{2.5}$ 와 종사자의 생체시료 수준은 차이가 나지 않았다. 종사자의 건강영향의 경우 실내 면적이 ≥150 m^2 업소에서 감각 증상만이 금연정책 시행 전 52%에서 금연정책 시행 후 40%로 유의하게 감소하였다($p<0.05$). 실내 금연정책의 시행은 ≥150 m^2 술집의 $PM_{2.5}$ 와 종사자의 총 NNAL 농도를 감소시켰고 ≥150 m^2 업소에 근무하는 종사자들의 감각 증상을 개선시켰다. 하지만

금연정책이 시행되지 않은 <150 m^2 업소에서는 실내공기질과 종사자의 건강이 개선되지 않았다.

두 번째 연구는 공동주택에서 간접흡연 침투 비율을 평가하고 간접흡연 침투와 사회인구학적 요인 및 건축환경 요인과의 관계를 평가하였다. 웹 패널을 기반하여 서울시 인구를 반영한 공동주택에 거주하는 성인 2,600 명의 표본을 수집하였다. 거주자는 사회인구학적 요인, 흡연상태, 간접흡연 침투 빈도, 그리고 건축환경 요인의 문항에 응답하였다. 거주자가 집안에서 흡연하는 사람이 없다고 응답했을 때 가구 내 금연규칙(personal smoke-free rule)이 있다고 정의하였다. 총 2,600 명 중 가구 내 금연규칙이 있다고 응답한 비흡연 거주자를 대상으로 추가분석을 하였다(n = 1,784). 비흡연 거주자 중 74.7%가 지난 12 개월 동안 간접흡연 침투를 경험하였다. 다중 순서형 로지스틱 회귀분석에서 거주자 중 집에 더 오랜 시간 머물거나, 아이와 거주하거나, 공동주택 내 금연정책의 시행을 지지하거나, 가구 면적이 작거나, 출입문을 열어 환기 또는 출입문과 창문을 함께 열어 환기하는 가구에 거주하거나, 자연 환기 빈도가 높은 가구에 거주할수록 간접흡연 침투의 위험이 높았다. 이 연구 결과 공동주택 내 비흡연 가구에 거주하는 비흡연 거주자 대부분이 집안에서 간접흡연 침투를 경험하였다.

세 번째 연구는 공동주택 내 흡연자와 함께 거주하지 않는 집에 거주하는 아이들의 알레르기 증상과 간접흡연 침투와의 관계를

평가하였다. 2015 년 서울에서 실시한 단면연구로서 초등학교, 유치원, 어린이집에서 아이들을 모집하였다. 아이들 중 공동주택에 거주하고, 집에 흡연자가 거주하지 않으며, 연령이 만 1-13 세인 총 16,676 명이 분석에 포함되었다. 아이들의 부모 또는 보호자가 기입한 설문지를 이용하여 아이들의 지난 12 개월 동안 알레르기 증상(현재 천식, 알레르기비염, 아토피피부염)과 지난 12 개월 동안 간접흡연 침투 빈도를 포함한 주거환경 요인을 평가하였다. 아이들의 현재 알레르기 증상 유병률은 천식의 경우 4.9%, 알레르기비염의 경우 42.0%, 아토피피부염의 경우 28.1%이었다. 아이들이 거주하는 집의 간접흡연 침투 비율은 61.6%이었다. 인구학적 요인과 주거환경 요인을 보정한 다변수 로지스틱 회귀분석에서 간접흡연 침투가 있는 가구(월 1 회 이하 또는 월 1 회 초과)에 거주하는 아이들이 간접흡연 침투가 없는 가구에 거주하는 아이들보다 현재 천식, 알레르기비염, 아토피피부염 증상의 위험이 높았다. 이 연구의 결과 가구 내 간접흡연 침투는 흡연자가 없는 집에 거주하는 아이들의 천식, 알레르기비염, 아토피피부염 증상과 관련이 있었다.

마지막 연구는 비흡연 가구 내 비흡연 거주자들의 요 중 cotinine 농도를 평가하고 요 중 cotinine 과 주거형태, 그리고 사회인구학적 특성 및 간접흡연 노출 요인과의 관계를 평가하였다. 이 연구는 환경보건기초조사(Korean National Environmental

Health Survey, KoNEHS) 1 기 자료를 이용하였다. 아파트, 다세대주택, 단독주택에서 흡연자와 거주하지 않으며, 집에서 주로 생활하는 비흡연 거주자 814 명의 자료를 이용하였다. 우리나라의 단독주택은 단독주택과 다중/다가구 주택(예, 원룸)을 포함한다. 비흡연 거주자 총 814 명 중 88%에서 요 중 cotinine 이 검출되었다. 요 중 cotinine 농도는 다세대주택(기하평균: 1.25 ng/ml; 95% 신뢰구간: 1.00−1.55)과 단독주택 거주자(기하평균: 1.37 ng/ml; 95% 신뢰구간: 1.09−1.73)가 아파트 거주자(기하평균: 0.81 ng/ml; 95% 신뢰구간: 0.64−1.01)보다 통계적으로 유의하게 높았다. 요 중 cotinine 농도는 응답자 중 남성이거나, 가구소득이 월 100 만원 이하이거나, 금연한지 1 년 이하 또는 1 년 초과했거나, 담배 냄새를 매일 경험하는 경우 통계적으로 유의하게 높았다. 다중 회귀분석에서 요 중 cotinine 농도는 주거형태, 과거 흡연 상태, 그리고 담배 냄새를 경험한 빈도와 관련이 있었다(R^2=0.15). 다중 회귀분석에서 크레아티닌으로 보정한 요 중 cotinine 농도는 보정되지 않은 요 중 cotinine 농도와 비슷한 결과를 보였다. 이 연구 결과 비흡연 가구에 거주하는 대부분 비흡연 거주자의 요에서 cotinine 이 검출되었다. 주거 형태, 과거 흡연 상태, 그리고 담배 냄새를 맡은 빈도는 요 중 cotinine 농도의 예측요인이었다. 요 중 cotinine 농도와 담배 냄새를 경험한 빈도와의 관련성은 비흡연 가구에 거주하는 비흡연 거주자들이 집안에서 간접흡연 침투 또는

3 차 흡연(third-hand smoke)으로 인한 담배 오염물질에 노출되었을 가능성을 보여준다.

본 연구에서는 실내 공공장소에서 금연정책의 효과를 평가하고 주거장소 내 비흡연 가구에서 간접흡연 침투로 인한 노출과 건강영향의 위험에 대한 과학적인 근거를 마련하고자 하였다. 식당과 술집의 금연정책의 시행은 ≥150 m^2 술집의 실내공기질의 개선과 종사자들의 간접흡연 노출의 감소뿐만 아니라 ≥150 m^2 업소에 근무하는 종사자들의 감각 증상을 개선시켰다. 하지만 금연정책 시행이 적용되지 않은 <150 m^2 업소에서는 실내공기질과 종사자의 건강이 개선되지 않았다. 이 연구의 결과는 <150 m^2 업소를 포함한 모든 실내 공공장소에서 금연정책의 확대를 지원하는데 활용될 수 있을 것이다. 추가연구를 통하여 규제의 준수 여부, 종사자의 장기적 건강영향, 그리고 시간에 따라 변하는 사회적 결정요인에 대한 평가가 필요하다.

금연정책이 적용되지 않은 주거장소에서는 공동주택 내 자발적 금연규칙이 있는 집에 거주하는 비흡연 거주자들의 대부분(74.7%)이 간접흡연 침투를 경험하였다. 공동주택의 비흡연 가구 내 간접흡연 침투는 일부 사회인구학적 요인 및 건축환경 요인과 관련이 있었다. 공동주택 내 흡연자와 거주하지 않는 아이들을 대상으로 한 연구에서는 인구학적 요인과 주거환경 요인을 보정한 후에도 간접흡연 침투 빈도는 아이들의 현재 천식,

알레르기비염, 아토피피부염 증상과 용량 반응(dose-dependent)적 관계가 관측되었다. 환경보건기초조사 1 기 자료에 의하면 집에 흡연자와 거주하지 않는 비흡연 거주자들의 대부분 요(88%)에서 cotinine 이 검출되었다. 주거 형태, 과거 흡연 상태, 그리고 담배 냄새를 맡은 빈도는 요 중 cotinine 농도의 예측요인이었다.

주거지를 대상으로 수행한 연구에서는 집에 흡연자가 없더라도 비흡연 거주자의 대부분이 간접흡연 침투로 인한 노출과 건강의 위험성이 있었다. 국민건강영향조사 1 기 자료에서 비흡연 가구 내 비흡연 거주자의 요 중 cotinine 농도는 주거 형태와 관련이 있었다. 특히 다세대주택과 단독주택 거주자의 요 중 cotinine 농도가 높았기 때문에 금연 환경을 조성하기 위하여 주거 형태에 따른 접근법(예, 금연정책, 교육, 또는 홍보자료의 개발)이 필요할 것이다. 이 연구들의 결과는 실내 공공장소에서 금연정책의 확대를 지원하고 실내 모든 생활 공간에서 금연 환경에 대한 대중의 인식변화를 가져오는데 활용될 수 있을 것이다.

주요어: 간접흡연, 공동주택, 금연정책, 비흡연 가구, 알레르기, 접객업소, 침투

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