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Ambient Endotoxin and Chemical Pollutant (PM_{10}, $PM_{2.5}$, and O_3) Levels in South Korea

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ABSTRACT

We measured the levels of airborne endotoxins in South Korea and compared them to PM_{10}, $PM_{2.5}$, and O_3 levels in ambient environments; environmental factors affecting these levels were also analyzed. A total of 81 air samples were collected and analyzed using the kinetic *Limulus Amebocyte Lysate* (LAL) assay. The geometric mean was determined for the levels of endotoxin (0.132 EU m^{-3}), PM_{10} (51.9 μg m^{-3}), $PM_{2.5}$ (22.6 μg m^{-3}), and O_3 (0.018 ppm). The endotoxin levels were significantly higher in fall and winter than in summer. The levels of PM_{10} and $PM_{2.5}$ were significantly higher, and the level of O_3 was by far its highest, in spring. Negative correlations were found between the endotoxin and O_3 levels ($r = –0.491$) and between the endotoxin levels and temperature ($r = –0.302$). The PM_{10} levels were also negatively associated with the O_3 levels and temperature but positively associated with the $PM_{2.5}$ levels. Given the negative relationship between airborne endotoxins and O_3 determined here, further studies with larger sample sizes are needed to identify the responsible mechanisms.

Keywords: Endotoxins; Particulate matter; Ozone; Ambient conditions; Seasons.

INTRODUCTION

A variety of air pollutants are legally required to be monitored in South Korea, including particulate matter with a diameter less than 10 and 2.5 μm (PM_{10} and $PM_{2.5}$, respectively), carbon monoxide, nitrogen dioxide, sulphur dioxide, and ozone (Ministry of Environment of Korea, 2017). However, due to a legal oversight, biological agents such as airborne endotoxins are not monitored in outdoor environments. Endotoxins such as lipopolysaccharides (LPSs) are ubiquitous in the environment and are an important structural component of the outer membranes of gram-negative bacteria (Beutler and Rietschel, 2003). Exposure to endotoxins has been found to cause and exacerbate asthma and wheezing in both children and adults (Abbing-Karahagopian *et al.*, 2012) and has also been linked to lung function impairment (Liebers *et al.*, 2008) and the pathogenesis of pulmonary diseases (Loh *et al.*, 2006). In addition, a recent study found that endotoxin exposure can dramatically alter the body's white blood cell count, leading to disorders in immune function (Shang *et al.*, 2016).

The health effects of PM_{10} are predominantly respiratory and cardiovascular, with impacts ranging from functional changes (e.g., reduced lung function) and impaired activities (e.g., school absenteeism, days off work) to reduced life expectancy and ultimately death (Kuschel *et al.*, 2012). Ambient $PM_{2.5}$ was the fifth-ranking global mortality risk factor in 2015, with exposure causing 4.2 million deaths (95% uncertainty interval, 3.7–4.8 million people) (Cohen *et al.*, 2017). A study of 500,000 adults in the urban United States reported that overall mortality, mortality of cardiopulmonary diseases, and lung cancer increased by 4%, 6%, and 8%, respectively, for every 10 μg m^{-3} $PM_{2.5}$ increase, after ruling out smoking, diet, drinking, occupational, and other risk factors (Pope *et al.*, 2002). $PM_{2.5}$ is also known to have neurotoxic effects, as these particles can enter human circulatory systems and affect various organs (Genc *et al.*, 2012), in addition to coming into contact with the brain through the nasal olfactory mucosa (Garcia *et al.*, 2015).

Exposure to O_3 causes respiratory symptoms, increases susceptibility to pulmonary infections, and even increases the risk of mortality in those with underlying cardiorespiratory conditions (Turner *et al.*, 2016). Moreover, endotoxin inactivation in the presence of O_3 becomes more efficient with increasing exposure time (Rezaee *et al.*, 2008).

Past research has evaluated airborne endotoxins, PM, and O_3 in outdoor environments, such as ambient endotoxins and PM_{10} in Chitwan, Nepal (Mahapatra *et al.*, 2018); ambient concentrations of PM_{10} and $PM_{2.5}$ in Palermo, Italy

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(Dongarra *et al.*, 2010); spatio-temporal variations in ambient PM_{10} and $PM_{2.5}$ concentrations in Beijing (Jie *et al.*, 2016); exposure to outdoor PM_{10}, $PM_{2.5}$, and O_3 in Singapore (Gall *et al.*, 2015); and ambient concentrations of O_3 under the influence of $PM_{2.5}$, NO_2, and SO_2 in Zhejiang, China (Chen *et al.*, 2017). However, no research has evaluated the relationships between airborne endotoxins and PM_{10}, $PM_{2.5}$, and O_3, although doing so would improve scientific understanding of these pollutants' airborne levels and distributions while collecting important background data for comparison between different countries.

Therefore, in this study we measured the ambient levels of airborne endotoxins atop two buildings in urban South Korea for one year and analyzed them with reference to PM_{10}, $PM_{2.5}$, and O_3 levels collected from Airkorea (www.airkorea.or.kr) to determine the relationship between these substances and the potential influence of environmental factors such as temperature and relative humidity.

METHODS

Study Setting

We collected endotoxin samples from two buildings in Ilsan, Goyang-si, Gyeonggi-do, near Seoul (Fig. 1), in the spring, summer, autumn, and winter (from March 2016 to February 2017). Sampling Point A was located on the roof of a 12-story building, while Sampling Point B was located at the top of the highest apartment in a 19-story building. These buildings were selected to determine the differences between cities with high-traffic roads (Sampling Point A) and residential areas without high-traffic roads (Sampling Point B). Air samples were collected from 100–150 cm above floor level for about three days per month at both locations (81 total samples).

Endotoxin Sampling and Analysis

During endotoxin sampling, temperature and relative humidity (RH) were recorded at each location using a Unis digital thermometer (YTH-104 series, Unis Inc., Korea). Samples were collected onto glass fiber filters (37 mm diameter; SKC Inc., USA) preloaded in a three-piece clear plastic cassette using an air sampler (17G9 GilAir Sampler, Sensidyne, Inc., USA) at a flowrate of 2.0 L min^{-1} (± 5%) for an average of 6 h. One field blank was collected on each sampling day and analyzed by kinetic-turbidimetric *Limulus Amebocyte Lysate* (LAL) assay (Associations of Cape Cod, Inc., USA) with no contamination. Precautions were taken to avoid breathing on, touching, or otherwise exposing the sampling containers to human contamination while sampling airborne endotoxins, including the use of gloves while connecting or disconnecting the cassette and the pump. After sampling, a protective covering back (cap) was placed on the cassette's inlet and outlet, and the entire cassette was wrapped in its original packing and sealed with tape.

The samples were stored at 4 ± 2°C, sent to an analytical laboratory within a week of sampling, and analyzed immediately upon arrival. Detection and quantification of endotoxin levels were conducted by kinetic-turbidimetric LAL assay. The entire endotoxin extraction procedure was conducted at room temperature (25 ± 2°C). An extraction volume of 15 mL of pyrogen-free water was added to a test tube, which was then capped and sonicated at a minimum peak frequency of 48 kHz for 1 h. After that, samples were centrifuged at 1000 g for 15 min, and the supernatant was transferred to a pyrogen-free test tube. 100 µL of each sample was distributed into a pyrogen-free 96-well micro-plate and incubated at 37°C for 10 min in an automated micro-plate reader (ELx808, BioTek Instruments, USA).

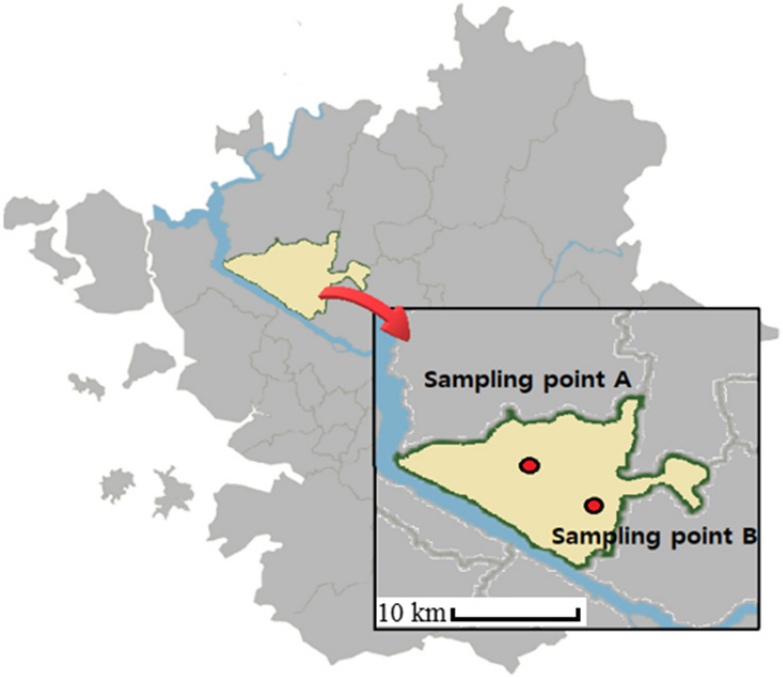

Fig. 1. Location of Sampling Points A and B on building rooftops in Goyang-si Gyeonggi-do, South Korea.

100 μL of LAL reagent was added to each well and analyzed in duplicate at 340 nm using Win KQCL Software (BioWhittaker, Cambrex Co., USA). The *Escherichia coli* O55:B5 control standard endotoxin (Lonza, USA) was utilized to draw a standard curve ranging from 0.005 to 50 endotoxin unit mL^{-1}. Only calibration curves greater than or equal to 0.98 were accepted for further analysis. Positive product control (PPC) recoveries within 50–200% and coefficients of variation (CV) less than 10% were considered valid. The endotoxin levels were expressed as endotoxin units per cubic meter of air (EU m^{-3}). The assay limit of detection (LOD) was 0.01 EU mL^{-1} extract. Values below the LOD were assigned a value of LOD/√2 (Hornung and Reed, 1990).

PM_{10}, $PM_{2.5}$, and O_3 Data

The ambient PM_{10}, $PM_{2.5}$, and O_3 levels were obtained from publicly available Airkorea data (www.airkorea.or.kr), a program of the government's National Ambient air quality Monitoring Information System (NAMIS), and compared with the sampled endotoxin levels (Airkorea, 2017). The same dates used for endotoxin sampling were used for outdoor PM_{10}, $PM_{2.5}$, and O_3 data along with the same 6 h sampling time, and the outdoor concentrations were obtained at locations within 1 km of Sampling Point A and 2.6 km of Sampling Point B.

Statistical Analyses

Statistical analyses were conducted using SAS software, version 9.3 (SAS Institute, Inc., USA). A nonparametric analysis was performed since the endotoxin and PM levels were not distributed normally or log-normally according to a Shapiro–Wilk test. The relationships between the endotoxin, PM_{10}, $PM_{2.5}$, and O_3 level distributions and the recorded ambient temperature and RH were analyzed using descriptive statistics. Kruskal–Wallis tests were performed to determine the differences between the endotoxin, PM_{10}, $PM_{2.5}$, and O_3 levels and the seasons, including between Sampling Points A and B. Mann-Whitney tests with Bonferroni adjustments were also carried out to determine which seasons were significantly different. In addition, Spearman's correlation analyses were employed to examine the associations between the endotoxin, O_3, PM_{10}, and $PM_{2.5}$ levels and temperature and RH.

RESULTS

Endotoxin levels ranged from 0.007 to 1.681 EU m^{-3} with a geometric mean (GM) of 0.132 EU m^{-3}, PM_{10} levels ranged from 23.0 to 166.0 μg m^{-3} with a GM of 51.9 μg m^{-3}, $PM_{2.5}$ levels ranged from 4.0 to 92.0 μg m^{-3} with a GM of 22.6 μg m^{-3}, and O_3 levels ranged from 0.003 to 0.059 ppm with a GM of 0.018 ppm (Table 1). Although endotoxin concentrations were higher at Sampling Point A (average GM of 0.147 EU m^{-3}) than at Sampling Point B (average GM of 0.115 EU m^{-3}), there was no significant difference ($p > 0.05$) between the two sampling points.

At the monthly scale, endotoxin levels were highest in October and lowest in April, PM_{10} and $PM_{2.5}$ levels were highest in March and lowest in September, and O_3 levels were highest in June and lowest in January (Table 2). To evaluate seasonal variations in these pollutants, we grouped the monthly levels by season: spring (March–May), summer (June–August), fall (September–October), and winter (November–February) (Fig. 2). Endotoxins were highest in fall and winter, followed by summer and spring, with significant differences between fall and spring ($p = 0.0003$) and between winter and spring ($p = 0.0008$). PM_{10} levels were highest in spring and winter and lowest in fall ($p = 0.0091$ between spring and fall, $p = 0.0037$ between winter and fall). $PM_{2.5}$ levels were highest in winter and lowest in fall ($p = 0.0027$ between fall and winter). O_3 levels were highest in summer and lowest in winter ($p < 0.0001$ between summer and winter).

Correlation analysis between endotoxin levels and PM_{10}, $PM_{2.5}$, O_3, temperature, and RH showed a negative association between endotoxins and O_3 ($r = –0.491$) and between endotoxins and temperature ($r = –0.302$); the remaining factors were not clearly correlated with endotoxins (Table 3). PM_{10} was negatively associated with O_3 and temperature.

DISCUSSION

This study analyzed the distribution of ambient airborne endotoxin levels for a year at the top of two buildings in the Ilsan area of South Korea and assessed the influence of environmental factors on endotoxin levels. Endotoxin levels ranged from 0.007 to 1.681 EU m^{-3} (GM of 0.132 EU m^{-3}). In comparison, airborne endotoxin levels in outdoor urban areas of Stockholm, Sweden, ranged from 0.020 to 0.107 EU m^{-3} (GM of 0.05 EU m^{-3}) (Nilsson *et al.*, 2011), while areas with intensive livestock production in the Netherlands recorded endotoxin levels of 2.0–2.9 EU m^{-3} and 0.46–0.66 EU m^{-3} in residential gardens at least 500 m from the nearest farm (Schulze *et al.*, 2006; Rooij *et al.*, 2010). These variations in reported endotoxin levels may be due to differences in sampling and extraction methods, as well

Table 1. Overall levels of chemical pollutants and climate (temperature and relative humidity) in ambient environments.

Materials and climate	No. of samples	GM (GSD)	Min	Median	Max
Endotoxin (EU m^{-3})	81	0.132 (1.3)	0.007	0.118	1.681
PM_{10} (μg m^{-3})	81	51.9 (1.5)	23.0	49.0	166.0
$PM_{2.5}$ (μg m^{-3})	65	22.6 (1.9)	4.0	26.0	92.0
O_3 (ppm)	66	0.018 (2.1)	0.003	0.018	0.059
Temperature (°C)	-	15.9 (11.3)	–4.2	18.5	34.9
Relative humidity (%)	-	46.6 (12.4)	21.1	46.7	74.5

Table 2. Characteristics of endotoxin levels (measured in this study) and PM_{10}, $PM_{2.5}$, and O_3 levels (measured by Airkorea).

Month	N*	Endotoxin level (EU m^{-3})			N*	PM_{10} level (μg m^{-3})			N*	$PM_{2.5}$ level (μg m^{-3})			N*	O_3 (ppm)		
		GM (GSD)	Min	Max		GM (GSD)	Min	Max		GM (GSD)	Min	Max		GM (GSD)	Min	Max
Jan.	7	0.054(1.909)	0.025	0.124	7	51.4 (1.4)	35.0	90.0	6	28.4 (1.9)	10.0	54.0	1	0.005 (-)	0.005	0.005
Feb.	7	0.111(2.024)	0.040	0.333	7	52.5 (1.3)	39.0	74.0	6	32.4 (1.6)	18.0	64.0	2	0.010 (1.5)	0.007	0.013
Mar.	8	0.100(1.949)	0.034	0.210	8	102.7 (1.4)	49.0	166.0	6	35.4 (1.8)	15.0	92.0	8	0.014 (2.0)	0.005	0.034
April	8	0.032(2.223)	0.007	0.075	8	75.3 (1.4)	44.0	118.0	6	21.6 (1.3)	14.0	30.0	4	0.013 (1.1)	0.012	0.015
May	5	0.087(2.025)	0.037	0.239	5	39.0 (1.2)	29.0	49.0	5	18.1 (1.6)	11.0	34.0	5	0.037 (1.3)	0.029	0.048
Jun.	7	0.054(3.148)	0.009	0.212	7	44.0 (1.4)	29.0	81.0	6	20.1 (1.9)	7.0	45.0	7	0.042 (1.3)	0.026	0.059
July	5	0.058(3.603)	0.017	0.237	5	39.6 (1.3)	28.0	52.0	4	24.8 (1.5)	17.0	43.0	5	0.030 (1.3)	0.023	0.040
Aug.	7	0.095(1.801)	0.031	0.212	7	44.2 (1.3)	34.0	67.0	6	21.3 (2.6)	5.0	64.0	7	0.032 (1.3)	0.024	0.045
Sep.	6	0.385(2.383)	0.139	1.224	6	32.3 (1.4)	23.0	52.0	4	10.1 (2.1)	6.0	31.0	6	0.020 (1.5)	0.011	0.030
Oct.	7	0.600(2.381)	0.180	1.681	7	41.7 (1.3)	31.0	63.0	4	10.1 (2.3)	4.0	29.0	7	0.016 (1.4)	0.008	0.023
Nov.	7	0.528(2.187)	0.162	1.524	7	61.2 (1.2)	45.0	75.0	6	27.6 (1.8)	11.0	51.0	7	0.008 (1.8)	0.003	0.019
Dec.	7	0.506(2.447)	0.075	1.052	7	51.3 (1.3)	31.0	64.0	6	27.2 (1.3)	17.0	35.0	7	0.008 (2.0)	0.003	0.018

*Number of samples.

as prevalent environmental conditions (Balasubramanian *et al.*, 2012; Duquenne *et al.*, 2013). Currently, there are no established standards for endotoxin exposure, although the National Health Council of the Netherlands has set a recommended threshold value of 90 EU m^{-3} (Health Council of the Netherlands, 2010). However, studies have shown that endotoxins affect health even at much lower concentrations (Ryan *et al.*, 2009; Bennett *et al.*, 2012). Rabinovitch *et al.* (2005) reported an increase in the severity of asthma in children exposed to endotoxin levels of 0.08 EU m^{-3}.

Our results showed that endotoxin concentrations were higher in a high-traffic urban setting (Sampling Point A) than in a low-traffic residential area (Sampling Point B), similar to a previous study reporting that endotoxin concentrations on congested streets (median = 4.4 EU m^{-3}) were higher than in residential areas (median = 0.33 EU m^{-3}) (Madsen, 2006). Exposure to traffic-related particles is associated with childhood respiratory problems, and a synergistic relationship exists between co-exposure to traffic-related particles and endotoxins with regard to persistent respiratory problems during infancy through 3 years of age (Ryan *et al.*, 2009).

The average ambient GM (GSD) PM_{10} and $PM_{2.5}$ levels in this study were 51.9 (1.5) and 22.6 (1.9) μg m^{-3}, respectively—less than the 100 and 50 μg m^{-3} from Airkorea (2017) and the 50 and 25 μg m^{-3} from the WHO (2016). However, these average GM PM_{10} levels were higher than those reported for areas with livestock farms (19.8–22.3 μg m^{-3}; Rooij *et al.*, 2017), and the average $PM_{2.5}$ levels were higher than reported for urban rooftops near busy roads in Brisbane, Australia (8.0–19.0 μg m^{-3}; Quang *et al.*, 2012). Other PM reports include those from atop a six-story building in Beijing (GM of $PM_{2.5}$ levels ranging from 6.4 to 463.5 μg m^{-3} and averaging 61.7 μg m^{-3}; Guan *et al.*, 2014), from 38 of China's largest cities (daily mean PM_{10} level of 92.9 μg m^{-3}; Yin *et al.*, 2017), from a traffic site in Algeria (GMs of 105.2 μg m^{-3} for PM_{10} and 57.8 μg m^{-3} for $PM_{2.5}$; Terrouche *et al.*, 2016), and from two traffic sites in Lahore, Pakistan (GMs of 286 and 365 μg m^{-3} for PM_{10} and 222 and 302 μg m^{-3} for $PM_{2.5}$; Ali *et al.*, 2015), all of which were higher than those measured in this study. These differences between PM levels in different cities are likely caused by measurement differences, which can vary widely due to sampling procedures and equipment, even for the same pollutant in the same location (Amaral *et al.*, 2015).

Ambient O_3 levels in this study ranged from 0.003 to 0.059 ppm (overall GM of 0.018 ppm), less than the 8 hour levels measured by Airkorea (2017). Other reports of outdoor O_3 levels include those from British Columbia (0.028 ppm) and southern Ontario (0.037 ppm) in 2014 (ECCC, 2016). A European study noted that the concentration of surface ozone had increased from an estimated preindustrial value of 0.01 ppm to 0.03–0.05 ppm (Pritchard and Amthor, 2005). According to the United States Environmental Protection Agency (EPA), controlled studies of prolonged human ozone exposure at levels below 0.08 ppm showed respiratory effects, changes in lung function, and increased airway responsiveness; animal toxicology studies have provided additional evidence of such effects (NAAQSO, 2008).

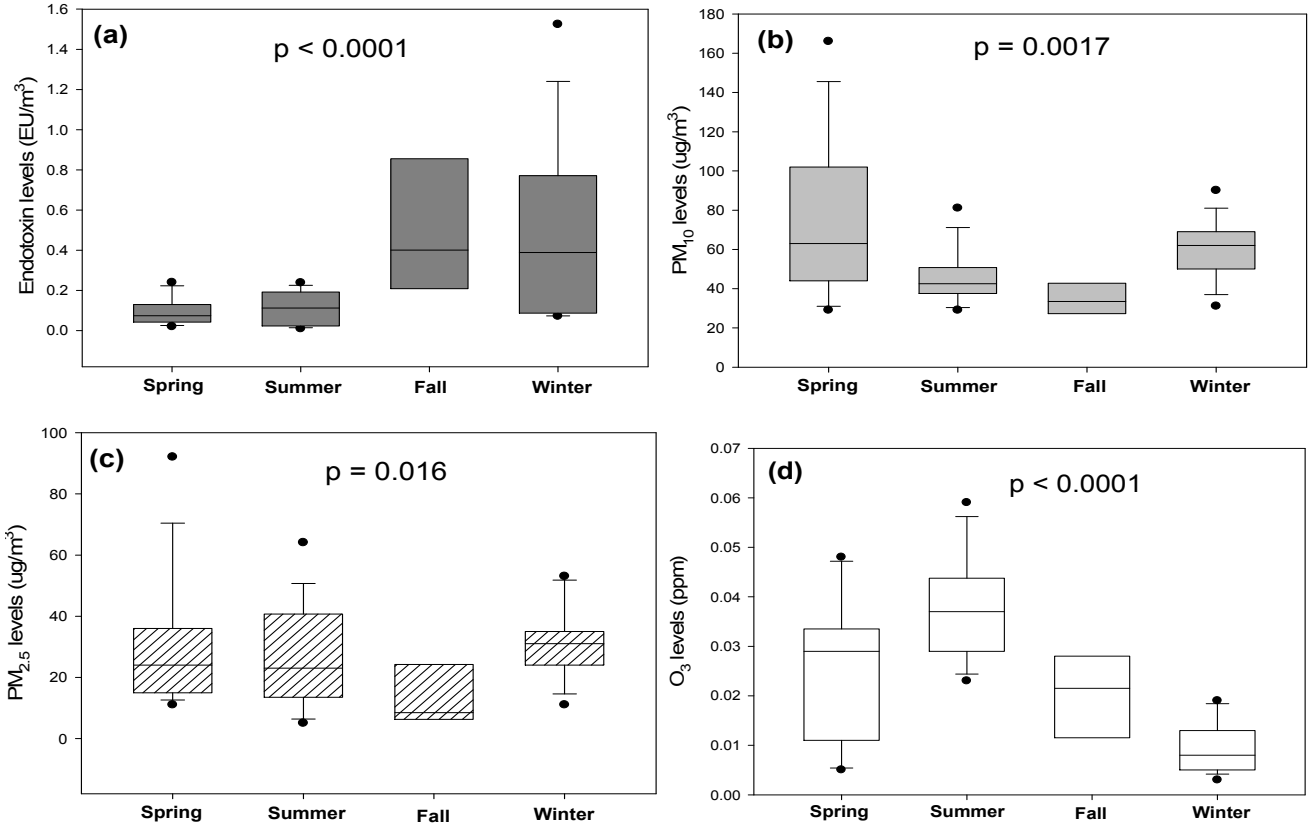

Fig. 2. Seasonal variations in levels of (a) endotoxin, (b) PM_{10}, (c) $PM_{2.5}$, and (d) O_3.

Table 3. Correlation analysis between levels of endotoxins, PM_{10}, $PM_{2.5}$, O_3, temperature, and relative humidity.

	Endotoxin level (EU m^{-3})	PM_{10} (μg m^{-3})	$PM_{2.5}$ (μg m^{-3})	O (ppm)	Temperature (°C)	Relative humidity (%)
Endotoxin level (EU m^{-3})	1.000					
PM_{10} (μg m^{-3})	0.027	1.000				
$PM_{2.5}$ (μg m^{-3})	0.026	0.755**	1.000			
O_3 (ppm)	-0.491**	-0.357*	-0.096	1.000		
Temperature (°C)	-0.302*	-0.356*	-0.159	0.672**	1.000	
Relative humidity (%)	-0.121	-0.277*	-0.046	0.445*	0.608**	1.000

*$p < 0.05$; **$p < 0.001$, n = 52

Previous studies have shown that airborne endotoxin levels were higher in spring and summer than in fall and winter (Carty *et al.*, 2003; Kallawicha *et al.*, 2015). In contrast, our results found the highest levels in fall (September–October) and the lowest levels in spring (March–May), with significant seasonal differences (Fig. 2). Differences in meteorological factors (e.g., rain, wind, sunlight hours, temperature, and humidity) likely explain this seasonal variability in endotoxin levels (Carty *et al.*, 2003). Temperature and RH were found to be the most influential, with the highest endotoxin concentrations recorded during warm periods and moderate RH (35–75%) in ambient environments (Allen *et al.*, 2011). Traversi (2010) observed that temperature plays a predominant role in endotoxin modulation within the environment and showed that temperature has a negative correlation with endotoxin levels. On the other hand, Mahapatra *et al.* (2017) reported that endotoxin levels showed a weak positive correlation with temperature (r = 0.34). This discrepancy might be due to temperature variations because Mahapatra *et al.* (2017) mentioned that a temperature variation of 22–28°C was not sufficient to affect endotoxin levels; Su *et al.* (2001) also observed no significant correlation with a small change in temperature. Although we found no significant correlation between RH and endotoxin levels, RH had a weak negative correlation with endotoxin in a similar pattern as reported by Mahapatra *et al.* (2017) in which a weak positive correlation (r = 0.2) between endotoxin concentration and RH from 38–60% was observed, suggesting that moderate RH aided bacterial growth but higher RH levels (60–90%) reduced endotoxin levels.

We found no clear correlations between endotoxin and PM_{10} or $PM_{2.5}$ levels; this was consistent with Rooij (2017), who suggested that lower correlations related to inherent

variability in endotoxin levels were due to the influence of sampling and analytical variability. However, PM_{10} levels were significantly negatively correlated with O_3 levels ($p < 0.05$), similar to results given in Chen *et al.* (2017), because high particle concentrations in ambient air could make the atmosphere cooler and reflect sunlight above the ground, resisting the formation of O_3 (Moss *et al.*, 2010).

O_3 levels varied significantly between seasons and were much higher during summer than winter (Fig. 2). This result was consistent with Chen *et al.* (2017) and other studies, suggesting that severe O_3 pollution can benefit from higher sunlight intensity and temperature (Stathopoulou *et al.*, 2008). O_3 was positively correlated with temperature ($p < 0.001$), suggesting that higher temperature is beneficial to O_3 formation in ambient air because higher temperature can accelerate reaction among precursors and their intermediate products such as free radicals; this result was consistent with the principle reported by Coates *et al.* (2016). High RH indicates that the atmosphere contains more water molecules, which play a vital role in the formation of O_3 (Calvert *et al.*, 2015), as demonstrated by the significantly positive correlation between O_3 and RH.

Our study is the first to show a correlation between endotoxin levels and chemical pollutants such as PM_{10}, $PM_{2.5}$, and O_3 in ambient environments, but some limitations should be considered. First, our outdoor measurements of endotoxins, PM, and O_3 did not necessarily accurately reflect the correlation between endotoxin levels and outcomes as they were not conducted simultaneously. Second, the sources of PM_{10} and $PM_{2.5}$ were not specifically identified; further studies would need to measure PM more directly to better understand the components of atmospheric environments. Third, the short daily sampling period (6 h) may have introduced some variation among measurements, resulting in poorer representation and weaker consistency between the concentrations for the entire day. Finally, the limited sample size may not have been representative of the ambient levels of endotoxins in comparison with pollutant levels, resulting in possible biases. Despite these limitations, this study was conducted for a substantial period of time using standard air sampling methods, which increases the validity of the measurement comparisons. Moreover, identifying levels of ambient endotoxins in relation to PM_{10}, $PM_{2.5}$, and O_3 is a new step toward a better understanding of their interrelated dynamics across a large metropolitan area in South Korea.

CONCLUSION

Airborne endotoxin levels were measured from the tops of two buildings in the city of Goyang in Gyeonggi Province, South Korea, and compared with PM_{10}, $PM_{2.5}$, and O_3 data recorded in nearby locations. The endotoxin levels were significantly higher in fall and winter than in summer. The levels of PM_{10} and $PM_{2.5}$ were significantly higher in spring than the other seasons, and spring also had the highest O_3 levels. The endotoxin and O_3 levels were found to be negatively correlated ($r = –0.491$), as were the endotoxin and temperature levels ($r = –0.302$). The PM_{10} levels were also negatively associated with the O_3 levels and temperature. Further studies, especially with a larger sample size, are needed to identify the prevalent mechanism causing these relationships.

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