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Short-term exposure to ambient air pollution and injuries due to external causes according to intentions and mechanisms

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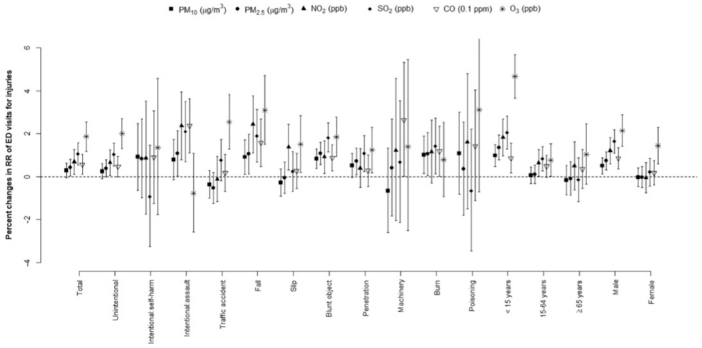
HIGHLIGHTS

- We assessed air pollution-injury associations by intention and mechanism of injury.
- Immediate (lag 0 or lag 1) exposure significantly increased the risk of injuries.
- A significant association was found for unintentional and assault injuries.
- Most mechanisms, except for burns and poisoning, showed a significant association.
- Stronger associations were found in patients aged <15 years and males.

GRAPHICAL ABSTRACT



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ABSTRACT

Although injuries are a leading cause of death and affect the life expectancy of individuals who live with disabilities globally, the potential role of air pollution exposure on injuries due to external causes has received little scientific attention, especially compared with that given to the association of air pollution and non-external causes of morbidity and mortality. We investigated the association between emergency department visits for externally caused injuries and short-term exposure to major ambient air pollutants, with focus on the intentions and mechanisms of injuries. We identified 2,049,855 injured patients in Seoul, South Korea between 2008 and 2016 using the National Emergency Database. Daily short-term exposure to air pollution including particles <10

Abbreviations: DALY, disability-adjusted life year; df, degrees of freedom; ED, emergency department; NEDIS, National Emergency Department Information System; NEMC, National Emergency Medical Center; RR, relative risk.

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Mechanism
Emergency department visit

μm (PM_{10}) and <2.5 μm ($PM_{2.5}$), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3) was estimated based on hourly concentrations. We employed a time-stratified case-crossover study design using a conditional Poisson regression model adjusted for meteorological variables, influenza epidemics, and holidays. Immediate exposure (lag 0) to most pollutants significantly increased the risk of total injuries ($PM_{2.5}$, 0.42 %; NO_2, 0.68 %; SO_2, 1.05 %; CO, 0.57 %; O_3, 1.86 % per interquartile range increment), and the associations differed according to the intention and mechanism of injury. Unintentional and assault injuries were significantly associated with air pollution exposure, whereas self-harm injuries showed no association. In mechanism-specific analyses, injuries caused by falls, blunt objects, penetration, traffic accidents, machinery, and slips were associated with specific air pollutants, even in the co-pollutant models. The associations were stronger in injured patients aged <15 years, and in males than in their counterparts. Our results suggest that short-term air pollution exposure might play a role in the risk of externally caused injuries and the association may differ depending on the intention and mechanism of injury, which provide important evidence for injury prevention and air quality strategies.

1. Introduction

Ambient air pollution has emerged as a leading environmental health risk over the past few decades and is responsible for numerous adverse health outcomes (Brook et al., 2010; Landrigan, 2017; Peel et al., 2005; Zhang et al., 2018). It caused an estimated 4.2 million deaths and 103 million disability-adjusted life years (DALYs) worldwide in 2015, which accounted for approximately 13 % and 10 % of all deaths and DALYs by all risk factors (GBD 2015 Risk Factors Collaborators, 2016). According to the World Health Organization (WHO), ambient air pollution-related premature deaths were caused by ischemic heart disease and stroke (37 %), chronic obstructive pulmonary disease (18 %), lower respiratory infections (23 %), and respiratory tract cancer (11 %) (WHO, 2019). Despite continuous warnings to reduce air pollution levels, 99 % of the global population in 2019 lived in polluted areas wherein the WHO air quality guideline levels were not met (WHO, 2019). To encourage policymakers, stakeholders, and the public to make efforts to reduce air pollution levels, it is essential to assess its various negative health impacts and provide scientific evidence to support related policies and interventions.

However, the majority of air pollution studies have focused on non-external causes of death and morbidities, such as cardiovascular and respiratory diseases (Brook et al., 2010; Guan et al., 2016; Samet et al., 2000), although it is plausible that short-term exposure to ambient air pollution could affect injuries via relevant mechanisms through oxidative stress and neuroinflammation in the central nervous system (Block and Calderón-Garcidueñas, 2009; Sahel et al., 2019). Previous studies from neuroscientific experiments have observed the neurological impairments after short-term air pollution exposure including decreased motor activity (Dorado-Martínez et al., 2001), memory/cognitive impairment (Avila-Costa et al., 1999), and neurobehavioral performance impairment (Sorace et al., 2001), which are related to injury-related factors such as distractibility/attention deficits, impaired judgment, and executive dysfunction (Day et al., 2012; Kass et al., 2010; Pelicioni et al., 2021). It has also been hypothesized that short-term air pollution exposure could trigger intentional self-harms or aggressive behaviors through dysfunction of neurotransmitters and systemic inflammation (Batty et al., 2016; Yokota et al., 2013).

To date, limited studies have given attention to the impacts of ambient air pollution on injuries resulting from external causes, despite the fact that injuries—both unintentional and intentional—account for 4.4 million deaths worldwide each year (WHO, 2021). Globally, three of the top five causes of death in people aged 5–29 years are injuries due to traffic accidents, homicide, and suicide (WHO, 2021). In South Korea, unintentional injuries due to traffic accidents, falls, suicide, and homicide were ranked among the top five age-specific causes of death in 2020 (KOSIS, 2021).

Injuries can occur unintentionally or intentionally through diverse mechanisms including poisoning, falls, and traffic accidents (WHO, 2021). Separate studies have examined the associations between ambient air pollution and suicide (Bakian et al., 2015; Lee et al., 2018; Ng et al., 2016), violent behavior (Berman et al., 2019; Burkhardt et al., 2020), occupational accidents (Vega-Calderón et al., 2021), and injuries due to traffic accidents (Chan et al., 2022). However, to the best of our knowledge, no efforts have been made to comprehensively understand the association between ambient air pollution exposure and injuries due to external causes while considering the intentions and mechanisms of injury.

In the present study, we hypothesized that short-term exposure to high ambient air pollution may play a role in the risk of injuries due to external causes and that the association may differ depending on the intention and mechanism of injury. To investigate this relationship, we conducted a time-stratified case-crossover study in the capital city of South Korea where injuries are among the leading causes of death.

2. Methods

2.1. Data source for the study population

We obtained data of patients who had visited emergency departments (EDs) from the National Emergency Medical Center (NEMC) founded by the Ministry of Health and Welfare in 2001 (https://dw.nemc.or.kr/). The NEMC was established to maintain and manage the emergency medical system in South Korea, and emergency medical records from regional and local emergency medical centers were collected and standardized. In 2003, the NEMC established the National Emergency Department Information System (NEDIS), which is the largest database of patients with recorded ED visits across South Korea. The NEDIS has been stable since 2005 and had registered approximately 98 % of emergency medical records from all emergency medical facilities in 2016. We obtained the following information from the NEDIS: 1) demographic information: age, sex, medical insurance type, and region of emergency medical facilities; 2) ED information: date/time of ED visit and symptom onset, route of and reason for ED visit, and mental status on ED arrival (alert or unresponsive); 3) injury information: intentions (unintentional, self-harm, or assault), mechanisms (traffic accident, falling, slipping, blunt object use, penetration, machinery, burns, drowning, poisoning, or suffocation/hanging); and 4) medical information: initial diagnosis, results of ED treatment (discharge, transfer, admission, or death), and discharge/admission information (final diagnosis and date/time at discharge/admission) (Lee et al., 2020).

2.2. Definition of injuries

The outcome of interest in this study was injuries due to external causes. From the NEDIS database, we extracted data of patients who had visited EDs with an injury as the primary diagnosis at discharge (International Classification of Disease 10th revision [ICD-10] codes S00–T98), except for T78, T80–T88, and T90–T98 (adverse effects, complications of surgical/medical care, sequelae of injuries/poisoning, and other consequences of external causes) in Seoul between 2008 and 2016. For all injured patients, the external causes (V01–Y98) that

induced injuries were coded accordingly. Seoul, the capital city of South Korea, accounts for approximately one-fifth of the country's total population (≈50 million). As the most urbanized city, Seoul's population density (16,541/km^2) and traffic volume (32 million vehicle trips/day) are extremely high (SMG, 2021). Moreover, regular measurements of $PM_{2.5}$ (particulate matter <2.5 μm in diameter) have been conducted in Seoul since 2002, whereas in other cities, the measuring only began in 2015. Given these factors, Seoul is the optimal city for studying the association between exposure to high ambient air pollution and injuries due to external causes as it provides sufficient information and statistical power.

The study protocol was approved by the Institutional Review Board of Soonchunhyang University, South Korea (IRB no. 202108-SB-076-02). The requirement for informed consent was waived by the IRB because anonymous data were used.

2.3. Assessment of ambient air pollution exposure

The primary exposure of interest in this study was short-term air pollution exposure, defined as the daily levels of six major air pollutants (particulate matter <10 μm [PM_{10}], particulate matter <2.5 μm [$PM_{2.5}$], nitrogen dioxide [NO_2], sulfur dioxide [SO_2], ozone [O_3], and carbon monoxide [CO]). As the capital city, Seoul consists of 25 administrative districts (area mean: 24.21 km^2; range: 9.96–47.03 km^2), and each district has one air pollution monitoring site. We obtained hourly concentration data of the six air pollutants measured at 25 monitoring sites from the National Institute of Environmental Research (Incheon, South Korea). Each pollutant was measured every 5 min using the following methods: beta-ray absorption (PM_{10}), gravimetry ($PM_{2.5}$), chemiluminescence (NO_2), pulse ultraviolet fluorescence (SO_2), nondispersive infrared (CO), and ultraviolet photometry (O_3). Hourly concentrations were computed if 75 % of the measured values were effective. These measurements were conducted according to the standard protocols of the Korean Ministry of Environment (MOE, 2011).

We calculated daily city-level representative values because information on patient- or ED-specific addresses was not provided owing to privacy issues. First, we averaged the hourly concentrations obtained from 25 monitoring sites. Second, based on the 24 averaged hourly concentrations, we constructed daily representative values (24-h mean for PM_{10}, $PM_{2.5}$, NO_2, and SO_2; 8-h maximum mean for O_3 and CO). We confirmed that the 25 site-specific concentrations were highly correlated with each other and distributed unimodally, thereby suggesting homogeneity of air pollution levels within Seoul (Pearson correlation coefficients r [$N = {}_{25}C_2 = 300$], PM_{10}: mean = 0.953 [standard deviation = 0.020]; $PM_{2.5}$: 0.908 [0.044]; NO_2: 0.838 [0.050]; SO_2: 0.721 [0.073]; O_3: 0.894 [0.028]; CO: 0.779 [0.074]).

2.4. Potential confounders

Hourly data of meteorological variables (ambient temperature, relative humidity, barometric pressure, precipitation, and sunlight) that had been measured at a weather monitoring station in Seoul were obtained from the Korea Meteorological Administration. As short-term covariates, we calculated the 24-h mean values of the meteorological variables, except for precipitation (mm) and sunlight (h), for which the total amount per day was calculated. We also constructed indicator variables representing influenza epidemics and holidays to control for these potential confounding factors.

2.5. Study design

A time-stratified case-crossover study design was employed to investigate the association between short-term exposure to ambient air pollution and injuries due to external causes. The case-crossover design is an appropriate method for the evaluation of a short-term association as it can control for time-invariant characteristics (such as sex and genetic predisposition) and slowly time-varying factors (lifestyle, chronic morbidity, and long-term time trends) because each patient serves as their own control (Maclure, 1991). In this study, we used a time-stratified method in which each ED visit day (case) of the injured patients was matched to three or four control days on the same day of the week, month, and year. For instance, if a patient visited an ED due to injuries on Friday, March 13, 2015, all other Fridays in March 2015 were selected as control days.

2.6. Statistical analysis

We employed a conditional Poisson regression model, which is a flexible alternative method for analyzing the case-crossover dataset, instead of a conditional logistic regression model (Armstrong et al., 2014) for the effective analyses of our large dataset on injured patients. In this conditional Poisson model, the outcome variable is the aggregated count of ED visit events occurring on the same day and is regressed in the same way as in a daily time-series analysis. Moreover, the conditional Poisson regression model can adjust for overdispersion and autocorrelation and provide an estimate identical to that estimated from a conditional logistic regression model (Armstrong et al., 2014). The relative risk (RR) of ED visits for total injuries due to external causes associated with short-term air pollution exposure was estimated using the conditional Poisson regression model. The realized model in R software has the following form:

$$\begin{aligned}\text{model} = \text{gnm}\Bigg(&\text{Injury Count}_t \sim \text{air pollutant}_{t-l} + ns\left(\frac{1}{3}\sum_{i=0}^{2}\text{temperature}_{t-i}, 4\right) + \\ &\frac{1}{3}\sum_{i=0}^{2}\text{relative humidity}_{t-i} + \frac{1}{3}\sum_{i=0}^{2}\text{barometric pressure}_{t-i} + \\ &\frac{1}{3}\sum_{i=0}^{2}\text{sunlight hours}_{t-i} + \frac{1}{3}\sum_{i=0}^{2}\text{precipitation}_{t-i} + \\ &\text{factor}(\text{influenza}_t) + \text{factor}(\text{holiday}_t), \\ &\text{eliminate} = \text{factor}(\text{year}:\text{month}:\text{dow}), \text{data} = \text{data}, \ \text{family} = \text{quasipoisson}\Bigg) \\ &(l = 0, 1, 2, 3)\end{aligned}$$

where gnm is the function to fit generalized nonlinear models; InjuryCount$_t$ is the number of ED visits for injuries on day t; air pollutant$_{t-l}$ is each six air pollutants on day t-l; three-day moving averages (lag 0–2) of temperature, relative humidity, barometric pressure, sunlight hour, and precipitation were adjusted; ns () represents a natural spline for temperature and the degree of freedom (df) for the spline was four; influenza epidemic and holiday variables were included in the model as factors; eliminate term is the stratum for a conditional Poisson regression used for the case-crossover design. Covariate selection was based on previous studies (Lee et al., 2018; Ng et al., 2016). We constructed single and cumulative lag structures up to three days prior to ED visits for all air pollutants. Each lag-specific air pollutant was included separately in the regression model. The best fitting lag structure was selected based on the quasi-Akaike information criterion (QAIC) with the smallest value which indicates the best goodness-of-fit (Gasparrini et al., 2010), and was used for sensitivity analyses.

We further examined whether the association between short-term air pollution and injuries due to external causes differed with respect to the intention behind the injuries (unintentional or intentional [self-harm and assault]), mechanisms of injuries (traffic accident, falling, slipping, blunt object use, penetration, machinery, burns, and poisoning), and demographic characteristics (age [<15, 15–64, >64 years] and sex), by running separate models for each subgroup. Additionally, we assessed whether air pollution-injury association by mechanism of injury differed depending on the intention behind the injuries to identify a more predominant mechanism for the unintentional and intentional injuries. These analyses were conducted only for mechanism-specific subgroups with sufficient sample sizes (n = 2500) within each

intention-specific subgroup.

Several sensitivity analyses were conducted to confirm robustness of the primary findings. First, we applied a moving average (lag 0–1, 0–2, and 0–3) instead of a single lag for air pollutants. Second, we conducted co-pollutant analyses to investigate the potential confounding effects of other air pollutants. The same lag structure as the primary pollutant was selected to adjust for other pollutants. Third, we changed the model specifications for meteorological variables as follows: 1) constructing natural cubic splines for all meteorological variables other than temperature and changing the df of natural cubic splines (from 3 to 4, 5, and 6); and 2) changing lag structures (from lag 0–2 to lag 0–1, 0–3, and 0–4). Fourth, we analyzed the extended lag effects of air pollutants up to 7 days instead of 3 days to confirm whether the air pollution effects are immediate. All sensitivity analyses were conducted for the total injuries and each subgroup according to mechanisms, intentions, and demographic characteristics.

All analytical processes were performed using R software (version 3.5.3; R Development Core Team) and SAS (version 9.4; SAS Institute Inc., Cary, NC, USA). Results are presented as percent changes in the RRs of ED visits per interquartile range (IQR) increment of air pollutants with two-sided 95 % confidence intervals (CIs).

3. Results

Table 1 presents the descriptive statistics of daily ED visits for injuries, daily levels of air pollutants, and meteorological variables between 2008 and 2016. The average daily count for total injuries was 623.4, and most occurred unintentionally (587.4). Injuries caused by blunt objects (170.6) were the most frequent, followed by slipping (150.4), car accidents (123.8), and penetration (94.5). Injuries occurred more frequently in males (366.8) and those aged 15–64 years (392.6) than in females and other age groups. The 24-h mean (SD) of air pollution concentrations was 48.7 (29.4) $\mu g/m^3$ for PM_{10}, 24.6 (13.3) $\mu g/m^3$ for $PM_{2.5}$, 35.3 (12.3) ppb for NO_2, and 5.3 (1.9) ppb for SO_2, and the 8-h maximum mean was 6.5 (2.6) (0.1 ppm) for CO and 31.2 (16.8) ppb for O_3. All air pollutants were highly intercorrelated (r = 0.52–0.83), except for O_3 (r = −0.38–0.08), and moderately or weakly correlated with meteorological variables (Table S1).

Table 2 shows the mechanisms of injury for each unintentional and intentional (self-harm and assault) injury in the total and sex- and age-specific subgroups. For unintentional injuries, slipping, blunt object use, car accidents, and penetration accounted for 25.5 %, 24.6 %, 21.1 %, and 15.2 % of injuries, respectively. The most frequent causes for self-harm injuries were poisoning (46.5 %), penetration (32.5 %), and blunt object use (10.7 %), and 87.8 % of assault injuries occurred via blunt objects. The sex- and age-specific subgroups shared similar patterns with total injuries, except that self-harm injuries caused by blunt objects occurred more frequently in males (19.4 %) than in females (3.4 %), and that the predominant mechanism for unintentional injuries was via blunt objects (37.8 %) for injured patients aged <15 years, car accidents (27.0 %) for those aged 15–64 years, and slipping (49.9 %) for those aged >64 years.

Fig. 1 presents the association between short-term air pollution exposure and ED visits for total injuries and subgroups according to the intention behind the injuries. The IQR increment for $PM_{2.5}$, NO_2, SO_2, CO, and O_3 on the day of ED visits (lag 0) significantly increased the risk of ED visits for total injuries by 0.42 % (95 % CI, 0.03 %–0.81 %), 0.68 % (0.11–1.25 %), 1.05 % (0.54–1.57 %), 0.57 % (0.11–1.03 %), and 1.86

Table 1
Descriptive statistics of sample sizes, daily ED visits for injuries, ambient air pollutants, and meteorological variables in Seoul, South Korea, 2008–2016.

Variable	Sample size	Mean	SD	Percentile					IQR
				0	25	50	75	100	
Injury (n)									
Total	2,049,855	623.4	179.4	224	499	599	719	1298	
Intention									
Unintentional	1,931,424	587.4	171.6	210	469	565	678	1244	
Intentional: Self-harm	24,603	7.5	3.3	0	5	7	10	21	
Intentional: Assault	93,828	28.5	9.7	5	22	27	34	71	
Mechanism									
Traffic accident	406,912	123.8	37.8	35	97	119	145	268	
Fall	127,736	38.8	18.3	4	24	37	51	104	
Slip	494,621	150.4	49.8	54	118	141	174	696	
Blunt object	560,883	170.6	52.5	52	134	161	199	364	
Penetration	310,583	94.5	39.1	17	64	90	119	255	
Machinery	16,970	5.2	2.7	0	3	5	7	20	
Burns	106,888	32.5	14.3	8	23	29	39	130	
Poisoning	21,876	6.7	3.7	0	4	6	9	39	
Age									
<15 years	544,206	165.5	54.0	51	127	157	193	395	
15–64 years	1,290,832	392.6	114.7	141	312	374	454	842	
>64 years	214,817	65.3	25.3	14	46	63	82	216	
Sex									
Male	1,206,173	366.8	103.1	125	294	354	423	763	
Female	843,682	256.6	80.1	92	200	246	299	644	
Air pollutants									
PM_{10} ($\mu g/m^3$)		48.7	29.4	4.6	30.9	43.1	59.1	568.7	28.2
$PM_{2.5}$ ($\mu g/m^3$)		24.6	13.3	3.3	15.5	22.0	30.3	121.6	14.9
NO_2 (ppb)		35.3	12.3	9.0	25.9	33.7	43.5	82.0	17.6
SO_2 (ppb)		5.3	1.9	2.4	4.0	4.9	6.2	21.7	2.1
CO (0.1 ppm)		6.5	2.6	2.3	4.7	5.8	7.5	25.5	2.9
O_3 (ppb)		31.2	16.8	2.1	18.8	28.7	41.6	105.4	22.8
Meteorological variables									
Mean temperature (°C)		12.8	10.8	−14.6	3.6	14.4	22.8	31.8	
Barometric pressure (hPa)		1016.1	8.2	990.8	1009.6	1016.4	1022.7	1038.1	
Sunlight (h)		6.2	4.0	0	2.4	7.2	9.5	13.5	
Precipitation (mm)		16.0	117.2	0	0	0	0.5	3153.0	
Relative humidity (%)		60.2	14.9	19.4	49.2	60.2	70.7	99.4	

Abbreviations: ED, emergency department; SD, standard deviation; IQR, interquartile range; PM_{10}, particulate matter with an aerodynamic diameter of <10 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter of <2.5 μm; NO_2, nitrogen dioxide; SO_2, sulfur dioxide; CO, carbon monoxide; O_3, ozone.

Table 2
Total and subgroup (age and sex) counts of ED visits due to injury according to intentions and mechanisms in Seoul, South Korea, 2008–2016.

	Total	Traffic accident	Fall	Slip	Blunt object	Penetration	Machinery	Burn	Drowning	Poisoning	Suffocation, hanging
Total											
Unintentional	1,931,424	406,585	126,747	492,362	475,925	293,983	16,708	106,579	370	10,376	1789
	(100)	(21.1)	(6.6)	(25.5)	(24.6)	(15.2)	(0.9)	(5.5)	(0.0)	(0.5)	(0.1)
Self-harm	24,603	229	737	171	2623	8006	170	184	462	11,452	569
	(100)	(0.9)	(3.0)	(0.7)	(10.7)	(32.5)	(0.7)	(0.7)	(1.9)	(46.5)	(2.3)
Assault	93,828	98	252	2088	82,335	8594	92	125	4	48	192
	(100)	(0.1)	(0.3)	(2.2)	(87.8)	(9.2)	(0.1)	(0.1)	(0.0)	(0.1)	(0.2)
<15 years											
Unintentional	538,358	48,901	58,046	123,953	203,318	61,065	2009	37,904	132	2311	719
	(100)	(9.1)	(10.8)	(23.0)	(37.8)	(11.3)	(0.4)	(7.0)	(0.0)	(0.4)	(0.1)
Self-harm	490	16	33	27	100	148	2	14	2	146	2
	(100)	(3.3)	(6.7)	(5.5)	(20.4)	(30.2)	(0.4)	(2.9)	(0.4)	(29.8)	(0.4)
Assault	5358	9	15	41	4984	284	5	5	0	6	9
	(100)	(0.2)	(0.3)	(0.8)	(93.0)	(5.3)	(0.1)	(0.1)	(0.0)	(0.1)	(0.2)
15–64 years											
Unintentional	1,183,579	319,713	51,915	263,934	243,646	218,171	13,794	64,373	190	6942	901
	(100)	(27.0)	(4.4)	(22.3)	(20.6)	(18.4)	(1.2)	(5.4)	(0.0)	(0.6)	(0.1)
Self-harm	22,322	195	658	103	2469	7601	164	161	429	10,068	474
	(100)	(0.9)	(2.9)	(0.5)	(11.1)	(34.1)	(0.7)	(0.7)	(1.9)	(45.1)	(2.1)
Assault	84,931	86	213	1791	74,420	8000	83	116	4	41	177
	(100)	(0.1)	(0.3)	(2.1)	(87.6)	(9.4)	(0.1)	(0.1)	(0.0)	(0.0)	(0.2)
>64 years											
Unintentional	209,487	37,971	16,786	104,475	28,961	14,747	905	4302	48	1123	169
	(100)	(18.1)	(8.0)	(49.9)	(13.8)	(7.0)	(0.4)	(2.1)	(0.0)	(0.5)	(0.1)
Self-harm	1791	18	46	41	54	257	4	9	31	1238	93
	(100)	(1.0)	(2.6)	(2.3)	(3.0)	(14.3)	(0.2)	(0.5)	(1.7)	(69.1)	(5.2)
Assault	3539	3	24	256	2931	310	4	4	0	1	6
	(100)	(0.1)	(0.7)	(7.2)	(82.8)	(8.8)	(0.1)	(0.1)	(0.0)	(0.0)	(0.2)
Male											
Unintentional	1,127,746	251,101	76,946	255,378	300,947	174,289	12,009	50,843	233	5044	956
	(100)	(22.3)	(6.8)	(22.6)	(26.7)	(15.5)	(1.1)	(4.5)	(0.0)	(0.4)	(0.1)
Self-harm	11,167	140	355	96	2161	3926	77	123	289	3695	305
	(100)	(1.3)	(3.2)	(0.9)	(19.4)	(35.2)	(0.7)	(1.1)	(2.6)	(33.1)	(2.7)
Assault	67,260	71	151	1157	58,998	6588	63	80	2	25	125
	(100)	(0.1)	(0.2)	(1.7)	(87.7)	(9.8)	(0.1)	(0.1)	(0.0)	(0.0)	(0.2)
Female											
Unintentional	803,678	155,484	49,801	236,984	174,978	119,694	4699	55,736	137	5332	833
	(100)	(19.3)	(6.2)	(29.5)	(21.8)	(14.9)	(0.6)	(6.9)	(0.0)	(0.7)	(0.1)
Self-harm	13,436	89	382	75	462	4080	93	61	173	7757	264
	(100)	(0.7)	(2.8)	(0.6)	(3.4)	(30.4)	(0.7)	(0.5)	(1.3)	(57.7)	(2.0)
Assault	26,568	27	101	931	23,337	2006	29	45	2	23	67
	(100)	(0.1)	(0.4)	(3.5)	(87.8)	(7.6)	(0.1)	(0.2)	(0.0)	(0.1)	(0.3)

Abbreviation: ED, emergency department.

% (1.17–2.55 %), respectively. Unintentional injuries showed identical results to the total injuries because they accounted for the majority of total injuries (94 %). For intentional injuries, short-term exposure to all air pollutants was not associated with a risk of self-harm injuries, whereas exposure to $PM_{2.5}$ (1.08 % [0.03–2.14 %] at lag 0), NO_2 (2.36 % [0.78–3.96 %] at lag 1), SO_2 (2.09 % [0.70–3.50 %] at lag 0), and CO (2.37 % [1.11–3.64 %] at lag 0) were significantly associated with the increased risk of assault injuries.

Fig. 2 shows the association between air pollution exposure and total injuries for the subgroups according to the injury mechanisms. The risks of injury caused by falling (all air pollutants), blunt object use (all air pollutants), penetration (PM_{10}, $PM_{2.5}$, SO_2, and O_3), and burns (PM_{10}, $PM_{2.5}$, SO_2, and CO) were associated with most air pollutants, whereas injuries caused by traffic accidents (O_3), machinery (CO), and slipping (NO_2 and O_3) were associated with specific air pollutants, except for poisoning injuries that showed no association. The mechanism-specific air pollution-injury associations for unintentional injuries were generally consistent with those for total injuries (Fig. S1). No significant association was found in any injury mechanism for self-harm injuries except for penetration (NO_2 at lag 3), whereas exposure to NO_2, SO_2, and CO was associated with an increased risk of assault injuries due to blunt objects and penetration (Fig. S2).

Fig. 3 shows the association between air pollution exposure and ED visits for total injuries according to age and sex subgroups. For people aged <15 years, the IQR increment of all air pollutants on the day of an ED visit (lag 0) significantly increased the risk of injuries (0.98 % [0.47–1.48 %] for PM_{10}, 1.35 % [0.77–1.94 %] for $PM_{2.5}$, 1.81 % [0.96–2.67 %] for NO_2, 2.04 % [1.28–2.81 %] for SO_2, 0.86 % [0.16–1.55 %] for CO, and 4.67 % [3.65–5.69 %] for O_3). For those aged 15–64 years, NO_2 (0.63 % [0.01–1.27 %] at lag 1), SO_2 (0.82 % [0.25–1.39 %] at lag 0) and O_3 (0.77 % [0.02–1.53 %] at lag 0) were significantly associated with the risk of injuries, whereas no association was observed among those aged >64 years. In sex-specific analyses, immediate exposure (lag 0) to all air pollutants significantly increased the risk of injuries among males (0.50 % [0.13–0.87 %] for PM_{10}, 0.73 % [0.31–1.15 %] for $PM_{2.5}$, 1.20 % [0.59–1.82 %] for NO_2, 1.64 % [1.08–2.19 %] for SO_2, 0.85 % [0.35–1.35 %] for CO, and 2.15 % [1.41–2.89 %] for O_3), whereas only O_3 exposure was associated with the risk of injuries in females (1.44 % [0.58–2.30 %]).

Sensitivity analysis that applied moving-average lag structures revealed that cumulative air pollution exposure was strongly associated with assault injuries, whereas immediate exposure (lag 0) mostly affected total and unintentional injuries (Fig. S3). The goodness-of-fit based on the QAIC values for further co-pollutant models are presented in Tables S2–S4. In the co-pollutant models for the total injuries and subgroups by the intention behind the injuries, the SO_2 and O_3 effects remained significant, whereas those for $PM_{2.5}$, NO_2, and CO became weak or non-significant for the total and unintentional injuries.

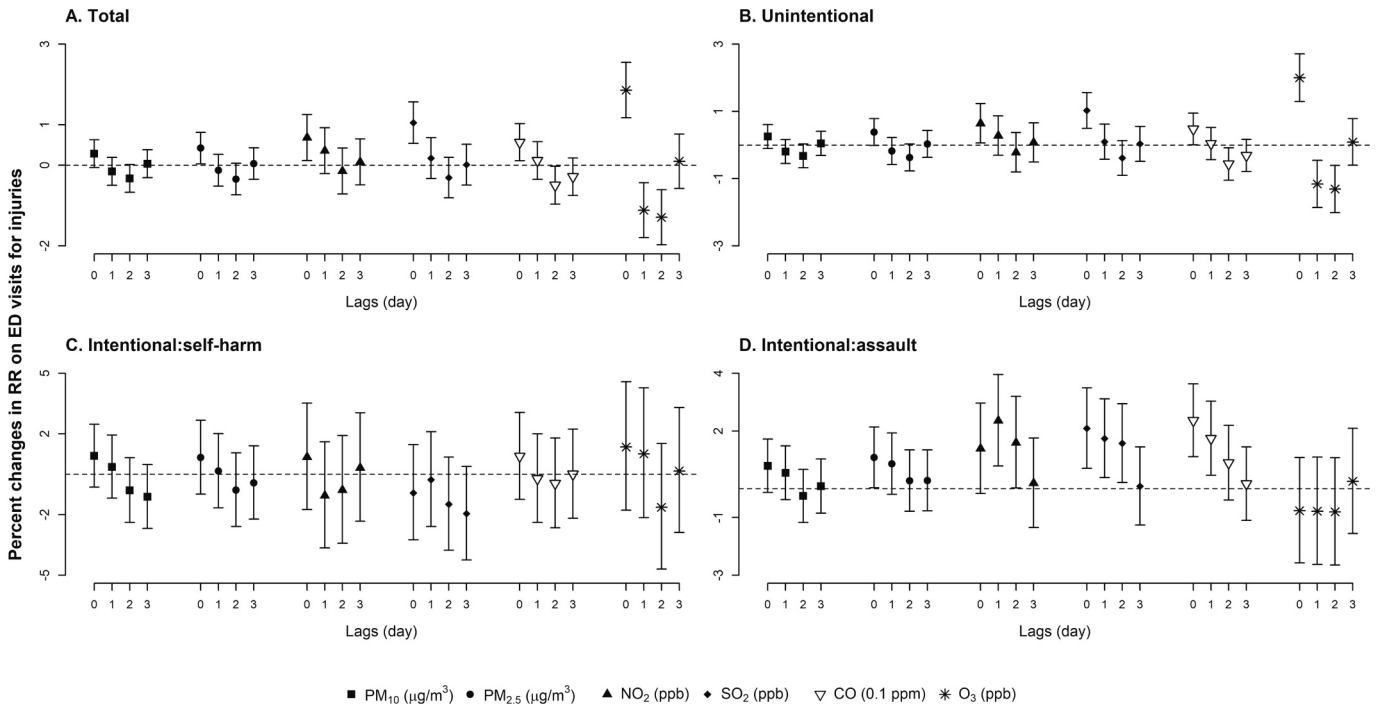

Fig. 1. Percent changes in relative risk of emergency department visits for total injuries and intention-specific subgroups per IQR increase of air pollutants over lag 0 to lag 3. RR, relative risk; ED, emergency visit; IQR, interquartile range.

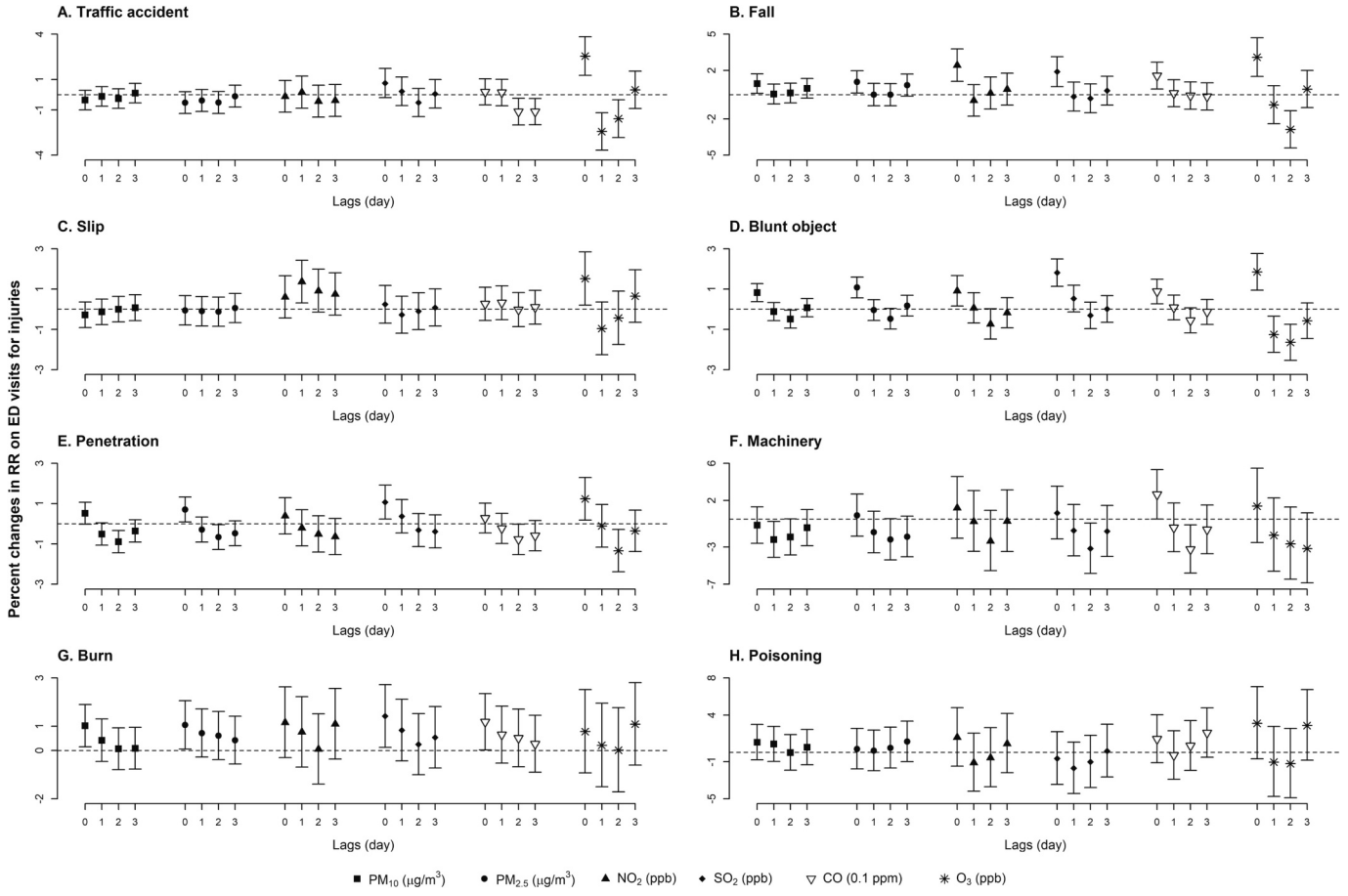

Fig. 2. Percent changes in relative risk of emergency department visits for mechanism-specific injuries per IQR increase of air pollutants over lag 0 to lag 3. RR, relative risk; ED, emergency visit; IQR, interquartile range.

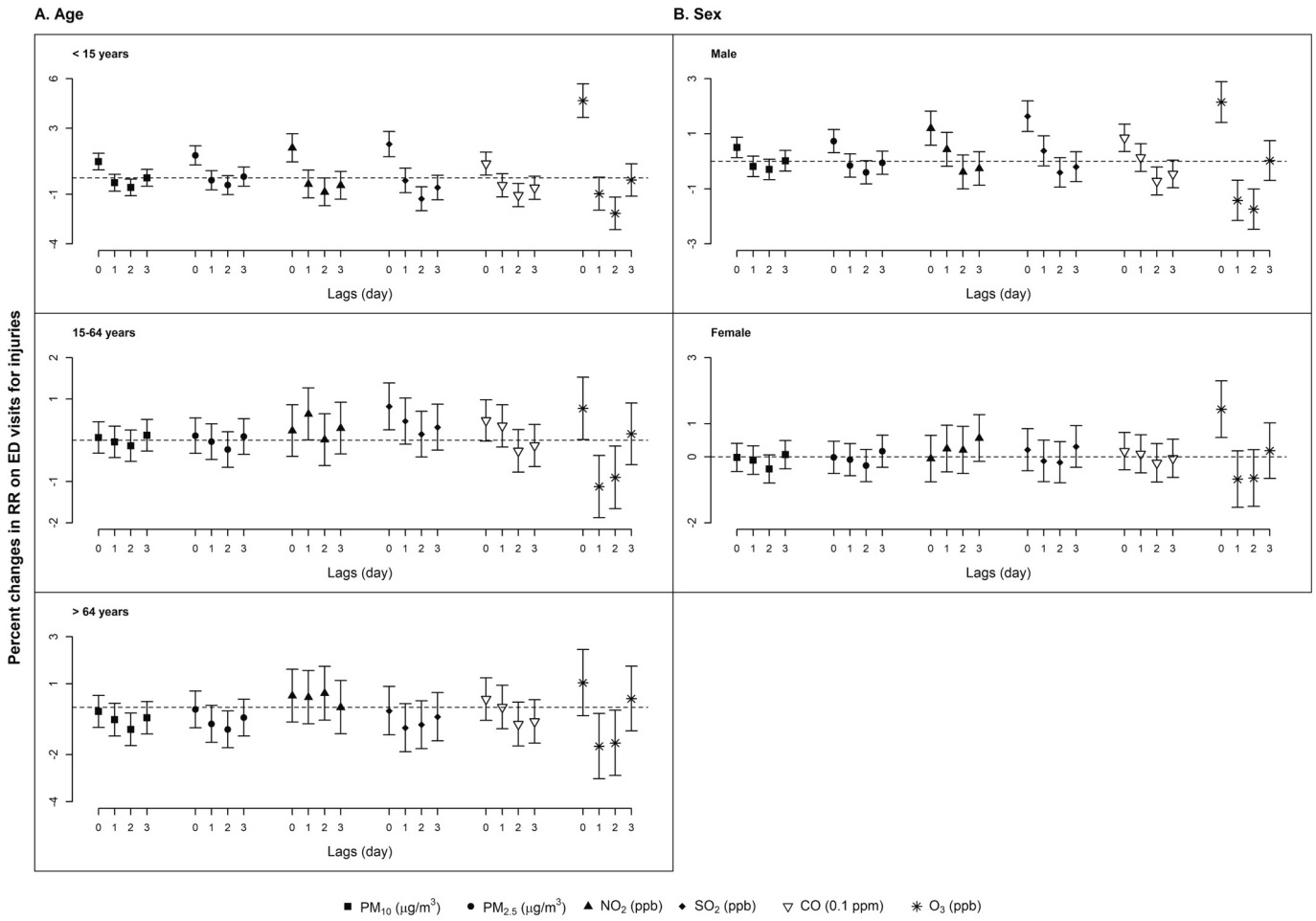

Fig. 3. Percent changes in relative risk of emergency department visits for age- and sex-specific injuries per IQR increase of air pollutants over lag 0 to lag 3. RR, relative risk; ED, emergency visit; IQR, interquartile range.

For assault injuries, air pollution effects generally remained significant, except for $PM_{2.5}$ (Table S5). In co-pollutant models for subgroups based on the mechanism of injury, most air pollution-injury associations remained significant (blunt object use [PM_{10}, $PM_{2.5}$, SO_2, and O_3], falling [NO_2, SO_2, and O_3], penetration [$PM_{2.5}$, SO_2, and O_3], slipping [NO_2 and O_3], traffic accidents [O_3], and machinery [CO]) (Table S6). The co-pollutant models for subgroups of age and sex generally showed similar results to those of the primary findings, except that the CO effect for people aged <15 years and that of PM_{10}, $PM_{2.5}$, and CO for males became non-significant (Table S7). However, the results should be interpreted carefully because of the high intercorrelations among the pollutants. Applying natural cubic splines for all meteorological variables with df 4–6 showed results consistent with those of the primary models (Tables S8–S10). Changing the lag structures of all meteorological variables also produced similar results to those of the main findings (Tables S11–S13). Lastly, immediate effects (lag 0 or lag1) of air pollution were consistently observed when extended lag effects up to 7 days were analyzed (Fig. S4).

4. Discussion

In this time-stratified case-crossover study, where 2,049,855 injured patients in the capital city of South Korea were analyzed, we found that short-term exposure to ambient air pollutants significantly increased the risk of ED visits for total injuries due to external causes, and that the associations differed by the intention and mechanism of injury. The risk of unintentional and intentional assault injuries were significantly associated with immediate air pollution exposure (lag 0 or lag 1), whereas self-harm injuries had no association with air pollution exposure. In the analyses of the mechanism of injury, the risks of injuries caused by falling, blunt object use, penetration, and burns were associated with most air pollutants, whereas those caused by traffic accidents, machinery, and slipping were associated with a specific air pollutant, except for poisoning injuries that had no association. Moreover, the associations were stronger in injured patients aged <15 years and males than in their counterparts. These primary findings were robust in various sensitivity analyses.

To the best of our knowledge, this is the first study that provides evidence of an association between short-term air pollution exposure and injuries due to external causes while comprehensively considering the intention and mechanism of injury. Previous studies on the relationship between air pollution and injuries have mainly focused on intentional injuries, including suicide attempts (Szyszkowicz et al., 2010), non-suicidal self-injuries (Liu et al., 2018), and violent behavior (Berman et al., 2019; Burkhardt et al., 2020) or a specific mechanism of injury, such as road traffic (Chan et al., 2022) and fall-related injuries (Guo et al., 2018). A recent study examined the association between urban air pollution and ED visits for total injuries in Edmonton and Toronto, Canada, and found a significant positive association in line with our findings (Szyszkowicz et al., 2022). However, they did not investigate the intention- or mechanism-specific associations behind the injuries.

In the present study, total and unintentional injuries showed identical patterns in association with air pollution exposure because unintentional injuries accounted for a large proportion of the total injuries (94 %). Immediate exposure (lag 0) to gaseous pollutants (NO_2, SO_2, CO,

and O_3) significantly increased the risk of unintentional injuries, and the effect of O_3 was predominant. In the mechanism-specific analyses of unintentional injuries, most mechanisms, except for burns and poisoning, showed a significant relationship with immediate exposure to several or specific pollutants, even in co-pollutant models (blunt object use [PM_{10}, $PM_{2.5}$, SO_2, and O_3], falling [NO_2, SO_2, and O_3], penetration [$PM_{2.5}$, SO_2, and O_3], slipping [NO_2 and O_3], traffic accidents [O_3], and machinery [CO]). Notably, the effect of O_3 was also the strongest among most of the injury mechanisms. Ground-level O_3 is a highly reactive gas formed by photochemical reactions, and its harmful effects on the neurological and respiratory systems have been reported. Epidemiological studies have observed neurological manifestations (fatigue, lethargy, and headaches) in urban inhabitants exposed to high O_3 concentrations (Dales et al., 2009; Paz, 1997). Experimental studies in animals have found that exposure to a high O_3 dose induces decreased motor activity (Dorado-Martínez et al., 2001; Tepper et al., 1982), memory/cognitive impairment (Avila-Costa et al., 1999), impaired neurobehavioral performance (Sorace et al., 2001), and sleep-wake disorganization (Paz and Bazan-Perkins, 1992). Moreover, oxidative stress and neuroinflammation after O_3 exposure are possible underlying mechanisms responsible for these neurological effects (González-Guevara et al., 2014; Rivas-Arancibia et al., 2003). Thus, other pollutants that induce oxidative stress and neuroinflammation may affect the central nervous system via similar mechanisms (Gokirmak et al., 2003; Wang et al., 2017). A possible reason why pollutants exhibiting a significant effect differ depending on the mechanism of injury might be that exposure levels are different under varying injury circumstances. Neurological impairments caused by air pollution exposure may lead to unintentional injuries via various mechanisms, such as distractibility, impaired judgment, or executive dysfunction (Day et al., 2012; Kass et al., 2010; Pelicioni et al., 2021).

Subgroup-specific analyses by age and sex identified subpopulations that were more vulnerable to injuries related to ambient air pollution exposure. In the age-specific analyses, injured patients aged <15 years showed the strongest significant association with exposure to all six air pollutants, whereas those aged 15–64 years showed significant associations with gaseous pollutants (NO_2, SO_2, CO, and O_3). Older patients (>64 years) had no association with air pollution exposure. This difference may have resulted from different age-specific behavior patterns. Considering that the association was strongest for injured patients aged <15 years, ambient air pollution exposure is likely to mostly affect non-occupational unintentional injuries (Tracy et al., 2013). Short-term air pollution exposure has been associated with attention deficits (Compa et al., 2023) and reduced fundamental cognitive abilities, such as working memory and conflict attentional networks (Medrano et al., 2022). These neurological impediments caused by ambient air pollutants may result in unintentional injuries in those aged <15 years via impaired attention and hyperactive or impulsive behavior (Compa et al., 2023). Given that patients aged 15–64 years are primarily economically active, occupational exposure may account for a large proportion of total air pollution exposure and may differ depending on the occupation (Downward et al., 2018; Vinnikov et al., 2020). Thus, our findings, which did not consider occupational exposure, might reflect only air pollution exposure during commuting or outdoor activities and should be interpreted with these limitations in mind. On the contrary, older patients tend to stay indoors; therefore, they are likely to be less exposed to occupational/non-occupational ambient air pollutants (Abdel-Salam, 2022), which might explain the lack of association. In the sex-specific analyses, short-term exposure to all air pollutants was significantly associated with the risk of injury in males, whereas exposure to only O_3 was significantly associated with that in females. In South Korea, males participate in economic activities more frequently, tend to stay outdoors, and are more physically active than females, and these different behavioral patterns could contribute to different exposure levels of ambient air pollution between them (Kim et al., 2021; Matz et al., 2014).

No association was found between self-harm injuries and short-term air pollution exposure. This result is contrary to those of previous studies that found a significant association between suicide and air pollution exposure (Bakian et al., 2015; Lee et al., 2018; Ng et al., 2016). One difference between our study and previous ones is that the study population in the previous studies consisted of completed suicide cases, whereas our study dealt with suicide attempters who had visited the ED because of related injuries. It is possible that suicide completers and attempters have different characteristics; therefore, the effects of air pollution exposure differ. Suicide methods have been reported as a key difference: suicide attempters generally use non-lethal methods, whereas suicide completers mostly use lethal methods (Lim et al., 2014; Liu et al., 2016). In line with previous findings, 79 % of the mechanisms of self-harm injury cases in our study were poisoning and penetration (stabbing), which are non-lethal methods (Lim et al., 2014). Moreover, it has been found that air pollution effects were only significant or stronger in violent suicide cases than in non-violent ones (Bakian et al., 2015; Lee et al., 2018). A plausible mechanism is that air pollution provokes suicidal behavior through dysfunction of the serotonin system (Yokota et al., 2013) which is typically only found in individuals exhibiting violent suicidal behavior (Courtet et al., 2003; Spreux-Varoquaux et al., 2001). Another possible reason for the non-significant association might be the insufficient sample size of self-harm injuries, thereby increasing the CI values.

We found that exposure to several pollutants (NO_2, SO_2, and CO) was associated with an increased risk of assault injuries. This finding is consistent with that of a previous study that observed a significant association between CO exposure and crime incidents caused by male and black offenders across 109 cities in the USA (Cruz et al., 2022). Another study analyzing 86.1 million people in the USA also found that acute air pollution exposure significantly increased the risk of violent behavior (Berman et al., 2019). Although the exact causal mechanism for impulsive and aggressive behavior has not been proven, aggressiveness and territoriality associated with air pollution exposure have been demonstrated through experiments with canines and mice (Calderón-Garcidueñas et al., 2003; Yokota et al., 2013). Acutely absorbed air pollutants can induce cellular oxidative stress and systemic inflammation that are related to the impairment of cognitive and behavioral systems, which may lead to aggressive behavior (Cunningham et al., 2009). Additionally, psychological traits (i.e., perceived control and impulsiveness) that can be stimulated by air pollution exposure may contribute to impaired impulsive behavior (Burkhardt et al., 2019; Lu et al., 2018).

Although we found positive associations of ED visits for injuries with both gaseous air pollutants and particles (PM_{10} and $PM_{2.5}$), the effects of particles were smaller than those of gaseous pollutants and the affected subgroups by injury mechanisms and sex/age were different to a certain extent. Unlike gaseous air pollutants, particles are a heterogeneous mixture of organic compounds, ions, metals, or soil in the air from various emission sources (Yousuf et al., 2022). Differences in constituents and sources of particles among study areas have been suggested as a possible factor for different estimates (Yang et al., 2020). In our study, certain constituents or sources of ambient particles may have been associated with certain injury subgroups according to characteristics of injured patients, such as occupation (workplaces) and behavior patterns. Therefore, we anticipate that when a certain component of particles is associated with injuries, estimating the effect using total particles could underestimate the true association (Li et al., 2023). Future studies using PM component data are warranted to identify more relevant components associated with injuries.

This study has several limitations. First, a bias due to exposure measurement error might have occurred because the air pollution exposure measurements for injured patients were estimated based on concentrations measured at fixed monitoring sites. However, the exposure measurement error tends to cause a bias toward the null hypothesis, which underestimates the true association (Armstrong, 1998); therefore, our results were likely not exaggerated. Moreover, we constructed

aggregated city-level exposure estimates because information on the patients' residential addresses was not provided owing to confidentiality issues. Highly aggregated exposure data tend to induce a Berkson error; however, this causes a large variation but no bias in the effect estimates (Heid et al., 2004). Second, the selection of injured patients based on medical records may have caused a misclassification bias. However, this misclassification was non-differential in our study because it occurred equally among the cases and controls. Non-differential misclassification bias tends to underestimate true associations (Copeland et al., 1977). Third, we adjusted for same covariates for subgroup analyses by mechanisms of injuries, although potential confounding factors would be different by the injury mechanisms. For example, occupation (hazardous workplaces with risk of exposure to blunt object or penetration) or morbid states (poisoning/hanging related to depression) could be the confounding factors that differ by subgroups. However, our time-stratified case-crossover study design was automatically adjusted for time-invariant (sex and genetic predisposition) confounding factors through the design itself. Furthermore, time-dependent factors that vary slowly, such as occupation, life styles, and morbid states, can be adjusted for by selecting control periods close to the case (ED visit) day. We selected 3 or 4 control days that were matched to the day of the week, month, and year of the case day. Therefore, we believe that potential confounding factors that differ by causes of injuries were adjusted for through the study design itself. To control for short-term varying confounding factors such as weather variables (e.g., icy roads for slipping), we included daily-varying confounding factors (temperature, relative humidity, barometric pressure, sunlight hours, precipitation, influenza, and holidays) in the regression model. Nevertheless, there might still be short-term varying potential confounding factors that we could not consider. Fourth, we could not stratify injuries according to occupational and non-occupational injuries because of limited data availability. Since occupation is an important factor affecting air pollution exposure and most studies on unintentional injuries have focused on occupational injuries, future studies need to consider the occupation of injured patients. Finally, our findings from a single city may not be generalizable to other cities or countries with different environmental conditions or industry/infrastructure.

Despite these limitations, this study has several strengths. To the best of our knowledge, this is the first study to evaluate the association between short-term exposure to ambient air pollutants and the risk of injuries due to external causes by considering the intention and mechanism behind these injuries, and to determine more susceptible subpopulations. The employment of a case-crossover design through a conditional Poisson regression enabled comprehensive adjustment for time-invariant and slowly varying confounding factors as well as over-dispersion and autocorrelation in aggregated time counts. Our findings add to the limited evidence on the association between short-term air pollution exposure and the risk of injuries and could contribute to the establishment of appropriate policies and targeted interventions related to injury prevention and air pollution reduction by providing information about more relevant intentions, mechanisms, and subgroups of injuries.

5. Conclusions

The present study found a significant association between immediate air pollution exposure and the risk of unintentional and assault injuries, injuries caused by most mechanisms except for burns and poisoning, and among injured patients aged < 15 years and males. This finding is an essential addition to the limited literature on the potential role of air pollutants in the risk of injuries due to external causes, which has received little attention despite the increasing burden of injuries globally. Further investigations are warranted to verify whether our findings can be replicated in other regions and populations, and to elucidate the underlying biological mechanisms involved.

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CRediT authorship contribution statement

Jiyun Jung: Formal analysis, Funding acquisition, Visualization, Writing – original draft. **Gyeongchan Kim:** Data curation, Methodology. **Sun-Woo Kang:** Data curation, Methodology. **Subin Jeong:** Data curation, Methodology. **Yoonjung Kang:** Data curation, Methodology. **Jun-Young Lee:** Validation, Writing – review & editing. **Woojae Myung:** Validation, Writing – review & editing. **Ho Kim:** Methodology, Writing – review & editing. **Hyewon Lee:** Conceptualization, Funding acquisition, Methodology, Supervision, Writing – original draft.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The authors do not have permission to share data.

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None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scitotenv.2023.169202.

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