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Neighborhood violence and socioeconomic deprivation influence associations between acute air pollution and temperature on childhood asthma in New York city

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ABSTRACT

Ambient air pollution, temperature, and social stressor exposures are linked with asthma risk, with potential synergistic effects. We examined associations for acute pollution and temperature exposures, with modification by neighborhood violent crime and socioeconomic deprivation, on asthma morbidity among children aged 5–17 years year-round in New York City. Using conditional logistic regression in a time-stratified, case-crossover design, we quantified percent excess risk of asthma event per 10-unit increase in daily, residence-specific exposures to $PM_{2.5}$, NO_2, SO_2, O_3, and minimum daily temperature (Tmin). Data on 145,834 asthma cases presenting to NYC emergency departments from 2005 to 2011 were obtained from the New York Statewide Planning and Research Cooperative System (SPARCS). Residence- and day-specific spatiotemporal exposures were assigned using the NYC Community Air Survey (NYCCAS) spatial data and daily EPA pollution and NOAA weather data. Point-level NYPD violent crime data for 2009 (study midpoint) was aggregated, and Socioeconomic Deprivation Index (SDI) scores assigned, by census tract. Separate models were fit for each pollutant or temperature exposure for lag days 0–6, controlling for co-exposures and humidity, and mutually-adjusted interactions (modification) by quintile of violent crime and SDI were assessed. We observed stronger main effects for $PM_{2.5}$ and SO_2 in the cold season on lag day 1 [4.90% (95% CI: 3.77–6.04) and 8.57% (5.99–11.21), respectively]; Tmin in the cold season on lag day 0 [2.26% (1.25–3.28)]; and NO_2 and O_3 in the warm season on lag days 1 [7.86% (6.66–9.07)] and 2 [4.75% (3.53–5.97)], respectively. Violence and SDI modified the main effects in a non-linear manner; contrary to hypotheses, we found stronger associations in lower-violence and -deprivation quintiles. At very high stressor exposures, although asthma exacerbations were highly prevalent, pollution effects were less apparent—suggesting potential saturation effects in socio-environmental synergism.

1. Introduction

Primary air pollutants from fossil-fuel combustion and climate-linked secondary air pollutants like ground-level ozone and extreme ambient temperatures are among the leading environmental causes of population health disparities worldwide that continue to worsen under the rapidly changing global climate (Keswani et al., 2022; Romanello et al., 2022). Compared to adults, children are particularly vulnerable to the adverse health effects of air pollution and temperature due to their underdeveloped natural defense mechanisms like the blood-brain barrier and immune system, greater skin surface area relative to body mass, and lesser ability to regulate core body temperature (Arpin et al., 2021; Perera and Nadeau, 2022).

Asthma is the most prevalent chronic childhood illness in the United States (Johnson et al., 2021; Pate, 2021; Zhang and Zheng, 2022), with children of color, particularly Black and Hispanic children, and children belonging to low socio-economic position (SEP) communities affected disproportionately (Johnson et al., 2021; Keet et al., 2017; Pate, 2021; Zanobetti et al., 2022). Additionally, areas with higher levels of urbanization experience higher morbidity rates for childhood asthma,

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potentially due to higher levels of indoor asthma triggers, traffic density, and social stress (Keet et al., 2017; P. W. Sullivan et al., 2019). Exposures to ambient air pollution and temperature have been linked, both independently (Cong et al., 2017; Han et al., 2023; Tiotiu et al., 2020; Uibel et al., 2022; Zheng et al., 2015, 2021) and synergistically (Anenberg et al., 2020; Grigorieva and Lukyanets, 2021), to childhood asthma incidence and acute exacerbations of existing asthma that can result in emergency department (ED) visits and hospitalizations. Chronic stress from community-level social stressors such as violence, poverty, and racial residential segregation has also been associated with increased asthma morbidity (Fujiwara, 2008; Landeo-Gutierrez et al., 2020; K. Sullivan and Thakur, 2020; Yonas et al., 2012). Lower-SEP children are generally exposed to higher levels of air pollution (Collins and Grineski, 2022; Jbaily et al., 2022; J. J. Liu et al., 2021) and temperature (Benz and Burney, 2021; Gronlund, 2014; Renteria et al., 2022), and additionally, may bear a greater social stressor burden that might increase their susceptibility to developing adverse health outcomes, like asthma, in greater proportions than in socio-economically advantaged communities (Clougherty et al., 2014, 2022; Landeo-Gutierrez et al., 2020; K. Sullivan and Thakur, 2020; Yonas et al., 2012). Evidence suggests that such SEP-related susceptibility to air pollution and temperature health effects may be attributable to chronic stress resulting from exposures to various social stressors (Clougherty et al., 2014, 2022). Chronic stress negatively influences the immune, endocrine, and metabolic functions (McEwen, 2012, 2017; McEwen and Tucker, 2011), rendering the bodily systems vulnerable to environmental insults (Peters et al., 2021). As air pollution and temperature exposures, as well as social stressors, are usually spatially patterned by SEP, disentangling their relative contributions and quantifying their interactions could enhance our capacity to identify and characterize population groups vulnerable to their health effects (Clougherty et al., 2014, 2022). This in turn can effectively and efficiently direct research, policies, and interventions aimed at reducing socio-economic disparities in asthma morbidity (Clougherty et al., 2014, 2022).

Prior epidemiologic studies quantifying the combined effects of environmental exposures and social stressors like violence (Chiu et al., 2014; Clougherty et al., 2007; Sheffield et al., 2019; J. L. Shmool et al., 2014), SEP/material deprivation (Gleason et al., 2014; M. Lin et al., 2004; S. Lin et al., 2008; Neidell, 2004; O'Lenick et al., 2017; Rosenlund et al., 2009; Yang et al., 2003; Yap et al., 2013), and family/parental stress (Chen et al., 2008; Deng et al., 2018; Islam et al., 2011; Ranci et al., 2017; Shankardass et al., 2009) on childhood asthma generally suggest synergistic associations. However, most of these studies have exclusively focused on air pollution exposures and considered interactions between single exposure-stressor pairs (Appleton et al., 2016; Clougherty et al., 2014; Clougherty and Kubzansky, 2009). In the current study, we examined variations in childhood asthma morbidity in relation to acute exposures to multiple ambient air pollutants and ambient temperature within New York City (NYC). We also quantified interactions between environmental exposures (air pollution and temperature) and two critical urban social stressors in NYC—neighborhood violent crime and socioeconomic deprivation (J. L. Shmool et al., 2014; J. L. C. Shmool et al., 2015). We did so by leveraging the New York Statewide Planning and Research Cooperative System (SPARCS) ED visit data from 2005 to 2011 for NYC and assigning fine-scale spatiotemporal exposure estimates to each presenting asthma case during this period. We hypothesized that elevated levels of air pollution and temperature exposures would be positively associated with increased ED visit risk for childhood asthma and that the associations will be stronger among children belonging to higher violent crime and socioeconomically deprived communities.

2. Methods

2.1. Study population and outcome

We included all children, aged 5–17 years, who presented at NYC emergency departments between January 1, 2005, and December 31, 2011, and received a primary diagnosis of asthma corresponding to an International Classification of Diseases, Ninth Revision (ICD-9) code of 493 and the specific diagnoses therein. Data on all in-patient (admitted) and out-patient unscheduled asthma ED visits during the study period (n = 145,838) were obtained from the New York Statewide Planning and Research Cooperative System (SPARCS) (New York State Department of Health, 2022). SPARCS is a comprehensive data reporting system for New York State that collects patient-level information on demographic characteristics, diagnoses and treatments, services availed, and medical expenses for each encounter. Information on primary diagnostic code, admission type (inpatient or outpatient), date of admission, age at admission, sex, race/ethnicity, and location (latitude and longitude) of residence at admission were extracted for each asthma case from SPARCS. To focus on acute asthma events, we limited our analyses to ED visits marked 'emergent' or 'urgent', indicating events requiring immediate medical attention.

2.2. Study design

A time-stratified case-crossover study design was used (Carracedo et al., 2010; Maclure, 1991). Asthma cases who presented to the emergency department (case day events) were matched to themselves by the day of the week, calendar month, and year when they were not admitted, yielding three or four controls (control day events) per case. For instance, if an asthma ED visit occurred on the first Tuesday of January 2005, exposures on the 2nd, 3rd, and 4th Tuesdays of the same month would serve as the controls. As individual cases are self-matched, serving as their own control, the case-crossover design inherently adjusts for confounding by factors that do not vary or slowly vary with time (in this case, within one month) such as age, sex, race/ethnicity, body mass index, exposure to environmental tobacco smoke, etc. (Mittleman and Mostofsky, 2014). The design also eliminates measured or unmeasured confounding bias by any long-term trends (Mittleman and Mostofsky, 2014).

2.3. Environmental exposures

Daily, residence-level exposures to fine particulate matter with aerodynamic diameter $\leq$2.5 μm ($PM_{2.5}$), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and minimum temperature (Tmin)—henceforth, collectively referred to as environmental exposures—were assigned to each asthma case by integrating spatially- and temporally-refined exposure data.

We derived citywide spatial surfaces for each environmental exposure from the NYC Community Air Survey (NYCCAS). The overall NYCCAS sampling design and the modeling methods are detailed in Matte et al. (2013) and Clougherty et al. (2013), respectively. Briefly, air quality samples were randomly collected across 150 sites across NYC over a two-year period. While $PM_{2.5}$ and NO_2 were monitored year-round, O_3 was monitored only during summer and SO_2 only during winter when concentrations of each are elevated. Overnight temperatures (3:00 to 5:00 a.m.; Tmin) were preferred over daily maximum temperature or average temperature as they displayed greater spatial variation across seasons and were potentially less influenced by localized intermittent shading or sunlight-related microclimate effects. Intra-urban spatial variation in each exposure was modeled using land use regression (LUR) methods, and final spatial surfaces (maps) were derived for a 100-m x 100-m grid using kriging with external drift (KED), which combines LUR model output with spatial smoothing (Clougherty et al., 2013). These final spatial surfaces were used to create exposure

estimates for all individual asthma cases, using the mean concentration at 100-m NYCCAS grid centroids within 300 m of each case's residence.

Next, to construct a citywide time series for air pollution exposure estimates, we retrieved hourly data from the U.S. Environmental Protection Agency (EPA) Air Quality System (AQS) regulatory monitoring stations in NYC, for the period 2005 to 2011. We calculated daily averages at each monitor, which were averaged into one meantime trend for the city, as done by Sheffield et al. (2015). For each pollutant, the time series was computed on an annual basis. Time series data on daily temperature, relative humidity, and dew point temperature were collected from the four meteorological stations in the NYC area (JFK International Airport, LaGuardia International Airport, Central Park, and Newark International Airport), retrieved from the National Oceanic and Atmospheric Administration (NOAA) National Climatic Data Center.

Finally, we combined the temporal data with NYCCAS spatial surfaces to create year-round residence- and day-specific (i.e., spatio-temporal) exposure estimates—as previously in Ross et al. (2013) and Shmool et al. (2015a,b). We estimated and assigned exposures on the day of ED visit by multiplying the daily average concentration at AQS sites by the ratio of: (near-residence (300-m) concentration/mean NYCCAS concentration at AQS monitoring sites. Estimates were created separately for each pollutant, and for 7 days (lags 0–6) prior to each asthma case event., Spatio-temporal temperature estimates were created similarly, using NOAA weather monitoring sites. Estimates for relative humidity were also derived from NOAA weather data, but were strictly temporal, as they were not spatially measured and modeled under NYCCAS.

2.4. Social stressors

We assigned census tract-level spatial estimates of violent crime rate, Socioeconomic Deprivation Index (SDI) score, and racial composition indicators to each asthma case based on their residential location within a particular tract. There are 2167 census tracts in NYC per American Community Survey (ACS) year 2010 census boundaries (US Census Bureau, 2010). We excluded 63 smaller tracts (and the asthma events within) that had a residential population of fewer than 200 people, leaving 2104 final tracts.

Point-level, date- and time-stamped data on all violent offenses (murder and non-negligent manslaughter, aggravated assault, robbery) within NYC from 2006 to 2017 were obtained from NYC Police Department (New York City Police Department, 2018). Since rape crimes are not geocoded, they were not included. For compatibility with other studies, we followed the Federal Bureau of Investigation's (FBI's) Uniform Crime Reporting definitions to code crime (Federal Bureau of Investigation, 2022). Crime rates were calculated using the 2009 data, which corresponds to the midpoint of our study period. We confirmed that spatial patterns in crime are remarkably consistent in NYC, with census tract annual-average crime rates correlating at r (Spearman correlations) > 0.90 across all data years. Crimes were spatially joined to census tracts, summed to obtain counts per tract, and rates per 10,000 population were calculated using the tract-level residential population, obtained from the ACS 2007–2011 five-year estimates (US Census Bureau, 2010) that covered most of our study period.

SDI is a citywide index that was designed to capture relative material deprivation across NYC communities. It was developed using a spatially stratified principal components analysis with 25 indicators representing multiple dimensions of SEP (e.g., income, poverty, education, employment/occupation, housing, language, etc.) from the ACS 2007–2011 five-year estimates at the census tract-level. The process of SDI development is described in detail in Clougherty et al. (2021), Humphrey et al. (2019), and Shmool et al. (2015a,b). SDI was operationalized as an interquartile range (IQR)-standardized score, with higher scores indicating greater tract-level socioeconomic deprivation.

Racial composition was characterized from ACS 2007–2011 five-year estimates of the tract-level proportion of the Hispanic and non-Hispanic Black populations. Estimates on proportion of non-Hispanic other people of color (including Asians, American Indian and Alaska Native, and Native Hawaiian and Other Pacific Islander) were either small or not available.

2.5. Statistical analyses

We applied a conditional logistic regression (Cox proportional hazards) in the time-stratified case-crossover design, with the risk of ED visit for acute asthma (i.e., the case-control indicator) as the outcome, the daily estimates of the air pollutants and temperature as the environmental exposures, and tract-level estimates of violent crime rate and SDI score as potential effect modifiers. We quantified the associations as percent excess risk of asthma ED visit per 10-unit increment in each environmental exposure, across lag days 0–6. As air pollution and temperature exposures might vary in concentrations and have different effects in warm and cold seasons (Anenberg et al., 2020; Bergmann et al., 2020), we stratified all our models by warm season (April to September) and cold season (October to December and January to March). For $PM_{2.5}$ and NO_2, as year-round spatial surfaces were estimated under NYCCAS, both warm and cold season effects were considered. Only cold season effects were considered for SO_2 and warm season effects for O_3, as SO_2 was measured exclusively in the winter and O_3 in the summer under NYCCAS.

First, single-exposure distributed lag models (Schwartz, 2000) were constructed, adjusting only for case-day estimates of relative humidity fit as natural cubic spline with 3 degrees of freedom (df) (Perperoglou et al., 2019). Main exposures of interest were fit as continuous terms. Then, the single-exposure models were extended to include multiple exposures. Treated as co-exposures, the air pollutants were fit using penalized splines of their case-day concentration estimates, and temperature was fit as a natural cubic spline with 3 df. Models for NO_2, SO_2, and O_3 were adjusted only for $PM_{2.5}$ and Tmin. $PM_{2.5}$ models were adjusted only for O_3 and Tmin. Tmin models were only adjusted for $PM_{2.5}$ and NO_2. Prior to adjusting for any co-exposures, we examined relationships among the exposures, and developed models adjusting solely for temperature and relative humidity. We examined multicollinearity and Variance Inflation Factors (VIFs) with additional inclusion of any co-exposure. $PM_{2.5}$ models were not adjusted for SO_2 because they share a predominant local source in NYC (i.e., residual heating oil burning) and have very similar spatial and temporal patterns (Clougherty et al., 2013), thereby potentially over-adjusting. Further, NO_2 and O_3 were not mutually adjusted, given the strong inverse relationship between these two pollutants in NYC (Clougherty et al., 2013). Finally, adjusting any other exposures for the effects of NO_2 greatly increased VIFs and therefore, were not adjusted for NO_2. As part of the sensitivity analysis, we tested the robustness of results to two alternative co-exposure adjustment strategies. First, to assess the influence of co-exposure selection for adjustment per se, we adjusted each lag of each exposure of interest for case-day lags of all other co-exposures in the multi-exposure distributed lag models. Second, to assess the influence of lag structure beyond only the case-day lags, we adjusted for the lag days of each co-exposure that had the strongest effect estimate in the main multi-exposure distributed lag models.

The combined effects of the environmental exposures and the social stressors were assessed by fitting multiplicative interaction terms between each environmental exposure and quintiles of violent crime rate and SDI together in the same multi-exposure distributed lagged models; thereby, mutually adjusting for their potential independent interaction effects. Due to concerns about spatial clustering of social stressors by race and ethnicity (i.e., non-random assignment into census tracts), we additionally adjusted for interaction with proportions of Hispanic and non-Hispanic Black populations by census tract by including multiple interactions terms in a single model. Interaction results were reported as the effect of the environmental exposure of interest at each level

(quintile) of the social stressor, along with the corresponding p-value of the overall interaction term between the exposure and the stressor across each lag (significance level $\leq$0.05).

Additionally, we examined interactions between each air pollutant and temperature by fitting multiplicative interaction terms between the continuous terms of each air pollutant and median-dichotomized (low and high temperature) categories of spatio-temporal temperature estimates of the same lag day as that of the pollutant under consideration in the multi-exposure distributed lagged models. Interactions of the environmental exposures with categories of individual age at ED visit (5–9 years, 10–13 years, and 14–17 years), sex (female and male), and race/ethnicity (Hispanic, non-Hispanic Black, non-Hispanic other people of color, and non-Hispanic White) were also assessed.

All analyses were performed in SAS 9.4 (SAS Institute Inc. SAS Version 9.4. Cary, NC; 2014). The study was reviewed and approved by the Drexel University Institutional Review Board.

3. Results

A total of 145,834 ED visits for childhood asthma were recorded between January 1, 2005, and December 31, 2011. Fig. 1 illustrates the census tract-level distribution of all asthma case events per 1000 residents aged 5–17 years across NYC's boroughs. Most of the events occurred among children in the youngest (5–9 years) age group (52.82%), male (58.95%), and those who were identified as non-Hispanic Black (43.21%) and Hispanic (29.99%). The baseline characteristics of the study population are provided in Table 1.

The environmental exposure and social stressor profiles for the case events are provided in Table 2. Fig. 2 illustrates the NYCCAS spatial surfaces of the air pollutants and temperature for all case events. Fig. 3 illustrates the spatial distribution of the census-tract-level social stressors across NYC.

Table 1
Baseline characteristics of the study population, stratified by season.

Characteristic	Year-round [N (%)]	Warm season [N (%)]	Cold season [N (%)]
Total case events	145,838 (22.86)	62,772 (9.84)	83,066 (13.02)
Age at ED visit			
5–9 years	77,029 (52.82)	31,906 (21.88)	45,123 (30.94)
10–13 years	40,877 (28.03)	18,156 (12.45)	22,721 (15.58)
14–17 years	27,932 (19.15)	12,710 (8.72)	15,222 (10.44)
Sex			
Female	59,870 (41.05)	25,548 (17.52)	34,322 (23.53)
Male	85,968 (58.95)	37,224 (25.52)	48,744 (33.42)
Race/ethnicity			
Hispanic	43,734 (29.99)	17,796 (12.20)	25,938 (17.79)
Non-Hispanic Black	63,011 (43.21)	28,501 (19.54)	34,510 (23.66)
Non-Hispanic other people of color	6768 (4.64)	2706 (1.86)	4062 (2.79)
Non-Hispanic White	6716 (4.61)	3090 (2.12)	3626 (2.49)
Missing	25,609 (17.56)	10,679 (7.32)	14,930 (10.24)
Residential borough			
The Bronx	46,854 (32.13)	19,329 (13.25)	27,525 (18.87)
Brooklyn	42,926 (29.43)	18,625 (12.77)	24,301 (16.66)
Manhattan	26,999 (18.51)	12,057 (8.27)	14,942 (10.25)
Queens	24,226 (16.61)	13,616 (9.34)	10,610 (7.28)
Staten Island	4833 (3.31)	2682 (1.84)	2151 (1.47)

Each 10 μg/m^3 increase in $PM_{2.5}$ exposures was associated with an elevated risk of asthma ED visit in the cold season, with the strongest effect on lag day 1 [4.90% (95% CI: 3.77%–6.04%)]. Similar effects, but of lower magnitude were observed with $PM_{2.5}$ exposures in the warm

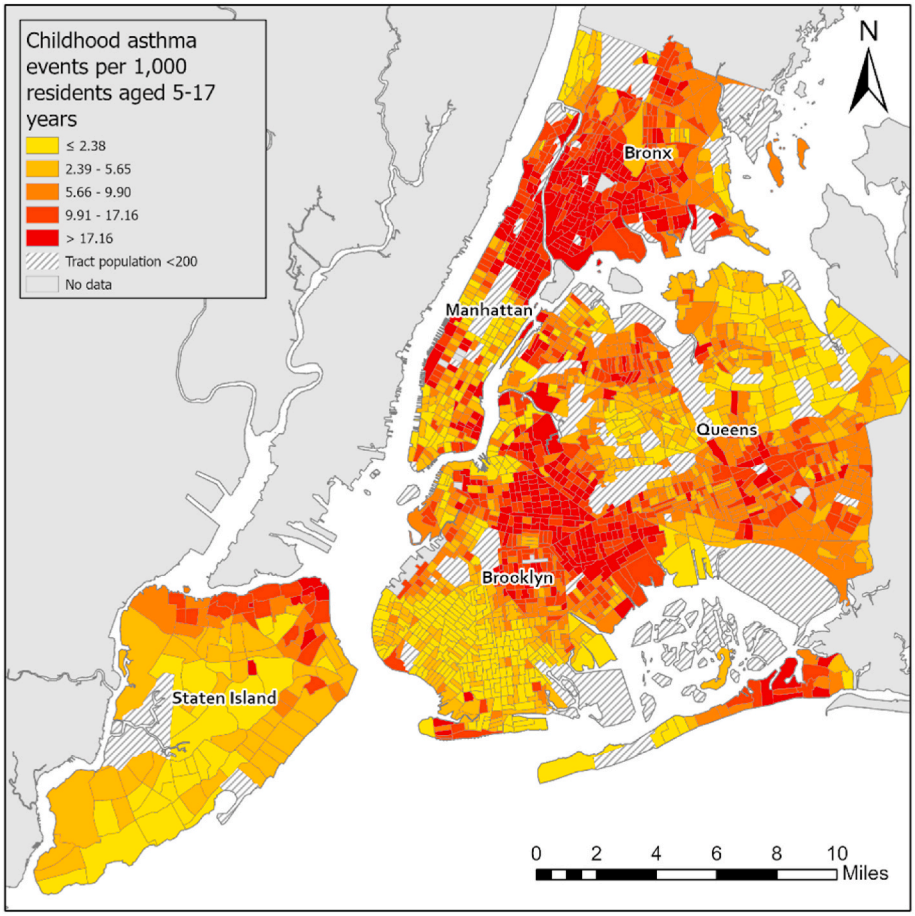

Fig. 1. Census-tract-level distribution of all asthma case events per 1000 residents aged 5–17 years across residential boroughs of New York City from 2005 to 2011.

Table 2
Residence-level, daily spatio-temporal environmental exposures and census tract-level social stressor profiles of asthma case events.

	Minimum	Maximum	Mean	Standard Deviation	Median	Interquartile Range
Residence-level, daily environmental exposures						
$PM_{2.5}$, $\mu g/m^3$	1.57	58.68	11.51	7.02	9.51	8.58
NO_2, ppb	3.73	100.84	27.59	10.73	25.97	13.89
SO_2, ppb	0.00	57.75	3.99	4.12	2.68	3.88
O_3, ppb	0.64	66.39	20.19	9.82	19.38	13.98
Tmin, °F	5.11	85.95	46.28	14.98	46.65	21.75
Tract-level social stressors						
Violent crime rate	0.00	2262.30	65.07	44.26	58.08	45.50
SDI	−1.63	2.39	0.65	0.66	0.69	1.01
% Hispanic	0.00	0.99	0.41	0.25	0.40	0.44
% Non-Hispanic Black	0.00	0.99	0.37	0.28	0.31	0.45

$PM_{2.5}$: fine particulate matter; NO_2: nitrogen dioxide; SO_2: sulfur dioxide (winter-only); O_3: ozone (summer-only); °F: degree Fahrenheit; $\mu g/m^3$: microgram per cubic meter; ppb: parts per billion; NYCCAS: New York City Community Air Survey; Violent crime rate per 100,000 residents; SDI: Socioeconomic Deprivation Index score.

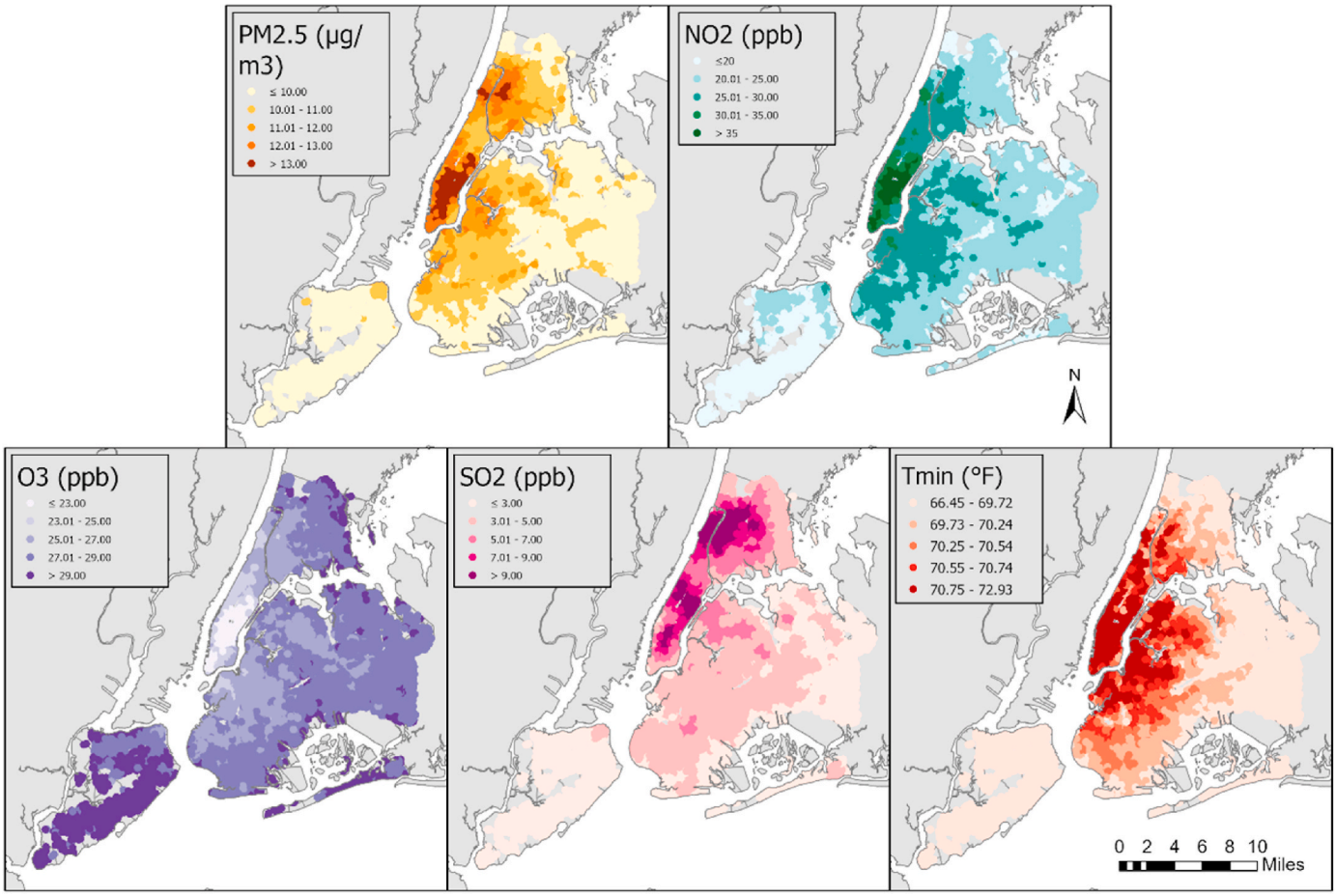

Fig. 2. Spatial surfaces (NYCCAS estimates) of the environmental exposures for all asthma case events.

season. Each 10-ppb increase in NO_2 exposures was associated with an elevated risk of asthma ED visit in the warm season across all lag days, with the strongest effect observed on lag day 1 [7.86% (95% CI: 6.66%–9.07%)]. Similar effects, but of lower magnitude, were observed with NO_2 exposures in the cold season. Each 10-ppb increase in SO_2 exposures was associated with an increased asthma ED visit risk in the cold season across lag days 1–6, with the strongest effects on lag day 1 [8.57% (95% CI: 5.99%–11.21%)]. Each 10-ppb increase in O_3 exposures was associated with an increased asthma ED visit risk in the warm season across lag days 1–4, with the strongest effects on lag day 1 [4.75% (95% CI: 3.53%–5.97%)]. Protective effects on asthma ED visit risk were observed with elevated temperatures in the warm season, with negative associations observed with each 10 °F increase in Tmin exposures across all lag days, with lag day 6 being the most protective [−16.97% (95% CI: 18.18% to −15.73%)]. In the cold season, however, increments in Tmin were positively associated with asthma ED visit risk across lag days 0–2, with lag day 0 demonstrating the strongest effect [2.26% (95% CI: 1.25%–3.28%)]. Complete details on the main effects of the environmental exposures are provided in Supplementary Table S1 and illustrated in Fig. 4, Fig. 5, and Supplementary Fig. S1.

The observed associations with NO_2, SO_2, O_3, and Tmin exposures were robust to both alternative co-exposure adjustment strategies, yielding similar results. Associations with $PM_{2.5}$, however, were sensitive to the strategy where we adjusted for the strongest lag day effects of all the co-exposures, identified in the main multi-exposure distributed lag models. We observed negative associations with $PM_{2.5}$ exposures in

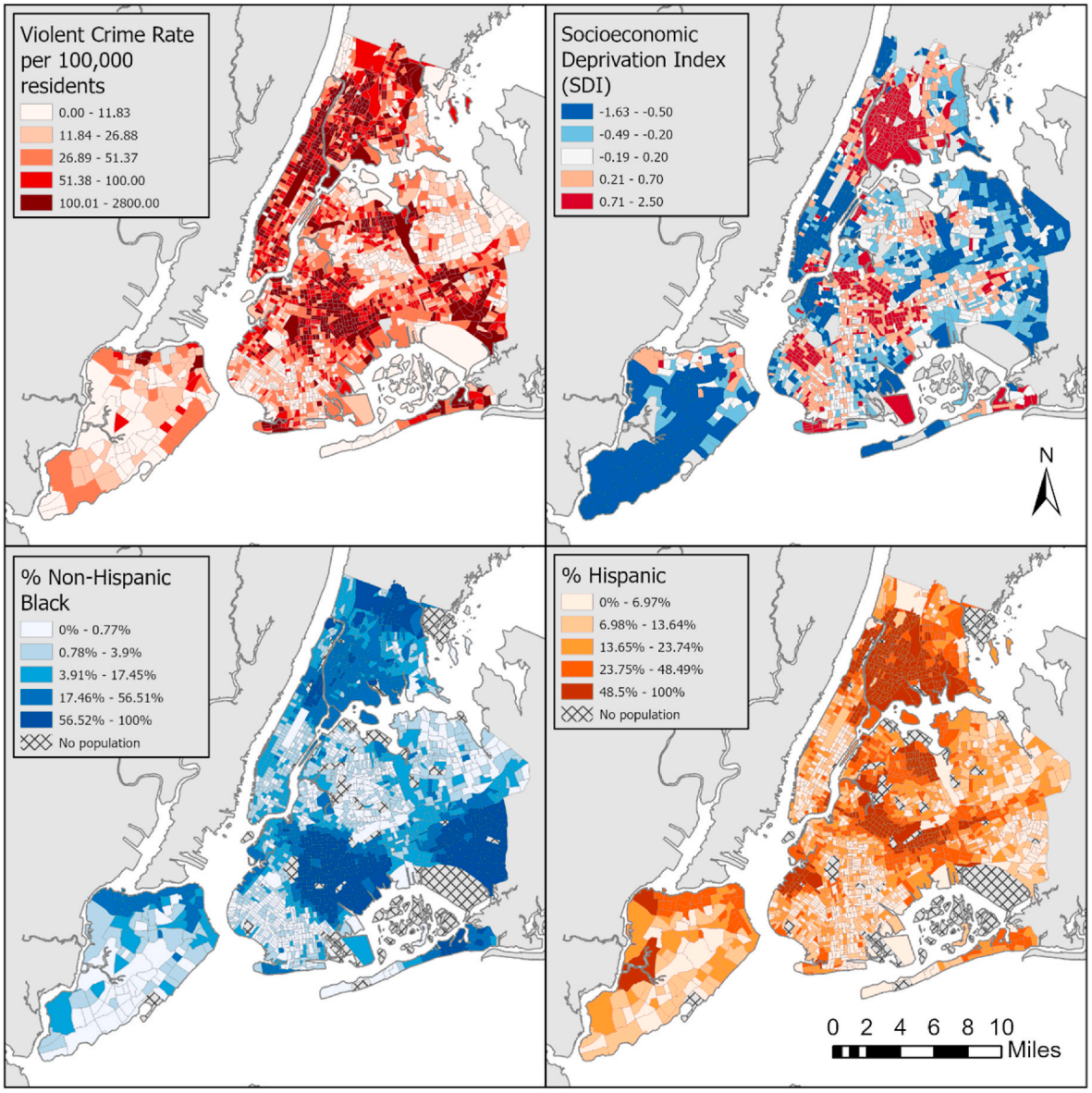

Fig. 3. Spatial distribution of the census tract-level social stressors and racial composition indicators across New York City.

the warm season on lag days 0–4 and positive associations with lag days 5 and 6. In the cold season, positive associations of about the same magnitude across all lag days were observed. The results of the sensitivity analyses are presented in Supplementary Tables S2 and S3 and illustrated in Supplementary Figs. S2 and S3.

On examining interactions between the environmental exposures and social stressors, we found evidence in the opposite-to-hypothesized direction—stronger effects were observed in the lower violence and SDI quintiles compared to higher violence and SDI quintiles.

With exposures to $PM_{2.5}$, interaction with violence was observed on lag day 1 in the warm season (p interaction = 0.05), with the strongest effect seen in violence quintile 2. With exposures to NO_2, interactions with violence were not evident across any of the seasons. However, we did observe a non-linear pattern of NO_2 effects. The strongest NO_2 effects were seen in the violence quintile 2 on lag days 1 and 2 in the warm season, and in violence quintile 1 on lag day 2 in the cold season. With exposures to SO_2, interactions with violence were also not evident, and no distinct effect pattern was observed in the cold season. With exposures to O_3, the interaction was evident on lag day 2 in the warm season (p interaction = <0.01), where a non-linear, 'U-shaped' pattern was observed, with the strongest effects seen in the lowest violence quintile 1 followed by quintile 5. With exposures to Tmin, interactions with violence were not evident across both seasons. However, an 'inverted V-shaped' pattern was observed on lag day 0 in the cold season, where violence quintile 3 had the strongest Tmin effect. Complete details on the interactions between the environmental exposures and quintiles of violent crime rate (adjusted for interactions with SDI and racial composition indicators) are provided in Supplementary Table S8 and illustrated in Figs. 6 and 7, and Supplementary Fig. S4.

On examining interactions with SDI, with exposures to $PM_{2.5}$, the interaction was evident on lag day 3 in the warm season (p interaction = 0.01), with the strongest effect seen in SDI quintile 3. In the cold season, interactions with SDI were observed on lag days 1 and 2 (p interaction = <0.01 and < 0.001, respectively) On lag day 1, an 'inverted J-shaped' pattern was seen with stronger $PM_{2.5}$ effects observed in the lower SDI quintiles 1 to 3, and on lag day 2, the strongest $PM_{2.5}$ effect was observed in SDI quintile 3. With exposures to NO_2, interactions with SDI were observed across both seasons. In the warm season, the interaction was evident on lag days 1 and 2 (p interaction = 0.03 and 0.05, respectively), with the strongest effects seen in SDI quintile 1. Likewise, in the cold

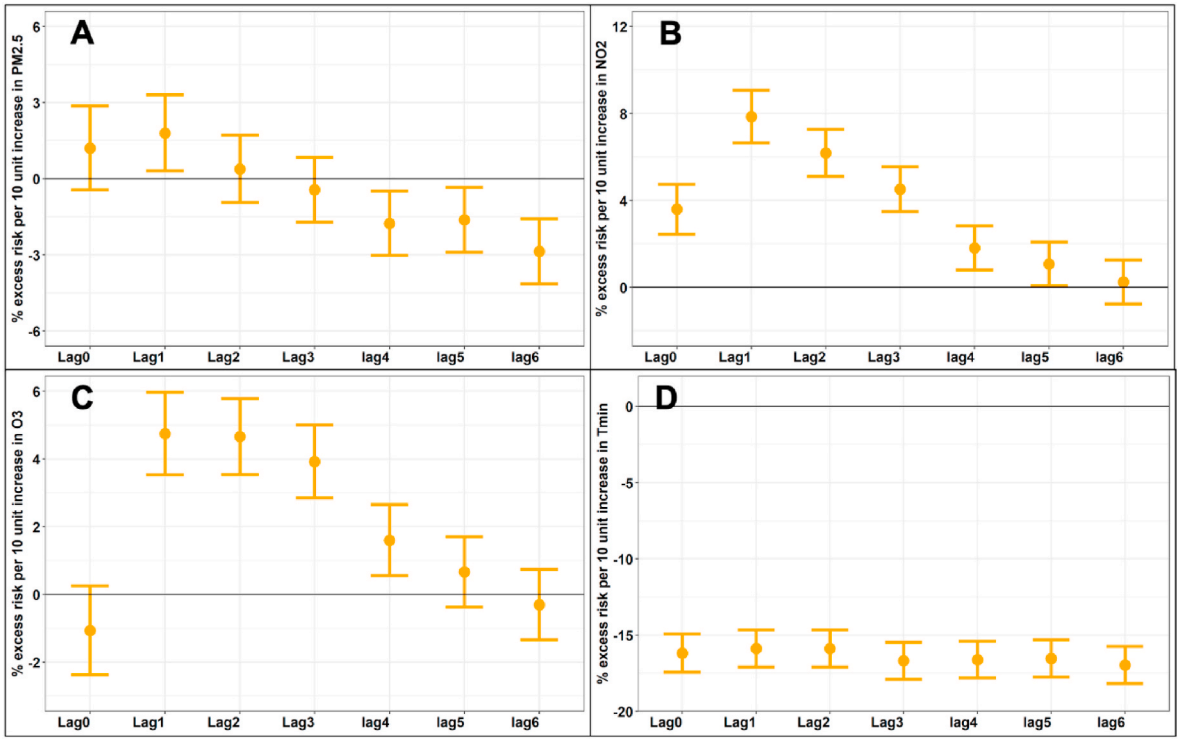

Fig. 4. Warm season main effects of (A) $PM_{2.5}$, (B) NO_2, (C) O_3, and (D) Tmin, following the main co-exposure adjustment strategy.

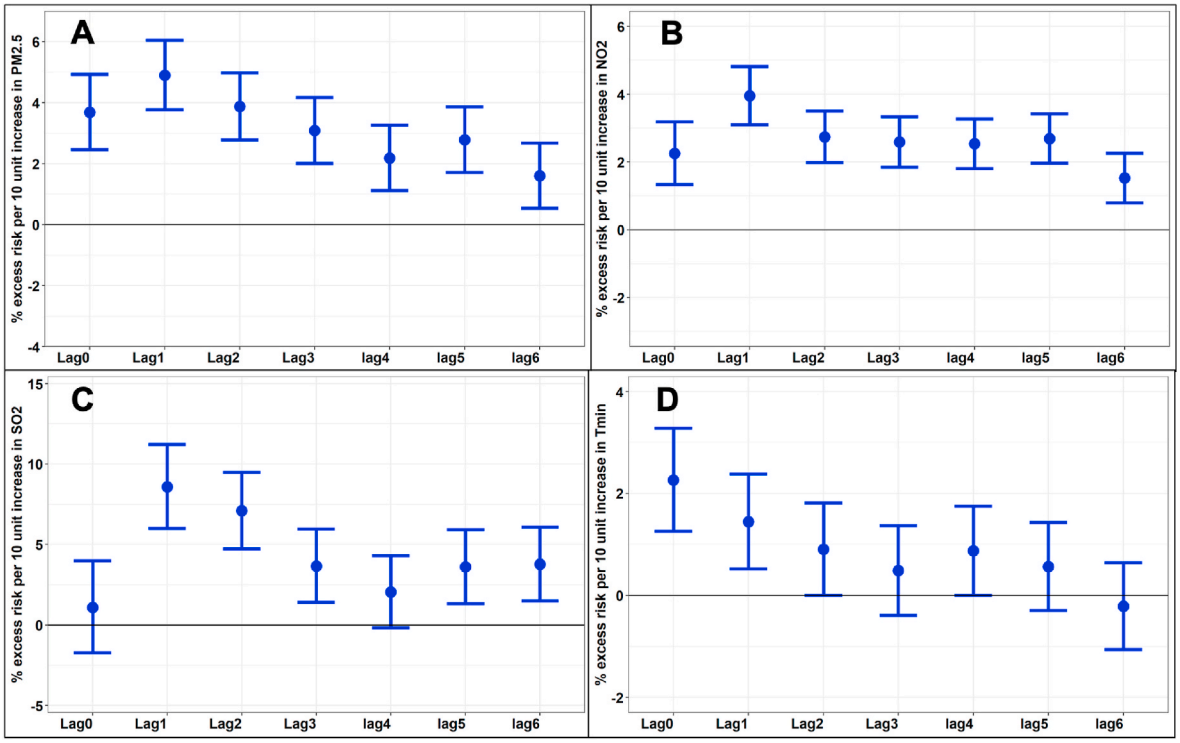

Fig. 5. Cold season main effects of (A) $PM_{2.5}$, (B) NO_2, (C) SO_2, and (D) Tmin, following the main co-exposure adjustment strategy.

season, the interactions were evident on lag days 1 and 2 (p interaction = <0.01 and < 0.001, respectively), where an 'inverted V-shaped' pattern was observed with the strongest effect seen in SDI quintile 3. With exposures to SO_2, interaction with SDI was observed on lag day 2 in the cold season (p interaction = <0.01), also with an 'inverted V-shaped' pattern of effects. With exposures to O_3, the interaction was evident on lag day 5 (p interaction = 0.01). However, we did not see a distinct pattern of effects. With exposures to Tmin, interaction with SDI was observed on lag day 2 (p interaction = <0.01) in the warm season, with the strongest protective effect of Tmin seen in SDI quintile 1. Similar results were observed for lag days 4, 5, and 6 (p interaction = 0.03, 0.05, and 0.02, respectively). In the cold season, interaction with SDI was

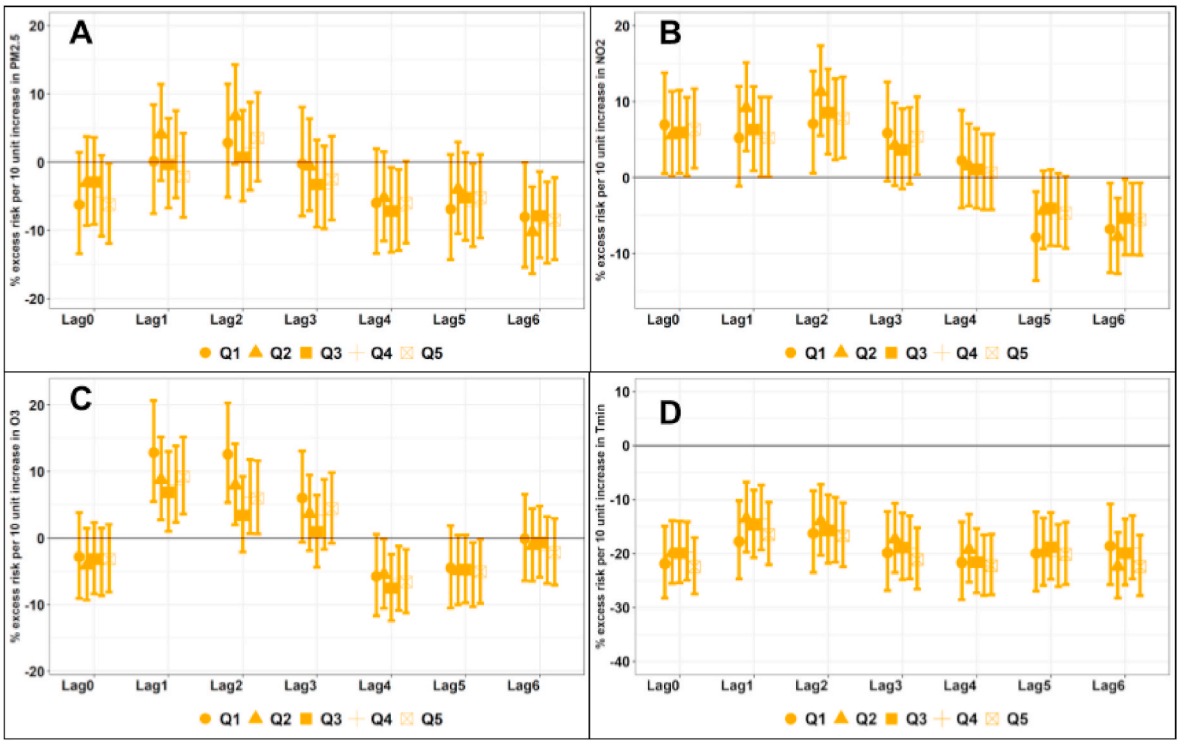

Fig. 6. Warm season effects of (A) $PM_{2.5}$, (B) NO_2, (C) O_3, (D) Tmin by quintiles of violent crime rate from models including interaction terms.

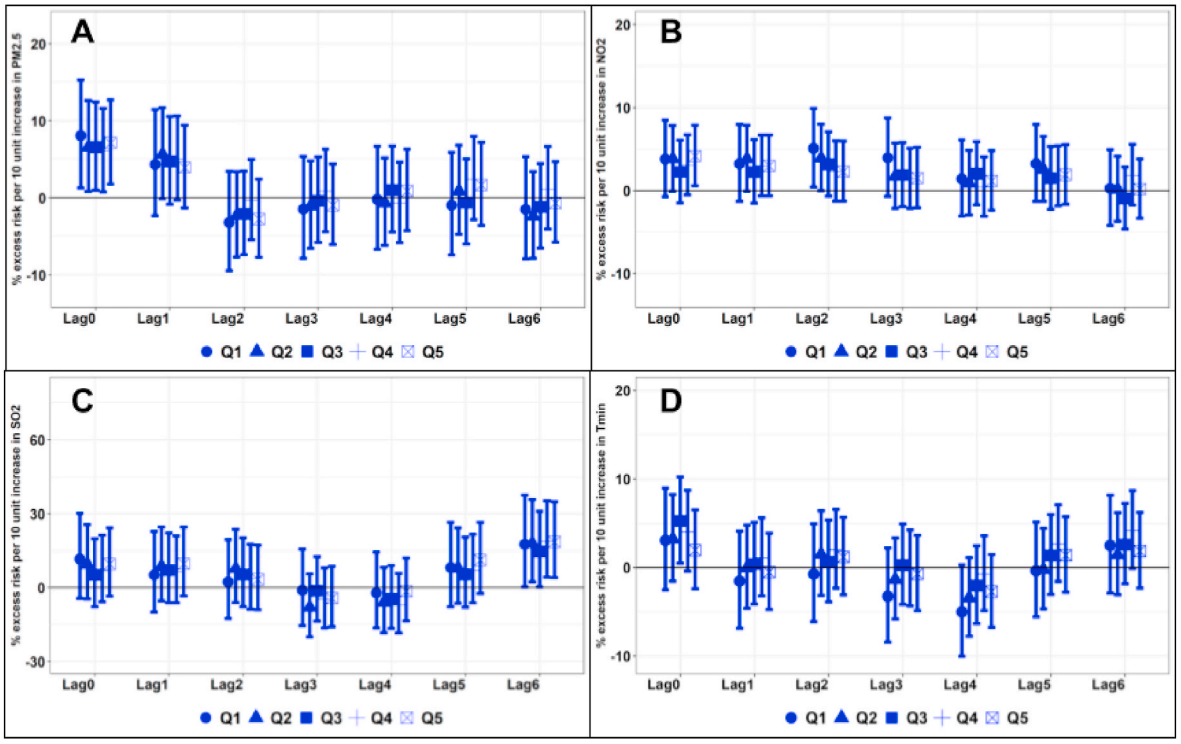

Fig. 7. Cold season effects of (A) $PM_{2.5}$, (B) NO_2, (C) SO_2, (D) Tmin by quintiles of violent crime rate from models including interaction terms.

evident on lag day 3 (p interaction = 0.05), where a 'U-shaped' pattern was observed with the strongest Tmin effects seen in SDI quintiles 1 and 2. Complete details on the interactions between the environmental exposures and quintiles of SDI score (adjusted for interaction with violent crime rate and racial composition indicators) are provided in Supplementary Table S9 and illustrated in Figs. 8 and 9, and Supplementary Fig. S5.

We also found evidence of interactions between the air pollutants and temperature, and between the environmental exposures and individual age at admission, sex, and race/ethnicity. These results are discussed in the Supplementary Text, and the complete details are provided in Supplementary Table S6—S9 and illustrated in Supplementary

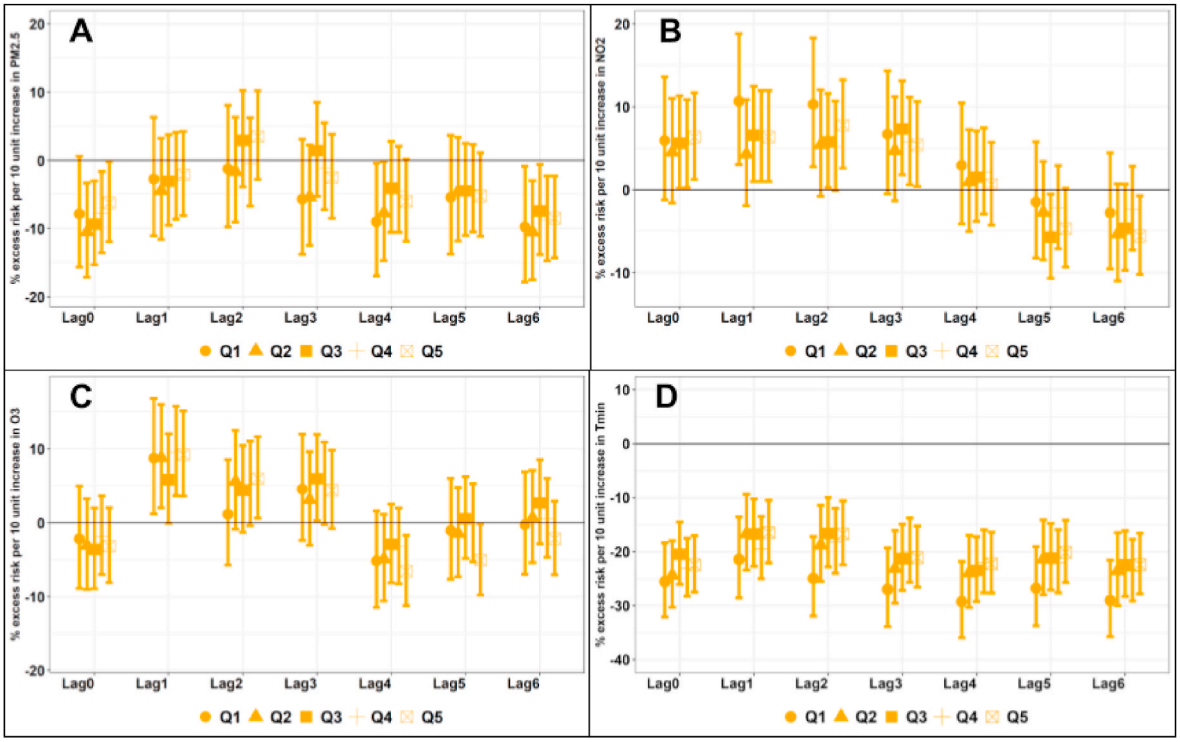

Fig. 8. Warm season effects of (A) $PM_{2.5}$, (B) NO_2, (C) O_3, (D) Tmin by quintiles of Socioeconomic Deprivation Index score from models including interaction terms.

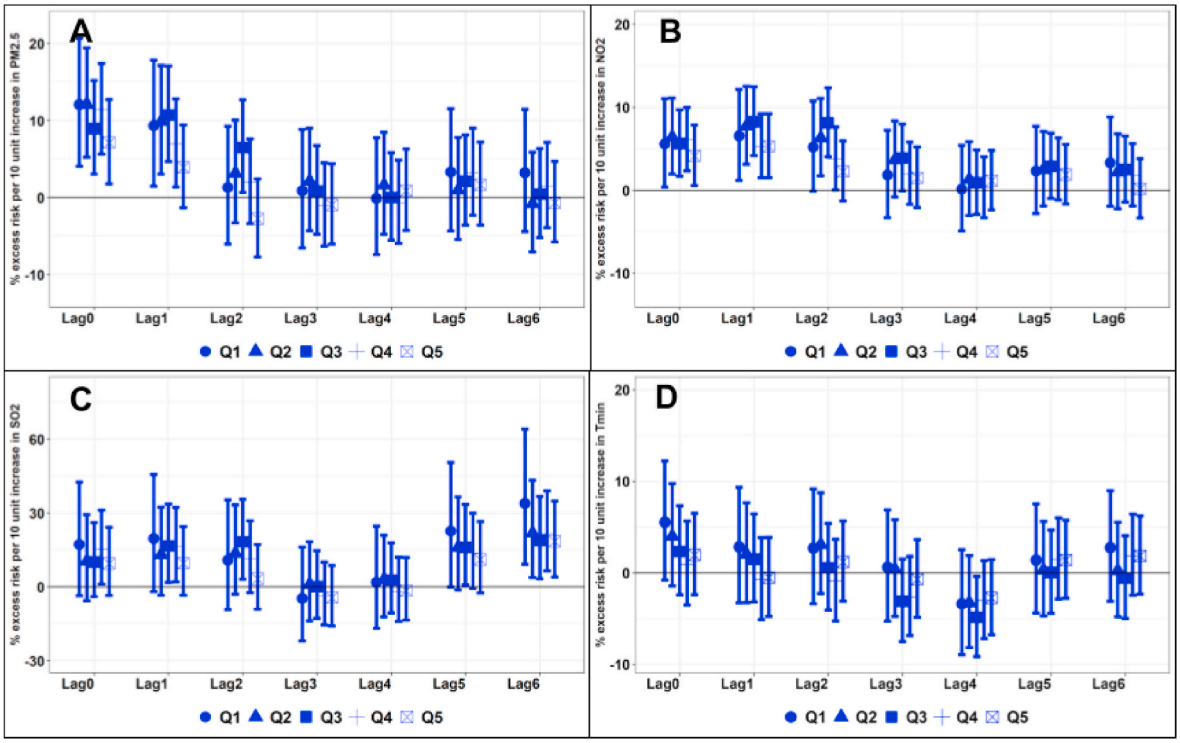

Fig. 9. Cold season effects of (A) $PM_{2.5}$, (B) NO_2, (C) SO_2, (D) Tmin by quintiles of Socioeconomic Deprivation Index score from models including interaction terms.

Fig. S6—S9.

4. Discussion

In this study, using SPARCS data from 2005 to 2011, we examined the season-specific independent and combined effects of multiple ambient air pollutants and temperature, and their interactions with neighborhood violent crime and socioeconomic deprivation to discern susceptibility to childhood asthma morbidity in New York City.

The positive associations observed between $PM_{2.5}$ exposures and asthma in the current study were more apparent during the cold season. This difference may be explained by increased $PM_{2.5}$ emissions (vis-à-vis

exposures) from local sources like burning of residual heating oil and vehicular traffic (with more idling and high cold-start emissions (Arumugam Sakunthalai et al., 2014) and less effective exhaust filtration systems (Mahadevan et al., 2015)) in the winter months in NYC (Clougherty et al., 2013). Similarly, the positive associations with SO_2 exposures could be explained by local emissions also from residual heating oil burning during the cold season as well as long-range interstate transport of SO_2 (e.g., from Ohio Valley) into the northeastern U.S. (Bergin et al., 2007). These cold season effects, however, could additionally be explained by increased likelihood of exposures to household allergens due to greater time spent indoors and peaks in seasonal influenza and other respiratory illnesses during winters that may exacerbate asthma. The positive associations observed with O_3 exposures in the warm season are in keeping with the fact that O_3, a secondary air pollutant, is formed from its precursor pollutants (oxides of nitrogen and volatile organic compounds) by undergoing photochemical reactions in sunlight and has greater concentrations during the warm season (Jhun et al., 2015; Sillman, 1999). In keeping with the season-specific measurements of SO_2 and O_3 under NYCCAS and to minimize the exposure measurement error, we presented only cold season SO_2 and warm season O_3 effects. However, associations of asthma with SO_2 during summer and O_3 during winter seasons have also been reported in prior literature, albeit the findings overall remain mixed. In their meta-analysis, Bergmann et al. (2020) found no conclusive evidence of effect modification by season on asthma with $PM_{2.5}$, NO_2, and SO_2 exposures and only found increased risks for asthma morbidity during the warm season with exposures to O_3.

Further, temperature elevations during the cold season were associated with a greater risk of asthma ED visits. This association could be due to behavioral changes involving greater outdoor mobility on warmer days during colder months, thereby increasing the likelihood of ambient environmental exposures and stronger asthma effects. In contrast, we observed a lower risk of asthma ED visits (i.e., protective effects) with elevated temperatures during the warm season. This inverse association could have resulted, in part, from better asthma treatment-seeking behavior and medication adherence, especially among individuals with chronic asthma, during the spring and summer allergy seasons. In the prior literature, strong evidence exists for positive associations of both hot and cold temperature extremes with adverse asthma outcomes. Cong et al. (2017) reported in their meta-analysis that decreases in short-term temperature exposures were associated with increased odds of childhood asthma. In a more recent meta-analysis, Han et al. (2023) reported that exposure to extreme cold was associated with increased asthma risk among children and extreme heat exposures also conferred an increased risk of asthma, albeit to a greater extent in adults.

We found evidence of non-linear effects and interactions between the environmental exposures and social stressors. These findings were in the opposite-to-hypothesized direction—the risk of asthma ED visits associated with elevated environmental exposures was generally stronger in children belonging to the lower violence and deprivation communities and not in higher violence and deprivation communities, as had been predicted. These findings are novel and add to the emerging evidence suggesting potential threshold or saturation effects in stress-related susceptibility to the health effects of air pollution and temperature—wherein, potential asthma impacts could be predominantly explained by social stressors in high-stressor (violence and deprivation) communities, with lesser apparent variation by air pollution or temperature exposures (Clougherty et al., 2014, 2022).

In a similar case-crossover analysis, using 2005 to 2011 SPARCS data, Sheffield et al. (2019) examined the effects of interactions between daily, residence-level O_3 exposures and census tract-level violent crime and socioeconomic deprivation on childhood asthma ED visits during the summer season (June to August) in NYC. The main effects of O_3 were similar to those observed in the current study but were mostly stronger in communities with greater violence or socioeconomic deprivation. However, the authors also noted that the highest quartile for violent crime did not consistently confer a greater risk than the lower quartiles and reported some evidence of threshold or saturation effect in crime-related susceptibility to O_3 effects. We anticipate that the non-linear relationships with violent crime observed with exposures to O_3 in the current study became more apparent with the inclusion of year-round ED visits data (vs summer-only in Sheffield et al.) that increased the sample size and statistical power to examine the interactions. Also in NYC, using vital records and SPARCS data on live births from 2008 to 2010, Shmool et al. (2015a,b) examined the interactions between spatial NYCCAS surfaces of NO_2 and census tract-level socioeconomic deprivation on term birth weight, adjusting for maternal SEP characteristics and $PM_{2.5}$ exposures. The authors found evidence of non-linear interaction with inverse associations observed between exposures to NO_2 and birthweight in the least and the most socioeconomically deprived neighborhoods. In a cross-sectional analysis, Hicken et al. (2013) assessed whether social disadvantage modified associations between blood pressure and monthly-average $PM_{2.5}$ exposures estimated before the baseline examination in the Multi-Ethnic Study of Atherosclerosis (MESA) cohort established across 6 U.S. cities, including Northern Manhattan, NYC. Contrary to their hypothesis, the authors found that greater levels of income were associated with stronger relationships between $PM_{2.5}$ and blood pressure. In a similar study based out of the MESA cohort, Allen et al. (2009) observed that the association between annual-average $PM_{2.5}$ exposures and aortic calcification was stronger in individuals with higher incomes than with those with lower incomes. In a more recent study, Miller et al. (2022) examined the effects of interactions between early life stress (ELS) and $PM_{2.5}$ exposures on structural brain development among adolescents in California. Exposures to $PM_{2.5}$ were linked to volumetric alterations in various areas of the grey and white matter of the brain in individuals who experienced less severe ELS. Those who experienced more severe ELS had fewer negative effects from exposures to $PM_{2.5}$.

This study has several noteworthy strengths. First, combined effects of exposures to multiple air pollutants, temperature, and social stressors on asthma morbidity have been rarely examined. This study, to our knowledge, is one of the few to examine interactions between multiple socio-environmental co-exposures and highlight potential non-linearities in their effects. Second, we had a large sample size with year-round data on childhood asthma ED visits included for all of NYC over the 7-year study period. Third, we assigned fine-scale spatio-temporal environmental exposure estimates (daily, at 300-m buffer using 100-m exposure concentrations) to each presenting asthma case, thereby reducing the possibilities of ecological bias in our observed associations. Fourth, individual-level covariates like sex, race/ethnicity, genetic predisposition, and exposure to environmental tobacco smoke, which are generally of concern in asthma epidemiology, were inherently adjusted for in the case-crossover design and could not have confounded our results.

This study, however, is also prone to some limitations. First, we examined exposure-response relationships in relation to acute, post-natal, ambient air pollution and temperature exposures. Long-term (Anenberg et al., 2022; S. S. Liu et al., 2021; Tiotiu et al., 2020), pre-natal (Hazlehurst et al., 2021; Hehua et al., 2017; Lee et al., 2018), as well as indoor air pollution (Breysse et al., 2010; Tiotiu et al., 2020) and environmental allergens such as mold (Dick et al., 2014; Michaels, 2017) and pollen (Erbas et al., 2018; Kitinoja et al., 2020) have been independently linked with childhood asthma but were not measured in this study and may have contributed to residual confounding. Second, we estimated environmental exposures at residence, but children could have been exposed at school, contributing to exposure measurement error. Third, we did not have data on assets like air conditioner use that could have influenced exposures to air pollution and temperature, also contributing to measurement error. Fourth, we lacked information on asthmatic medication and treatment use. This may have confounded our results as healthcare utilization for asthma varies by SEP and

race/ethnicity, differentially influencing asthma outcomes (Johnson et al., 2021; Keet et al., 2017; K. Sullivan and Thakur, 2020).

5. Conclusion

We observed elevated ED visit risks for childhood asthma with exposures to $PM_{2.5}$ and NO_2 during both the cold and warm seasons, SO_2 and Tmin during the cold season, and O_3 during the warm season in New York City. Non-linear effects and interactions with violent crime and socioeconomic deprivation were evident, with lower violence and deprivation quintiles generally demonstrating stronger exposure-response associations, indicating potential threshold or saturation effects in socio-environmental stressor synergism. Our findings contribute to a growing body of literature which seeks to disentangle the complex contributions of multiple social stressors in modifying the effects of environmental exposures resulting in health disparities.

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Credit author statement

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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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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2023.116235.

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