

Research Article

Association Between Exposure to Indoor Air Pollutants and Allergic Rhinitis Status Using the Korean National Health and Nutrition Examination Survey Data

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Background: Recent studies have shown that indoor air pollutants (IAPs) affect air quality and cause respiratory issues, particularly allergic rhinitis (AR). This study investigated the association between exposure to IAPs and AR risks and symptoms. **Methods:** We gathered data from 1864 individuals who responded to the 2022 Korea National Health and Nutrition Examination Survey, after excluding 116 individuals with missing data from the indoor air quality survey. Participants were categorized into AR and non-AR groups based on the history of AR. IAPs included particulate matter, carbon dioxide, formaldehyde, volatile organic compounds, benzene, toluene, ethylbenzene, xylene, styrene, and environmental tobacco smoke. Logistic regression determined the risk of AR according to a single IAP exposure level. Negative binomial regression was used to investigate the association between AR-related symptoms and number of highly exposed IAPs.

Results: The prevalence of AR was 6%. Exposure to several IAPs was higher in the AR group. The association between individual IAP and AR risk was not significant after adjusting for covariates. The odds ratios for the diagnosis, duration, and severity of AR increased with increasing numbers of exposure to high levels of IAPs.

Conclusions: There was no significant association with AR for each pollutant as a single exposure. However, the risk of AR-related symptoms significantly increased with the number of exposure to high levels of IAPs. These findings provide valuable insights for interventions to improve indoor air quality to mitigate the prevalence and severity of AR.

Keywords: allergic rhinitis; allergy; indoor air pollutants; KNHANES; VOC

1. Introduction

People spend approximately 90% of their time indoors, whether at home, school, work, or in other public places; therefore, the indoor environment is an important factor of human health [1]. The World Health Organization estimates that indoor air pollutants (IAPs) account for an estimated 4.3 million deaths annually [2].

Environmental air pollutants cause a variety of effects on human health and are strongly associated with respiratory diseases [3], and patients with allergic diseases, among other respiratory conditions, are particularly vulnerable to environmental air pollutants [4]. IAP components, such as volatile organic compounds (VOCs), carbon monoxide (CO), carbon dioxide (CO₂), and particulate matter (PM), are known to contribute to the increased risk of developing allergic diseases [5, 6].

Allergic rhinitis (AR) is a common chronic allergic disease triggered by Type 1 hypersensitivity reactions related with exposure to environmental air pollutants. The prevalence of AR is steadily increasing, affecting approximately 500 million individuals worldwide [7]. Several studies have shown that exposure to environmental respiratory hazards, such as air pollution and smoking, increases the risk of AR [8–10]. Additionally, it has been reported that exposure to these environmental respiratory hazards may be a major factor in the genetic and immune influences associated with the pathogenesis of AR [11, 12].

Research on the association between exposure to environmental respiratory hazards and AR is promising; however, previous studies have focused on estimating the risk of developing AR from exposure to outdoor environmental respiratory hazards or a single IAP [6, 13]. However, recent evidence suggests that exposure to multiple pollutants may exacerbate AR symptoms, increase subjective discomfort, and even impair quality of life beyond the effects of individual pollutants, highlighting the need to evaluate multipollutant exposures [14–16].

Therefore, we aimed to investigate the relationship of the exposure to several IAPs and incidence of AR, as well as to estimate the risk and severity of AR using data which was nationally representative of Koreans.

2. Materials and Methods

2.1. Study Participants. The data for this study were obtained from the 8th KNHANES, a nationally representative survey conducted by the Korea Disease Control and Prevention Agency (KDCA) from 2019 to 2021 [17]. A total of 22,559 individuals from 14,400 households participated in the 8th KNHANES, using a sampling framework based on the most recent Korean census data. In practice, KNHANES has collected data through a household screening survey, a health interview, a health examination, and a nutrition survey. The 8th KNHANES, however, was the first to include an indoor air quality survey, introduced in response to growing interest in the health effects of various IAPs. Indoor air quality was assessed between July 2020 and August 2021 in nearly 1,200 households through both on-site air quality measurements and a structured questionnaire. The present analysis included data from 1,864 individuals, out of 1,980 respondents to the indoor air quality survey, after excluding those with missing data or who declined to provide information.

All procedures were performed in accordance with the Declaration of Helsinki, and the KNHANES was approved by the Institutional Review Board (IRB) of the KDCA (IRB No. 2018-01-032C-A and 2018-01-03-5C-A). KNHANES data was publicly available, and written informed consent was obtained from all KNHANES participants.

2.2. IAPs. IAPs evaluated in this study included PM_{2.5} (micrograms per cubic meter), CO₂ (parts per million), formaldehyde (HCHO) (micrograms per cubic meter), total volatile organic compounds (TVOCs, micrograms per cubic meter), and specific VOCs including benzene, toluene, ethyl-

benzene, xylene, and styrene (all in micrograms per cubic meter). All pollutants were measured in accordance with the standard analytical methods specified by the Ministry of Environment of Korea. PM_{2.5} concentrations were determined using the gravimetric method with 24-h integrated sampling, employing a KMS-4100 sampler equipped with an inertial impactor, filter holder, suction pump, and flow rate controller. After sampling, filters were weighed using an electronic analytical balance (Sartorius). CO₂ levels were monitored continuously for 1 h using the nondispersive infrared method with a portable CO₂ analyzer (IQ-610 Xtra, Graywolf). HCHO was sampled using 2,4-dinitrophenylhydrazine (DNPH)-coated cartridges (MP-Σ100H, SIBATA) with an ozone (O₃) scrubber, followed by HPLC analysis (LC2030-Plus, Shimadzu). Air was collected twice for 30 min at a flow rate of 0.5 L/min. The method detection limit (MDL) for HCHO was 0.0004 µg/m³. TVOCs were collected using Tenax TA sorbent tubes at 0.1 L/min and analyzed via thermal desorption–gas chromatography with mass spectrometry and flame ionization detection (TD-20/GC-MS/FID, Shimadzu). Sampling was performed twice for 30 min using the KMS-100 sampler. MDLs for individual VOCs were as follows: benzene (0.000489 µg/m³), toluene (0.000809 µg/m³), ethylbenzene (0.000950 µg/m³), xylene (0.001736 µg/m³), and styrene (0.001083 µg/m³).

Environmental tobacco smoke (ETS) was included as an additional IAP. While not measured through direct air sampling, ETS exposure was assessed through self-reported responses from the KNHANES questionnaire. Participants who responded “Yes” to the question “Is there any person who smokes routinely inside the house?” were classified as exposed to ETS [18].

2.3. AR. A medical history of AR was based on the participants’ responses to the questionnaire. Participants who answered “Yes” to “Have you ever been diagnosed with allergic rhinitis by objective testing, such as blood tests for allergens or allergy skin tests?” were allocated to the AR group [19] and were given four additional questions related to the symptoms of AR. Seasonality was evaluated by the question “In what season do the AR symptoms occur?” with possible responses of “Only in certain seasons” (seasonal) and “It happens all year round” (nonseasonal). Weekly duration of AR symptoms was evaluated by the question “How many days per week do you experience the AR symptoms?” with possible responses of “Less than 4 days” (nonfrequent) and “More than 4 days” (frequent). Length of symptoms was evaluated by the question “How long have these symptoms lasted?” with possible responses of “Less than 1 month” (short) and “More than 1 month” (long). Symptom severity was evaluated using the question “Do the AR symptoms interfere with your study/work/sleep?”

2.4. Covariates. The general patient characteristics selected as confounding variables were age, sex, household income level, and residence. AR-related factors included obesity (based on body mass index) and lifestyle (alcohol consumption, smoking, and exercise). Daily average outdoor air

pollution (PM_{2.5}, micrograms per cubic meter) was also selected as a confounding variable.

2.5. Statistical Analysis. Chi-squared or Student's *t*-tests were used to determine the distribution of demographic and IAPs according to AR status. Logistic regression analysis was conducted to evaluate the risk of AR based on the level of exposure to IAPs, which was categorized as a binary variable based on the median. All analyses were adjusted for age, sex, household income, residence, obesity, alcohol consumption, smoking status, exercise level, and outdoor air pollution. Concentrations of each of the 10 IAPs were categorized into high and low groups based on their median values, and the number of high exposures per individual was counted, resulting in a dependent variable ranging from 0 to 10. To analyze the count data, we applied and compared three regression models: Poisson regression, zero-inflated negative binomial (ZINB) regression, and negative binomial regression. The best-fitting model was determined based on the Akaike information criterion (AIC) and Bayesian information criterion (BIC), both of which balance model complexity and goodness of fit. The negative binomial regression model demonstrated the lowest AIC and BIC values, indicating that it provided the most appropriate fit for the data. Furthermore, residual diagnostics, including an analysis of Pearson residuals, confirmed that the negative binomial model effectively captured the patterns in the data without systematic bias. As a result, negative binomial regression was selected as the primary analytical method. This model was used to estimate the odds ratios (ORs) and 95% confidence intervals (95% CIs) for the associations between the number of high-level IAP exposures and AR diagnosis, as well as related symptoms such as seasonality, frequency, duration, and severity. All analyses were adjusted for potential confounders, including age, sex, household income, residence, obesity, alcohol consumption, smoking status, exercise level, and outdoor air pollution levels. Given the multiple comparisons performed, we implemented false discovery rate (FDR) correction to control Type I errors. Adjusted *p*-values were calculated and reported to enhance result reliability. Statistical analyses were conducted using SAS (Version 9.4; SAS Institute Inc., Cary, NC, United States), and a *p*-value of < 0.05 was considered statistically significant.

3. Results

Table 1 shows the general characteristics and IAP exposures, based on AR status. Of the 1,864 participants, 112 (6.0%) were diagnosed with AR. The average age of the AR group was 44.8 years, which was lower than that of the non-AR group (54.2 years). The prevalence of AR in women and men was 6.5% and 5.4%, respectively, with no significant differences based on household income or residence. The prevalence of AR was significantly higher among individuals with normal weight (7.4%) compared to those with obesity (5.1%), reflecting a statistically robust difference (*p* = 0.037). In contrast, no significant differences in AR prevalence were observed with respect to alcohol consumption, smok-

ing status, or exercise level. Among the IAPs, exposure to TVOCs (*p* = 0.0176) and toluene (*p* = 0.0027) was significantly higher in the AR group compared to the non-AR group. ETS and outdoor air pollution did not significantly affect the prevalence of AR.

Table 2 shows the results of the logistic regression analyses for AR risk according to the level of IAP exposure. The risk of being diagnosed with AR was modestly elevated for exposures to benzene, ETS, and ethylbenzene, with ORs of 1.66 (95% CI: 0.90–3.08), 1.57 (95% CI: 0.34–7.28), and 1.39 (95% CI: 0.90–2.15), respectively. Similarly, exposure to TVOCs and PM_{2.5} was associated with ORs of 1.25 (95% CI: 0.81–1.94) and 1.19 (95% CI: 0.80–1.79). The ORs for toluene and styrene were 1.18 (95% CI: 0.78–1.80) and 1.02 (95% CI: 0.60–1.72), respectively. For exposures to CO₂ and HCHO, the ORs were below 1, at 0.96 (95% CI: 0.65–1.44) and 0.90 (95% CI: 0.61–1.33), respectively. Importantly, none of these associations reached statistical significance. After applying FDR correction to account for multiple comparisons, none of the individual IAP demonstrated a statistically significant association with AR risk.

Table 3 presents the risk of AR and AR-associated symptoms according to the number of high exposures to IAPs. The risk of being diagnosed with AR increased significantly with a higher number of high-level IAP exposures (OR: 1.46, 95% CI: 1.20–1.69). Although the risk of seasonal and frequent AR symptoms showed a slight increase, these associations did not reach statistical significance. In contrast, the risk of long-duration AR symptoms (OR: 1.27, 95% CI: 1.02–1.56) and severe symptoms (OR: 1.14, 95% CI: 1.04–1.41) increased significantly with greater IAP exposure. After applying FDR correction to account for multiple comparisons, the association between cumulative exposure to high-level IAP and AR diagnosis remained statistically significant. However, the previously observed association with long-duration symptoms became nonsignificant after FDR adjustment. The associations for seasonal, frequent, and severe symptoms remained nonsignificant as originally observed.

4. Discussion

We evaluated the association between AR and IAPs and found no significant association between individual IAPs and AR risk. However, multiple high-level IAP exposures significantly increased both the risk and severity of AR-related symptoms—even though our study participants comprised only adults with AR, who are generally less sensitive to air pollution than children or adolescents [15]. In particular, the risk of longer duration and increased severity of AR symptoms was significantly associated with exposure to an increasing number of IAPs, suggesting that even relatively low-level yet cumulative exposures may contribute to adverse health effects.

Most previous studies have focused on the association between exposure to each IAP and the risk of AR [20]. However, recent some studies suggest that multipollutant exposures may more accurately reflect real-world conditions. For example, a study showed that coexposure to PM_{2.5}, O₃, sulfur dioxide (SO₂), and nitrogen dioxide

TABLE 1: Characteristics of study participants according to allergic rhinitis status.

	Allergy rhinitis, <i>n</i> (%) or mean (standard error)		<i>p</i> -value
	No	Yes	
Total participants	1752 (94.0)	112 (6.0)	
Age	54.2 (0.40)	44.8 (1.4)	< 0.0001
Sex			0.2839
Men	795 (94.6)	45 (5.4)	
Women	957 (93.5)	67 (6.5)	
Household income level			0.1271
Low	348 (96.9)	11 (3.1)	
Middle low	436 (93.2)	32 (6.8)	
Middle high	436 (92.6)	35 (7.4)	
High	532 (94.0)	34 (6.0)	
Residence			0.4441
Urban	1436 (93.8)	95 (6.2)	
Rural	316 (94.9)	17 (5.1)	
Obesity			0.0370
No	701 (92.6)	56 (7.4)	
Yes	1051 (94.9)	56 (5.1)	
Excessive alcohol consumption			0.2245
No	1578 (93.8)	105 (6.2)	
Yes	170 (96.0)	7 (4.0)	
Current smoking			0.3360
No	1487 (93.8)	99 (6.2)	
Yes	261 (95.3)	13 (4.7)	
Regular exercise			0.7502
No	1037 (93.8)	68 (6.2)	
Yes	715 (94.2)	44 (5.8)	
Indoor air pollution			
PM _{2.5} (μg/m ³)	16.3 (0.4)	15.2 (1.3)	0.5352
CO ₂ (ppm)	767.0 (7.9)	829.2 (41.2)	0.1408
HCHO (μg/m ³)	27.4 (0.5)	28.8 (1.9)	0.5027
TVOC (μg/m ³)	233.8 (11.8)	360.3 (101.8)	0.0176
Benzene (μg/m ³)	5.3 (0.7)	5.8 (1.6)	0.7841
Toluene (μg/m ³)	22.7 (1.3)	40.9 (12.0)	0.0027
Ethylbenzene (μg/m ³)	5.8 (0.8)	5.0 (0.7)	0.4535
Xylene (μg/m ³)	10.3 (1.1)	10.3 (2.2)	0.9870
Styrene (μg/m ³)	3.8 (0.3)	4.6 (0.9)	0.4472
Environmental tobacco smoke			0.6326
No	1703 (94.0)	108 (6.0)	
Yes	49 (92.4)	4 (7.6)	
Outdoor air pollution			
PM _{2.5} (μg/m ³)	17.3 (0.3)	17.4 (1.2)	0.9223

Abbreviations: CO₂, carbon dioxide; HCHO, formaldehyde; PM, particulate matter; TVOC, total volatile organic compounds.

(NO₂) significantly increased clinic visits for allergic conditions, although it was combined to a double set of pollutants [21]. Another study observed a general upward trend in AR symptoms and quality of life impairment as simultaneous exposure to multiple pollutants increased, although the small sample size constrained statistical power [14]. Additionally, some research that employs a combined indoor

exposure index reported significant associations with various respiratory diseases, yet its link to AR specifically was not statistically significant [22]. These findings highlight the variability in outcomes and underscore the need for broader, integrated evaluations of multiple IAPs. In this perspective, our study evaluated a broader range of 10 IAPs across multifaceted indoor settings while assessing multiple AR

TABLE 2: Logistic regression analysis of indoor air pollution and allergy rhinitis.

Air pollution (median)	Odds ratio (95% confidence intervals) of allergy rhinitis	<i>p</i> -value	FDR adjusted <i>p</i> -value
PM _{2.5} (12.3)	1.08 (0.70–1.67)	0.7286	0.9108
CO ₂ (665.0)	0.96 (0.65–1.44)	0.8406	0.9340
HCHO (21.2)	0.90 (0.61–1.33)	0.5962	0.8517
TVOC (109.2)	1.19 (0.80–1.79)	0.3972	0.8517
Benzene (2.8)	1.66 (0.90–3.08)	0.1063	0.6912
Toluene (10.2)	1.18 (0.78–1.80)	0.4378	0.8517
Ethylbenzene (2.3)	1.39 (0.90–2.15)	0.1382	0.6912
Xylene (4.0)	1.25 (0.81–1.94)	0.3166	0.8517
Styrene (1.6)	1.02 (0.60–1.72)	0.9412	0.9412
Environmental tobacco smoke (n/a)	1.57 (0.34–7.28)	0.5639	0.8517

Note: All results were adjusted for age, sex, household income level, residence, obesity, alcohol consumption, smoking, exercise level, and outdoor air pollution referred to under the median of each indoor air pollution level.

Abbreviations: CO₂, carbon dioxide; HCHO, formaldehyde; PM, particulate matter; TVOC, total volatile organic compounds.

TABLE 3: Odds ratio and 95% confidence intervals for allergic rhinitis-associated symptoms according to the number of high exposures to indoor air pollution from negative binomial regression analyses.

Allergic rhinitis associated symptoms	Odds ratio (95% confidence intervals)	<i>p</i> -value	FDR adjusted <i>p</i> -value
Diagnosed allergic rhinitis			
No	Reference	< 0.0001	< 0.0001
Yes	1.46 (1.20–1.69)		
Seasonal symptom of allergic rhinitis			
Nonseasonal	Reference	0.0523	0.0871
Seasonal	1.16 (0.82–1.86)		
Frequency of allergic rhinitis symptom			
Nonfrequent	Reference	0.0523	0.0871
Frequent	1.31 (0.91–1.57)		
Duration of allergic rhinitis symptom			
Short	Reference	0.0274	0.0686
Long	1.27 (1.02–1.56)		
Severe symptom of allergic rhinitis			
No	Reference	0.0915	0.1144
Yes	1.14 (1.04–1.41)		

Note: All results were adjusted for age, sex, household income level, residence, obesity, alcohol consumption, smoking, exercise level, and outdoor air pollution referred to under the median of each indoor air pollution level.

outcomes, including diagnosis, symptom persistence, and severity. This underscores the potential importance of multi-pollutant interactions in the pathophysiology and clinical management of AR.

Some studies have explained the possible mechanisms by which IAPs affect AR. Exposure to fine PM and its constituents increased the risk of AR by triggering oxidative stress. Research confirms that exogenous particles can induce alterations in malondialdehyde and superoxide dismutase that are classic biomarkers that reflect oxidative stress level and antioxidant enzyme activity [14]. Oxidative stress in nasal epithelial cells exacerbates allergic airway disease and increases organ reactivity [23]. The epithelial barrier hypothesis suggests that a high number of IAP exposures

damage nasal epithelial cells, compromising the mucosal barrier, activating innate immune cells, and enhancing inflammatory responses [24, 25]. Ultrafine outdoor pollutants from urbanization and industrialization can penetrate indoor environments and exacerbate respiratory symptoms [4]. For instance, PM_{2.5}, a major component of both indoor and outdoor air pollutants, in conjunction with Mucin 5 subtype AC, promoted nasal goblet cell proliferation and mucosal damage in mice [26, 27]. These exposures can also cause epigenetic modifications that are influenced by genome–environment interactions that contribute to AR [28]. Short-term and long-term exposures to high levels of PM_{2.5} can cause increased FoxP3 methylation, a key transcription factor in immune tolerance [24]. Furthermore,

PM_{2.5} exposure enhances DNA methylation of the interferon- γ gene promoter in CD4⁺ T cells via the extracellular signal-regulated kinase–DNA methyltransferase pathway, exacerbating the development of AR [29].

Moreover, numerous indoor activities, including heating, cooking, cleaning, candle burning, using fragrances, and smoking, generate pollutants such as VOCs, BTEX, and HCHO, which are associated with the development of AR [30]. Exposure to VOCs produced by the aforementioned activities is associated with irritation of the nasal mucosa and worsening of rhinitis, further supporting the link between exposure to VOCs and AR risk [31, 32]. BTEX carcinogenic compounds, mostly derived from vehicle emissions and cigarette smoke, can lead to oxidative stress in the body, which is characterized by an imbalance between free radicals and antioxidants and has been implicated in the pathophysiology of AR [23, 33]. Additionally, exposure to high levels of HCHO in the environment is commonly associated with increased hypersensitivity of the nasal mucosa to histamine, resulting in clinical symptoms such as mucosal irritation and olfactory disorders [34].

Although our analysis indicated an elevated risk of AR associated with ETS (OR = 1.57, 95% CI: 0.34–7.28), this finding did not reach statistical significance. Nevertheless, prior research has elucidated various biological mechanisms through which tobacco smoke may increase the risk of AR. For instance, ETS can upregulate mucus production, impair mucosal ciliary clearance, and induce low-grade inflammation in the airway mucosa. Additionally, tobacco smoke carries diverse microorganisms capable of colonizing the airways, which can alter the resident microbial community and disrupt microbial balance, thereby increasing the risk of AR [35, 36].

The disease mechanisms described above suggest that exposure to IAPs may not only increase the risk of developing AR but also exacerbate its severity, which may explain our additional findings. In the present study, additional analyses examined the association between high levels of IAPs and the seasonality, frequency, duration, and severity of AR.

The seasonality of AR symptoms suggests the existence of a significant allergen [37, 38]. We hypothesized, therefore, that IAPs occur throughout the year and do not exhibit specific seasonality. The association between IAP and AR symptoms, although not significant, suggests nonseasonality rather than seasonality. The prevalence of AR was not significant after adjusting for a number of covariates; however, the increase in the duration and severity of AR symptoms was significant. Further research is needed to investigate the association between exposure to IAPs and AR symptoms and the factors that contribute to their generation and removal, such as ventilation and cooking.

This study has some limitations. First, the cross-sectional design precludes causal inference. Future longitudinal cohort studies are warranted to examine how varying levels of IAPs influence the incidence and severity of AR over time. Additionally, controlled experimental studies with systematic manipulation of exposure to specific IAPs could provide robust evidence for elucidating causal pathways. Second, there may be limitations related to recall bias from self-

reported questionnaires. This bias can be mitigated, however, as we utilized national biomonitoring data and included a large sample size to offset interindividual variability. Further clinical or epidemiological longitudinal studies are required to evaluate this causality. Third, our results showed a significant correlation with high-intensity exposure to IAPs, rather than single exposures, which may be because our study participants only included adults with AR who are less sensitive to air pollution than children or adolescents with AR [20]. Nevertheless, an important point to note from our results is that despite individual pollutant concentrations above the median, as defined in our study, being much lower than the international regulatory thresholds, multiple exposures showed a significant increase in the risk and severity of AR. These results suggest that even low levels of exposure to IAPs can have adverse health effects, indicating a need for strengthened policy management [39]. The sample size of our study limited statistical power to detect small differences in AR prevalence and related symptoms. However, our statistical power was adequate for moderate-to-large effect sizes (approximately 6.5% difference in AR prevalence). Sensitivity analyses using multiple imputations demonstrated consistency with primary findings, indicating robustness against potential bias from missing data exclusion. We conducted multiple comparisons when evaluating associations between cumulative exposure to high-level IAP and AR-related symptoms. After applying FDR correction, the association between cumulative pollutant exposures and AR diagnosis remained statistically significant, underscoring the robustness of this finding. However, the association with long-duration symptoms lost statistical significance following FDR adjustment. Other AR-related symptoms (seasonal, frequent, and severe) did not demonstrate significant associations before or after correction. These findings emphasize that cumulative exposure to multiple IAPs significantly affects AR diagnosis but suggest caution when interpreting specific symptom outcomes due to potential false-positive results.

5. Conclusion

The findings of this study demonstrated that although the levels of IAPs were below international regulatory standards, the risk of AR increased with increasing number of high-level IAP exposures. These results highlight the importance of evaluating combined health impacts from multiple pollutant exposures. Given that international organizations such as WHO and EPA emphasize adherence to emission limits for key pollutants like HCHO and benzene, our study underscores the need for strengthened indoor air quality management policies and a deeper understanding of the health impacts of multiple IAP exposures in AR patients.

Data Availability Statement

The data that support the findings of this study are openly available in Korea Disease Control and Prevention Agency at <https://www.kdca.go.kr>.

Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

W.L. and A.-R.J. designed the study and wrote the manuscript. A.-R.J. and X.M. contributed to data collection. S.L. and W.L. performed the statistical analysis and interpretation of the results. All authors read and approved the final manuscript. S.L. and A.-R.J. contributed equally to this study.

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