Environmental Health

RESEARCH

Open Access

Changes in the short-term relationship between air pollution and mortality in New York City, 1990–2019

Rebecca Goldberg[1], Ariel Spira-Cohen[1], Masha Pitiranggon[1], Sarah Johnson[1] and Kazuhiko Ito[1*]

Abstract

Background Few studies have examined how short-term associations between air pollution and mortality have evolved over recent decades when air quality has improved. Public health policy can benefit from timely information.

Methods We applied time-series models to estimate the mortality impacts of ambient nitrogen dioxide (NO_2), ozone (warm season only), and fine particulate matter ($PM_{2.5}$) in 5-year moving time windows between 1990 and 2019 (2000–2019 for $PM_{2.5}$) in New York City (NYC). We modeled full-year, warm (May through September) and cold (October through March) season NO_2 and $PM_{2.5}$, adjusting for temperature, temporal trends, day-of-week, and holidays. We also estimated Total Risk Index (TRI) to characterize changes in the combined risk from exposure to two and three pollutants.

Results All three pollutants showed the strongest association at one lag day. Despite major declines in $PM_{2.5}$ and NO_2 levels over the study period, risk estimates showed no apparent trend, remaining generally positive, but became less precise over time as concentration variability also declined. The estimated overall 1-day lag percent excess risk for $PM_{2.5}$ was 0.49% (95% confidence interval: 0.12, 0.86) per 10 μg/m^3 24-hr average, and for NO_2, 0.90% (0.30, 1.50) per 30 ppb daily 1-hr maximum for full year models. Ozone, which slightly increased over the period, had a 1-day lag risk estimate of 1.43% (0.56, 2.30) per 30 ppb daily 8-hr maximum. The TRI followed a similar pattern to individual pollutants' estimates.

Conclusions With no clear evidence of risk per unit increase changing over time, the reductions in $PM_{2.5}$ and NO_2 concentrations imply declines in excess deaths. Notably, ozone levels and health burden persist. NO_2, which was most robustly associated with mortality and represents two major combustion sources—traffic and fossil fuel combustion in buildings—may be the most relevant indicator of energy transition progress in urban areas like NYC in the coming decade.

Keywords Air pollution, Mortality, Concentration-response function, Short-term

*Correspondence:
Kazuhiko Ito
kito1@health.nyc.gov
[1]New York City Department of Health and Mental Hygiene, Bureau of Environmental Surveillance and Policy, 125 Worth Street, New York, NY 10013, USA

Background

An increasing number of public health institutions and researchers have been estimating mortality and morbidity burdens of ambient air pollutants, such as fine particulate matter ($PM_{2.5}$) [1, 2]. These studies—often called health impact assessments, or HIA—use available or estimated air pollution levels combined with concentration-response (CR) functions (i.e., relative mortality risk per unit increase in an air pollutant) from available epidemiological studies and baseline health outcomes for a given geographic boundary. To facilitate policy decisions on emission reduction options, HIA tools are available to assess the health burdens, including monetary values associated with loss of life and costs of medical care associated with specific emission reduction scenarios [3, 4]. Researchers have used such tools, for example, to estimate the benefits of avoided health impacts for specific policy scenarios in New York City (NYC) for $PM_{2.5}$ [5–8].

Local governments need to make policy decisions that meet their greenhouse gas emission goals while also estimating public health benefits of improved air quality. HIA tools facilitate such benefit-to-cost analysis, but applying these tools requires assessing uncertainties that may be relevant and specific to a given locality. First, the CR functions built into these tools come from past epidemiological studies typically conducted in other locations with different demographics at different time-periods (e.g., ten years ago) and may not be applicable to the current setting or location. Second, the CR functions in these tools are typically for a single pollutant, most commonly $PM_{2.5}$, and ignore the effects of other pollutants present. In other words, current HIA tools do not address the possible impacts from air pollution mixes. This issue is important because reducing one source's emissions typically results in reduction of multiple pollutants [9]. Finally, while the accountability of emission reductions leading to reduced adverse health impacts was often tested in "natural experiments" in which the health impacts of a sudden change in emissions were assessed [10–12], most emission regulations start with a phase-in period and take several years to complete. Several time-series studies examined how CR functions changed over an extended period of time [13–18], but the policy relevance of such findings may be unique to those cities' specific emission profiles, air pollution mixtures, and population demographics. Therefore, characterization of these uncertainties would benefit future policy planning for NYC, which has focused on climate sustainability goals and budgeting.

The objective of this study is to characterize changes in the short-term relationships between the currently policy-relevant air pollutants, $PM_{2.5}$, nitrogen dioxide (NO_2) and ozone (O_3). and non-external mortality in NYC over three decades, 1990 through 2019 ($PM_{2.5}$ data available only from 2000). Previous studies of the short-term effects of air pollution—mostly focused on particulate matter and/or O_3—on mortality that reported risk estimates for NYC are limited to those that analyzed data up to 2006 [19–23], after which the levels of NO_2 and $PM_{2.5}$ in NYC declined due to a combination of multiple local emission control regulations, market-driven change in building fuel types, and a reduction in regional aerosols [9, 24–27]. NYC's large population size allows for time-series modeling in incremental intervals and provides an illustrative example of how estimated risks for the three regulated air pollutants—ubiquitous in most urban environments—changed over decades.

Methods

Air pollution and weather data

We retrieved $PM_{2.5}$ (available from 2000), NO_2, and O_3 data from U.S. Environmental Protection Agency's (EPA's) Air Quality System [28] for the five boroughs in NYC and counties in New Jersey adjacent to NYC. We limited monitors to those assigned a population exposure monitoring objective. Completeness criteria were applied so that hourly data from a given day were excluded if less than 75% of hourly measurements were collected, and any data were excluded if collected from a monitor that did not meet EPA criteria for annual completeness. We used metrics consistent with the National Ambient Air Quality Standard for each pollutant: NO_2: daily 1-hr max; O_3: daily 8-hr max; and $PM_{2.5}$: 24-hr average. To obtain one daily value for epidemiologic modeling, we first computed the citywide average concentrations from multiple monitors in NYC only, taking into consideration the difference in the mean and variance across monitors [29]. We assessed the extent of missing data and repeated the citywide average computation, now including the monitors from the sites in New Jersey adjacent to NYC, and filled in any missing values in the NYC-only citywide average data. The remaining small number of missing values (< 3% for all the pollutants) were filled in with either the previous or following day's values to produce a complete time-series for each pollutant.

Weather data for LaGuardia airport was retrieved from the National Oceanic and Atmospheric Administration global hourly database [30], and daily mean temperature values were calculated.

Mortality data

Mortality records of non-external deaths of NYC residents who died within the five boroughs of NYC from 1990 to 2019 were obtained from NYC's Office of Vital Statistics and aggregated by date. Non-external cause deaths were collected by excluding external causes–ICD-9 codes greater than or equal to 800 or ICD-10 codes beginning with letters S through Y. Analyses were

limited to NYC residents with a known borough of residence.

Overview and rationale of the study design

Previous studies that characterized air quality trends in New York State (NYS) and NYC indicated that the declines in air pollution emissions in the past two decades were substantial but not abrupt, similar to the NO_2 and $PM_{2.5}$ concentration changes [9, 24–26]. Thus, we did not expect the short-term effects of these pollutants on mortality to change abruptly. Therefore, our main research question considered if the short-term mortality risk of an air pollutant changed over time, expressed as a percent excess risk (PER) per a fixed incremental increase of each pollutant. Additionally, we sought to characterize any change in trends of the total excess risks from these pollutants. The emission sources in NYC vary by season (e.g., NO_2 from building space heating increases in cold season), so we estimated PERs by season. Based on the range of effect size observed for $PM_{2.5}$ and O_3 from previous studies that included NYC [19, 20, 22, 31], we chose a 5-year moving time window to compute seasonal and full-year PERs of mortality for 1990–2019 for NO_2 and O_3 and 2000–2019 for $PM_{2.5}$. This approach is descriptive and similar to those applied in other recent studies that investigated changes in the short-term effects of air pollution over an extended period of time [14, 16, 18], except that the moving time windows, rather than fixed time windows, allows assessment of any individual year's influence on estimated PERs. Note also that a set of every 5th point of these moving time window estimates (26 estimates for 1990–2019 and 16 estimates for 2000–2019) is equivalent to non-overlapping time windows.

Descriptive analysis

We first conducted descriptive analysis of both air quality and mortality patterns over time to identify changes in average values, variability in the data, and cyclical seasonal patterns. These patterns aid in interpretation of changes in estimated risks. Air quality trends were examined by creating annual time series of mean values. We examined how the standard deviations of the air pollutants and mortality changed over time because the variability of both dependent and predictor variables influences the standard error of estimated risks (i.e., the lower the variability of x and y variables, the wider the confidence intervals for estimated risks) in the quasi-Poisson regression models for daily mortality. For each of the 5-year moving time windows, we also computed Pearson correlation coefficients for each pair of the daily air pollutants as well as correlations between each pollutant and the concurrent day temperature and the average of lag 1 through 3 days temperatures (i.e., the covariates for mortality models). We also characterized how age of decedents and proportion of major underlying causes have changed in NYC over time. To characterize how seasonality of air pollutants, temperature, and mortality changed over time, we fitted these time-series with a cyclical smooth function [32] of day-of-year with up to 5 degrees of freedom per year in generalized additive models using the Gaussian family for air pollutants and temperature and quasi-Poisson family for mortality by 5-year blocks of study days. Because the seasonality of mortality could be influenced by seasonal influenza, we included time series of influenza and pneumonia (ICD9: 480–488; ICD10: J09-J18) in the mortality model to adjust for their influence while estimating the cyclic seasonal pattern.

Model specifications

To assess any change in the short-term effects of air pollutants over a long study period (30 years for NO_2 and O_3 and 20 years for $PM_{2.5}$), we applied a time-series model in rolling five-year time windows. This approach assumes that the covariates' (e.g., temperature) relationships with mortality also change over time and allows characterization of gradual changes in the air pollution risks. In each of the five-year time windows, we ran generalized linear quasi-Poisson regression models of up to three lag days for each pollutant. When multiple lagged air pollution variables appeared associated with mortality, we also considered distributed lag non-linear models (DLNM) from the *dlnm* package [33]. These used a linear functional form with an unconstrained lag function, and we compared the cumulative risk estimates with those from a single lag model. Models were adjusted for immediate (lag 0 day) and delayed (average of lag 1 through 3 days) temperature, year, holidays, day of week, and the natural spline of study days with 7 degrees of freedom per year for the full year models to adjust for long-term and seasonal trends and influenza. Because air pollution mixture and exposure conditions can vary across seasons, we also ran separate models for warm months (May through September) and cold months (November through March) to estimate season-specific effects. O_3 was analyzed for its association with mortality in warm months only because its concentrations are only relevant during the warm season and to be consistent with past studies of O_3 and mortality [34]. For the seasonal models, we adjusted for within-season trends using the natural spline of study days with 3 degrees of freedom per season. The seasonal transition months, October and April, were excluded from the seasonal models to distinguish the pattern of associations between colder and warmer seasons. Cold months models adjusted for winter season period, as opposed to calendar year, to account for winter months spanning two calendar years. In computing PERs, we used the following fixed increments: NO_2: 30 ppb daily 1-hr max; O_3: 30 ppb daily 8-hr max; and $PM_{2.5}$: 10 μg/

m^3 24-hr average, which are often used in U.S. EPA's Integrated Science Assessment documents for these pollutants [34–36].

The issue of confounding among air pollutants is typically addressed through multi-pollutant models. A review of multi-pollutant models by Davalos et al. identified five broad classes of statistical methods to estimate the short-term effects from multi-pollutant exposures and provided guidance on the selection of the optimum method based on study objectives and prior knowledge of the multi-pollutant exposures [37]. Based on our study goal of assessing the changes in the short-term air pollution effects over time from the changes in emissions from major sources, we opted to use what Davalos et al. classified as the Additive Main Effects method—estimation of individual and combined risks without dimension reduction or specifying effect modification—to estimate the short-term mortality risks from $PM_{2.5}$, NO_2, and O_3. In implementing this method, we adopted the Total Risk Index (TRI) method that was originally developed for estimating the cumulative long-term effect from multiple pollutants [23]. The premise of TRI is that the linear combination of the risk coefficients from multiple correlated pollutants can be reliably estimated even when the individual pollutants' risk coefficients are unstable due to multicollinearity. This method is analogous to distributed lag models where a cumulative risk is computed from multiple days' (correlated) air pollution values. The standard error of the TRI risk coefficient takes into consideration the correlation of multiple pollutants' risk coefficients to mitigate an inflated standard error from multicollinearity.

When emissions policies target multiple pollutants, modeling only one or two pollutants cannot give a full picture of the risk or benefit associated with reductions. The TRI value is more useful than individual pollutant estimates for policy development as each pollutant is modeled independently, but effects are combined into one estimate. Thus, in addition to single pollutant models, we ran two- and three-pollutant TRI models for 2000–2019 when all pollutants were available to assess how much each pollutant contributes to the overall risk estimate, as well as to provide more useful estimates for future policy considerations. Two-pollutant models were compared to a three-pollutant model only in the warm season because we did not consider O_3 in the cold months. Single-pollutant models were compared to two-pollutant TRI models in the cold season and for full years. In calculating PERs for single and TRI models, we used the same increments used in single-pollutants models mentioned above.

Sensitivity analysis

We applied several alternative model specifications to check the sensitivity of results from our main model: alternative degrees of freedom (6 and 8 degrees of freedom per year for full year models and 2 and 4 degrees of freedom per season for warm and cold season models) for trend adjustment with natural spline of study days; individual lag vs. cumulative risk using DLNM with a linear functional form of air pollutants at lag 0 and 1 days; and differences in reported risk using the pollutant interquartile range (IQR) vs. EPA's Integrated Science Assessment (ISA) value as the increment. To consider potential changes to estimates from inclusion of a second pollutant, we also ran two-pollutant models with combinations of NO_2, O_3, and $PM_{2.5}$.

This study was approved by the Institutional Review Board of NYC Department of Health and Mental Hygiene.

Results

The annual average of daily 1-hour max NO_2 declined by 58% from 1990 (63 ppb) to 2019 (26 ppb) and 43% from 2000 (46 ppb), as shown in Fig. 1. NO_2 warm season annual average concentrations declined at a faster rate than cold season average concentrations. Accordingly, the seasonality of NO_2 changed during the study period: peaking in summer in the 1990s; becoming comparable in both seasons by 2009; then peaking in cold season by 2019 (Fig. 2). NO_2's 5-year window standard deviations for warm season declined from 18 ppb during 1990–1994 to 11 ppb during 2015–2019; however, for cold season, NO_2 standard deviations slightly increased since early 2000s after a sharp decline in the 1990s (Fig. S1).

The annual average of daily 24-hr $PM_{2.5}$ declined by 57% from 2000 (14.9 $\mu g/m^3$) to 2019 (6.5 $\mu g/m^3$). As with NO_2, warm season average $PM_{2.5}$ concentrations declined at a faster rate than in the cold season (Fig. 1). Unlike NO_2, whose seasonality reversed in the 30 years, $PM_{2.5}$ seasonality qualitatively remained the same between 2000 and 2019, with bimodal peaks, one in summer and the other in winter, both declining but with the winter peak now higher (Fig. 2). $PM_{2.5}$'s 5-year window standard deviations for warm season declined from ~9 $\mu g/m^3$ during 2000–2004 to 4 $\mu g/m^3$ during 2015–2019. The corresponding values for cold season were ~7 $\mu g/m^3$ to 4 $\mu g/m^3$, respectively, indicating that the rate of decline was steeper for warm season (Fig. S1).

The warm season annual average of daily 8-hr maximum O_3 levels fluctuated over time, generally following the year-to-year variation in summer temperature (i.e., lower O_3 in cooler summers), as shown in Fig. 1. While the overall trend of annual average of daily O_3 values was essentially flat during the study period, the amplitude of O_3's annual cycle shrunk slightly (Fig. 2) because the

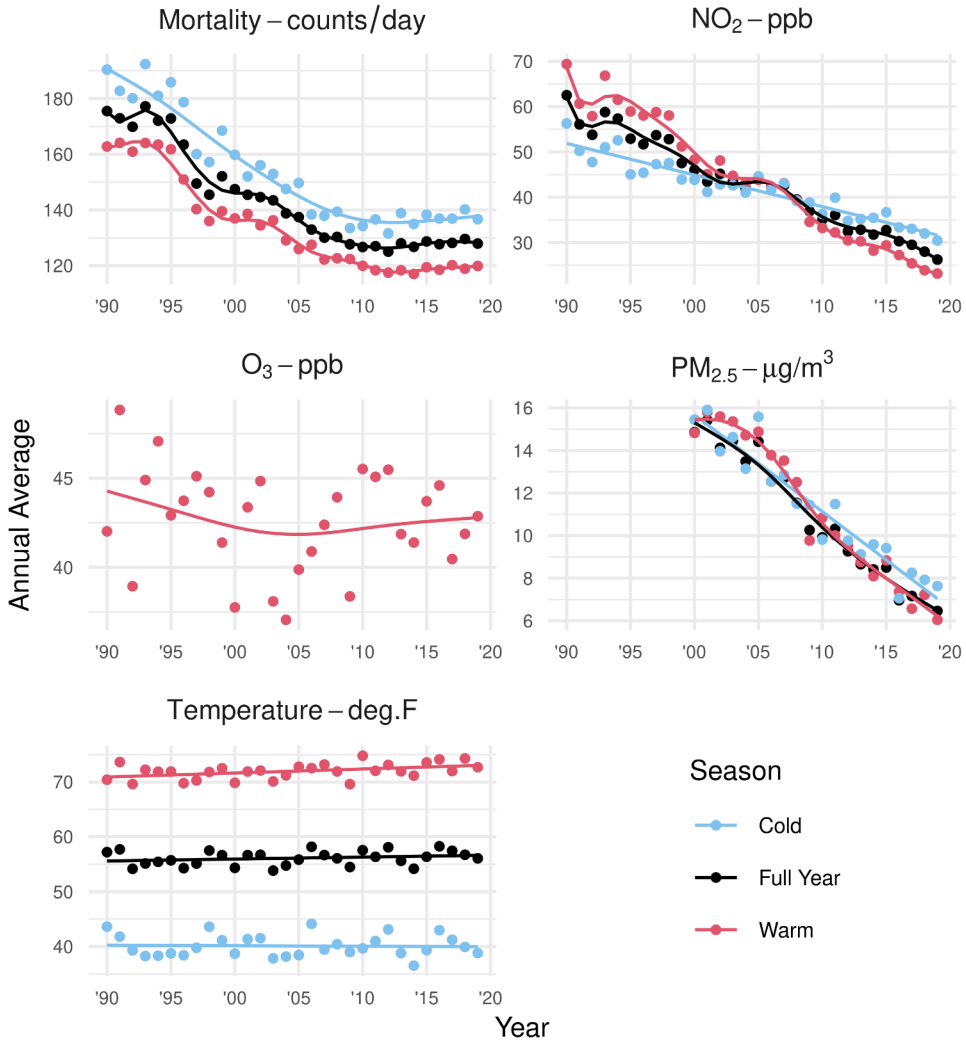

Fig. 1 Annual average for full year, warm season (May– September) and cold season (November– April), 1990–2019. The lines are smoothed spline fits with 10 degrees of freedom over the data period

upper range values of its distribution decreased, and the lower range increased (e.g., O_3 levels in May are higher in more recent years). These changes are also reflected in O_3's 5-year standard deviations, which declined from 19 ppb for 1990–1994 to 13 ppb for 2015–2019. Daily average warm season temperature increased by ~2 °F during the study period (Fig. 1), but its seasonality remained the same (Fig. 2). Temperature's 5-year window standard deviations have been generally steady for both warm and cold seasons (Fig. S1).

Temporal correlations in the 5-year time windows between NO_2 and $PM_{2.5}$ for warm months fluctuated between 0.45 and 0.60 during the study period with lower values in more recent years. Their correlations in cold months were more stable at approximately 0.6 (Fig. S2). NO_2's correlation with O_3 in warm months declined from approximately 0.6 in the 1990s to 0.35 in 2010s, as did its correlations with both the year-round same-day and average of 1- through 3-days temperature. The temperature and NO_2 correlation changed from weakly positive (r: 0.05 to 0.10) in the earlier period to negative ($r \approx$ -0.20) in the later period, likely reflecting the larger contribution from building fossil fuel burning compared to traffic sources in more recent years. Correlations between the temperature variables with $PM_{2.5}$ and O_3 generally remained consistent across the study period.

The deaths for underlying causes of diabetes and cancer slowly increased during the study period (Fig. S3). On average, there were 52,410 non-external deaths per year from 1990 to 2019, ranging from 64,054 in 1990 to 46,699 in 2019. There were 30,228 cardiovascular deaths and 5,721 respiratory deaths in 1990 compared to 20,358 and 3,897 in 2019, respectively. Daily mortality counts declined between 1990 and 2010 and have been steady since then (Fig. 1). The population in NYC during the study period increased—7.3, 8.0, 8.2,

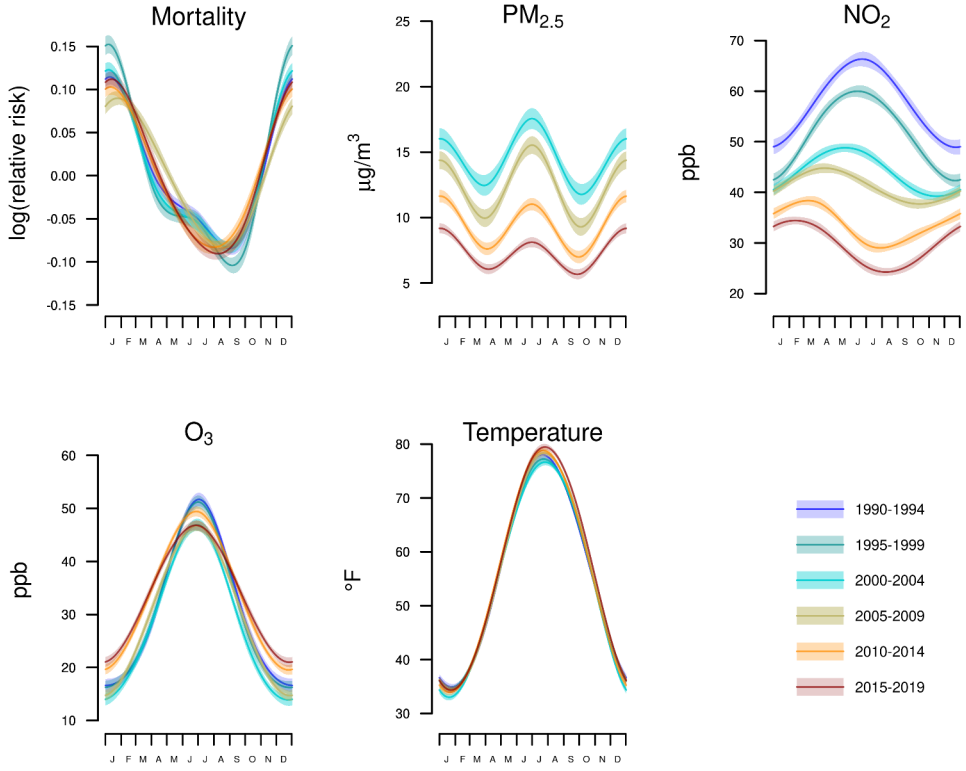

Fig. 2 Change in seasonality of non-external deaths, $PM_{2.5}$ (2000–2019), NO_2, O_3, and temperature. Fitted curves from cyclic smoothing in 5-year intervals are shown

and 8.8 million people for 1990, 2000, 2010, and 2020, respectively—indicating the declining number of deaths reflects decreasing death rates, particularly age-adjusted premature death (death at age < 65), which declined by 20% between 2006 and 2015 alone [38]. The seasonality of deaths, after accounting for influenza epidemics, did not meaningfully change (Fig. 2). The 5-year window standard deviations for mortality declined steeply before mid-2000s especially for cold season, but they are stable in the last decade of the study period (Fig. S1). The proportion of deaths for older adults (ages 85+) increased from 21 to 34% between 1990 and 2019, while those for younger age groups declined (Fig. S3). The deaths with underlying causes declined between 2008 (42%) and 2012 (34%), which is consistent with the reported decline that may be, in part, due to policy initiatives and the effort to reduce coding bias [39, 40].

Of the lags analyzed (0–3 days), 1-day lagged variables showed the most consistent associations with mortality for all three pollutants in the main model over the entire period (results for the other lags are shown in Fig. S5). We therefore focused on the 1-day lag pollutant results to depict PER trends. Figure 3 shows PERs from five-year moving time windows over the study period by season and for all year (warm season only for O_3). While most models showed positive associations, especially for warm season, there was no indication of a monotonic change in the trend of the risk estimates. A consistent widening of confidence bands over time was observed, however. The widening confidence bands of PERs were most pronounced for $PM_{2.5}$. These widening confidence bands are consistent with, and in part explained by, the pattern of decline in IQRs of these pollutants over the study period mentioned above: the standard error of the regression coefficient to estimate excess risk in a quasi-Poisson model is inversely proportional to the variance of the pollutant, and the variance, as reflected in the IQR, declined as the pollutant levels declined. In the cold season, NO_2 and $PM_{2.5}$ (which are consistently correlated with $r \approx 0.6$, as shown in Fig. S2) showed the same fluctuation pattern of PERs, becoming negative during the period that centers around 2008 to 2009. The warm season PERs generally followed the same pattern as the corresponding PERs in the cold season but were more positive. Fluctuations in PERs for O_3 only partly correspond to other pollutants' trends. These fluctuation patterns did not follow those of standard deviation of the variables (Fig. S1) nor the intercorrelations among the pollutants and temperature variables (Fig. S2).

PER estimates from the TRI models (from year 2000 on for $PM_{2.5}$) also showed variability over time without any trend, regardless of using two- or three-pollutant models (Fig. 4). The TRI model with NO_2 and $PM_{2.5}$ resulted in the same pattern as those for their single-pollutant

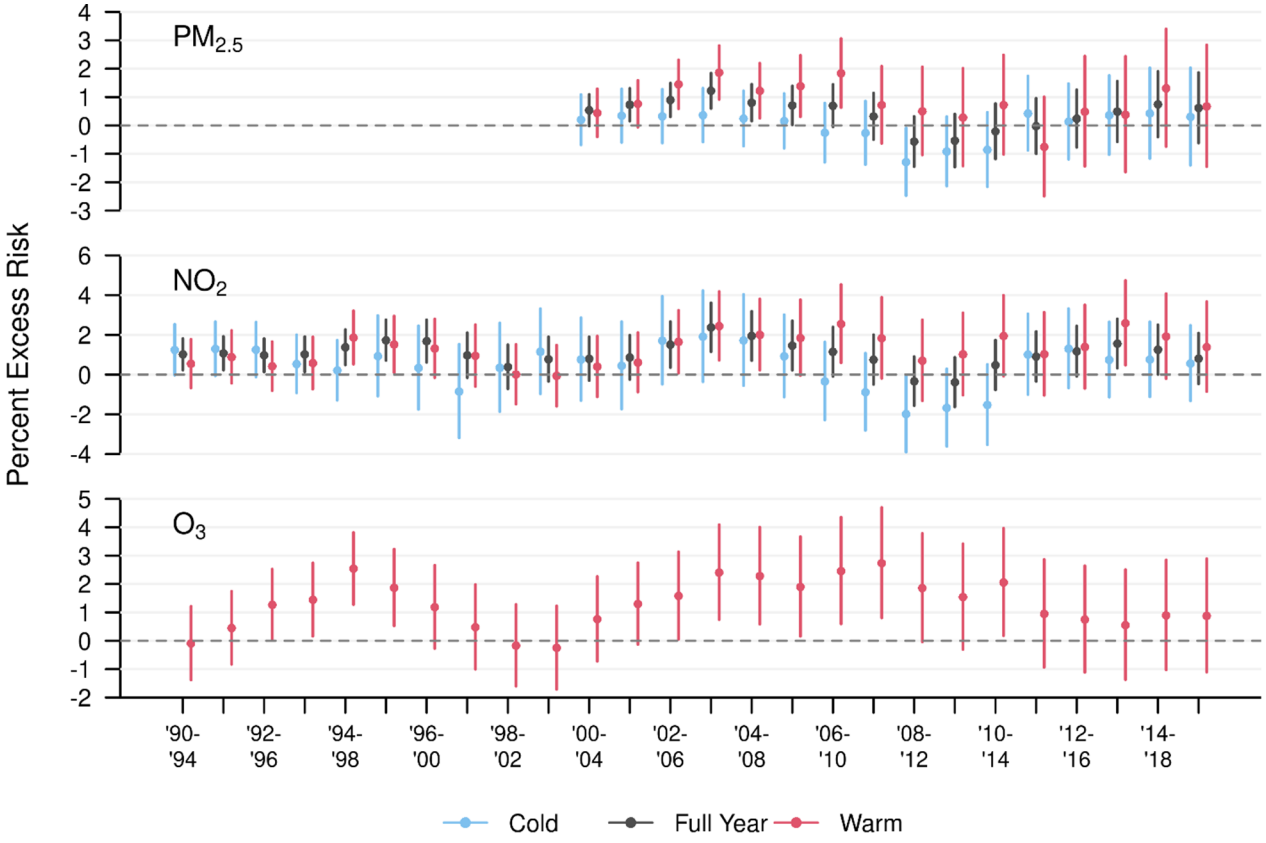

Fig. 3 Change in percent excess risk for non-external deaths for $PM_{2.5}$, NO_2, and O_3 at lag 1-day by season, 1990–2019 (2000–2019 for $PM_{2.5}$) using rolling 5-year data. The increments used to compute percent excess risks: 10 μg/m^3, 30 ppb, and 30 ppb, for $PM_{2.5}$, NO_2, and O_3, respectively

counterparts. The TRI model with NO_2 and O_3 resulted in generally larger PERs than those from corresponding single pollutant models. Adding $PM_{2.5}$ to the TRI model with NO_2 and O_3 hardly changed PERs but slightly increased their confidence bands, implying that $PM_{2.5}$ does not uniquely contribute to PERs once NO_2 and O_3 are in the model in warm season.

Because the PERs for individual pollutants or TRI models did not show any monotonic change during the study period, we computed PERs for individual pollutants and TRI models for the entire 2000–2019 period (Fig. 5). The PERs for the warm months were generally larger than those for the cold months. The PERs from two- and three-pollutant TRI models were larger than those from single-pollutant but with wider confidence intervals. Overall, $PM_{2.5}$ showed weaker associations with mortality than NO_2 (warm and cold seasons, and full year) and O_3 (warm season).

Results from sensitivity analysis with alternative model specifications for warm and cold seasons and full year models for lags 0 through 3 days are shown in Fig. S5. For lag 1 day pollution models (the main model), the results with two-pollutant models are also shown. Using one less degree of freedom than is used for the cold season lagged main model (i.e., 2 rather than 3 degrees of freedom per season) to adjust for within-season and seasonal trends generally had the largest influence in cold season PERs, increasing their value. Using one more degree of freedom per season or year for seasonal adjustment did not yield PERs that were meaningfully different from those of the main models. Using IQRs of the entire available period by season, rather than the fixed increment, to compute PERs yielded slightly smaller estimates for all the pollutants. Note that using IQRs, especially in the context of our study design, is not useful because the variability of most pollutants declined over the study period. Adding a co-pollutant reduced PERs for all pollutant/season combinations. The confidence bands for PERs in the co-pollutant models were all wider than those for single-pollutant models, as expected. PERs for the cumulative lag 0 and 1 days using distributed lag models were either comparable or smaller than those from the main lag 1 day model, with wider confidence bands.

Discussion

The ambient concentrations of $PM_{2.5}$ and NO_2 in New York City declined substantially between 2000 and 2019—by ~60% and ~40%, respectively—due to multiple federal and local emission regulations, combined with a market-driven switch in fuel types in the power sector.

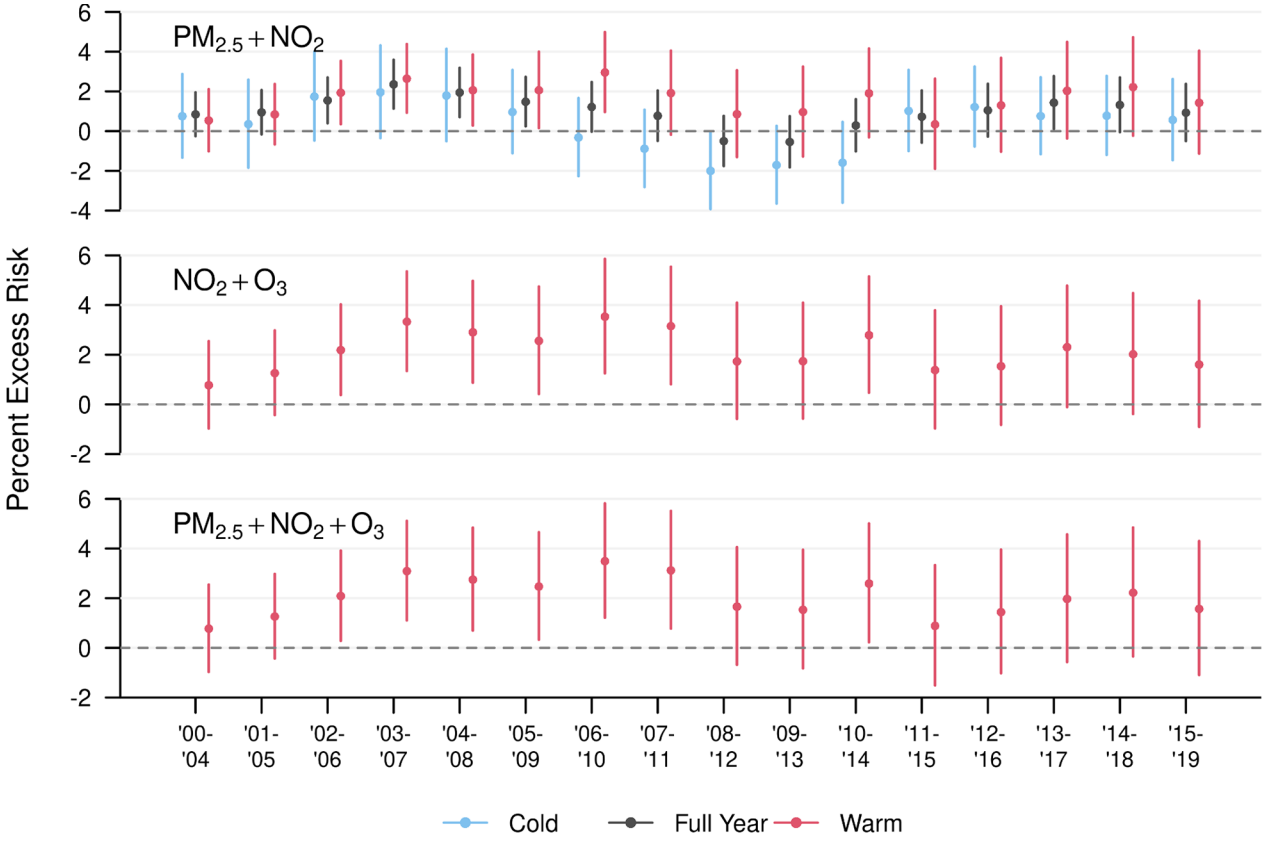

Fig. 4 Change in percent excess risk for non-external deaths for Total Risk Index by season, 2000–2019 using rolling 5-year data. The increments used to compute percent excess risks: 10 μg/m^3, 30 ppb, and 30 ppb, for $PM_{2.5}$, NO_2, and O_3, respectively

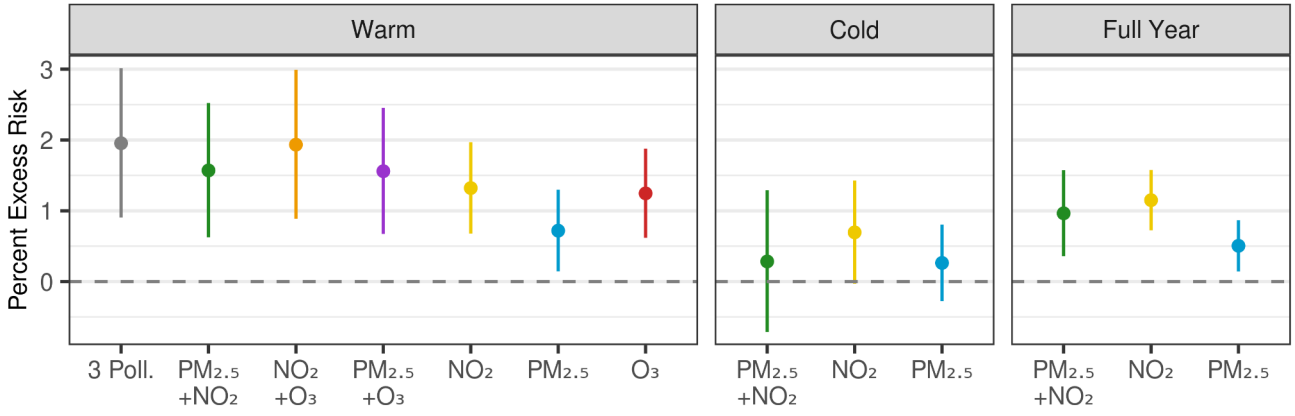

Fig. 5 Estimated percent excess risk of mortality in single-, two-, and three-pollutant models by season, 2000–2019. The increments used to compute percent excess risks: 10 μg/m^3, 30 ppb, and 30 ppb, for $PM_{2.5}$, NO_2, and O_3, respectively. Multi-pollutant models use the Total Risk Index for combined risk estimates

In contrast, warm season O_3 concentrations over the same 20 years have remained approximately the same. Our analysis of the short-term associations of these three pollutants with non-external cause mortality in a series of 5-year moving time windows showed that the estimated PERs per fixed increments remained approximately the same, though they varied over time. Consequently, the excess deaths attributable to $PM_{2.5}$ and NO_2 also declined, while those for O_3 remained the same. Our multi-pollutant assessment found that the TRI fluctuated in parallel to the risk estimates for the individual pollutants and that the total (two- or three-pollutant) risks are only incrementally larger than those of individual pollutants' risk estimates. We did identify the declining precision of risk estimates, most pronounced for $PM_{2.5}$, as a potential challenge for future monitoring of the

short-term effects of these pollutants, if their concentrations continue to decline, though this is not an issue in the larger public health context.

Several studies have investigated emission sources responsible for the decline in $PM_{2.5}$ concentrations over the past two decades in NYS and NYC using EPA's $PM_{2.5}$ chemical constituent data [9, 24, 26]. These studies generally suggested that the regional decline in transported secondary sulfate—due to the emission reduction from coal-fired power plants in the Ohio Valley region—contributed the most to the overall decline in $PM_{2.5}$ mass concentrations in NYS and NYC. Also for NYC, decreased emissions from residual oil burning from buildings, traffic, and ships contributed to a local decline in $PM_{2.5}$ [9].

Researchers from the University of Rochester and University at Albany hypothesized that the changes in $PM_{2.5}$'s chemical constituents associated with specific emission policies could modify $PM_{2.5}$ toxicity and, in turn, its short-term risk per unit mass. These studies categorized a range of years into specific periods associated with policy and/or economic changes that were used as an indicator of effect modification of $PM_{2.5}$ risk for cardiovascular hospitalizations [41], triggering of myocardial infarction [42], respiratory infection emergency department visits and hospitalizations [43], and asthma and COPD ED visits and hospitalizations [44]. The same research team also analyzed source-apportioned $PM_{2.5}$—eleven sources identified including spark ignition emission, diesel, road dust, etc.—for their associations with specific causes of cardiovascular hospitalizations [45]. These studies found associations between specific periods or source-apportioned $PM_{2.5}$ (generally combustion-related) and specific health outcomes. We are currently collaborating with these NYS researchers to analyze their source-apportioned $PM_{2.5}$ for its association with mortality in NYC. If $PM_{2.5}$ levels continue to decrease, analyzing the role of specific chemical components will become increasingly challenging because of the declining precision of estimates we identified, along with levels proportional to this change.

Several studies have examined changes in the short-term mortality effects of air pollution, including: NO_2 in 24 Canadian cities (1984–2004); [13] PM_{10} in Athens, Greece (2001–2012); [16] PM_{10} in Seoul, Korea (2001–2015); [14] NO_2, SO_2, oxidizing substances ("O_x", considered equivalent to O_3), and suspended particulate matter (equivalent to approximately 7 micrometer diameter cut-off) in 10 Japanese cities (1977–2015); [18] and, NO_2, PM_{10}, and $PM_{2.5}$ (1995–2016) in 380 cities from 24 countries [46]. These cities were mostly in mid- to high-income countries that observed some extent of declines in these pollutants, except for O_x in the Japanese study, the only study that examined this pollutant. While the results from the studies in Seoul and Athens suggested some changes in effect size over the period, a decline in PM_{10} risk estimate in the former and a suggestive increase in the latter, the three large multi-city studies indicated that the effect sizes per unit increase generally have not changed. Widening confidence intervals in later years can be observed in the figures in the Seoul and Athens studies (though not specifically mentioned), but none of these studies experienced the extent of concentrations reductions we observed in NYC.

The relatively unchanged PERs for $PM_{2.5}$ and NO_2, despite substantially lower levels, may appear counter-intuitive, but are consistent with the lack of evidence of a threshold in the short-term relationships between these pollutants and all-cause deaths. If there is no threshold and the underlying concentration-response relationship is approximately linear for the observed concentration range, then the observed slope at lower concentration ranges would remain the same until concentrations become so low—to the point where an elongated ellipse of air pollution (x-axis) and mortality (y-axis) data points from the earlier period becomes more of a circle—for a given study period that the risk estimate becomes too imprecise (i.e., wide confidence intervals) for interpretation. This happens because, in the log-linear (quasi-Poisson) regression model, the standard error of the regression coefficient for a pollutant is inversely related to the square-root of the pollutant's (and mortality's) variance, so that a pollutant with declining mean levels (and declining standard deviations, as shown in this analysis) would have a wide confidence band in the later years. This phenomenon is most prominent in the $PM_{2.5}$ result, which shows that the 95% confidence band in the most recent period (2015–2019) is at least twice that of the earliest period (2000–2004). Further reductions in $PM_{2.5}$ levels would make the excess risk estimate too imprecise, at least for a 5-year study period, even if the underlying relationship remained positive.

U.S. EPA publishes ISA documents for each of the criteria pollutants—for which National Ambient Air Quality Standards are established—every five years and provides a causal determination of the relationship between each pollutant and a specific health outcome based on the most recent evidence available from epidemiological and toxicological studies. For the short-term association with non-accidental mortality, the $PM_{2.5}$ determination in the 2019 PM ISA concluded that "there is a causal relationship" [36], and the 2022 supplement to the 2019 PM ISA supported this conclusion based on a review of more recent studies [47]. In contrast, the 2016 ISA for Oxides of Nitrogen concluded that the short-term association between NO_2 and non-accidental mortality is "suggestive of, but not sufficient to infer, a causal relationship" [35]. The 2016 NOx ISA also noted, regarding the

NO_2-mortality associations, "A limitation of this collection of studies was that the majority focused specifically on PM and did not conduct extensive analyses to examine the relationship between short-term NO_2 exposure and mortality" [35]. Likewise, the 2020 O_3 ISA concluded estimates were "suggestive of, but not sufficient to infer, a causal relationship" regarding the short-term mortality association [34], denoting a downward change from the 2013 O_3 ISA's causal determination, "suggestive of a causal relationship" [48].

In contrast to the 2016 EPA assessment of NO_2, in a 2020 systematic review of 196 studies of the short-term exposure to PM (PM_{10} and $PM_{2.5}$), NO_2, and O_3 and all-cause and cause-specific mortality, the authors found positive associations for short-term exposure to all these pollutants with all-cause mortality, with the level of evidence noted as "high" [49]. In a 2020 analysis of an open cohort constructed from Medicare data for Massachusetts, 2000–2012, with estimated $PM_{2.5}$, NO_2, and O_3 exposure levels assigned at residential zip-code level, the authors applied a generalized propensity score adjustment approach to assess causal associations and concluded that "long- and short-term exposures to $PM_{2.5}$, O_3, and NO_2 were all causally associated with increased risk of mortality." [50] Two systematic reviews (2015 and 2021) of NO_2-mortality short-term associations both found that NO_2 was associated with all-cause, cardiovascular, and respiratory mortality [51, 52]. Another systematic review (2016) of NO_2-mortality short-term associations specifically attempted to distinguish NO_2 associations from those from PM mass and found that the NO_2-mortality short-term association is "largely independent of PM mass." [53] Also, a 2021 time-series analysis of 398 cities from 22 countries found short-term associations between NO_2 and all-cause, cardiovascular, and respiratory mortality and reported that these associations "remained robust after adjusting for co-pollutants." [54] Thus, our findings provide additional evidence of NO_2-mortality associations established in recent systematic reviews and a large multi-city time-series study.

In our analysis NO_2 exhibited the most robust associations with daily mortality. To date, however, most health impact assessment studies of mortality conducted in the world and U.S. cities, including NYC, have focused on PM only [1, 5–8], or PM and O_3 [55, 56]. For NYC, our results demonstrate that NO_2 is increasingly the most policy-relevant indicator of air pollution for several reasons: (1) its short-term association with mortality in both warm and cold seasons; (2) NO_2 contributions to the formation of nitrate, a significant fraction of $PM_{2.5}$ especially in winter, and the formation of O_3 in summer, both of which are also associated with mortality; (3) its value as an indicator of two major combustion sources, buildings and traffic; and (4) its having the highest precision in excess mortality risk estimates among the three pollutants examined in this analysis. Both NYS and NYC have climate change mitigation plans to reduce greenhouse gas emissions drastically with goals set for 2030 and 2050 [57, 58], and NO_2 will be the most robust indicator of reducing combustion and associated health benefits, especially in NYC.

Although the large population size of NYC allowed this time-series analysis to estimate the short-term risk estimates in segments of the long study period, the analysis had several limitations. First, this analysis was limited in scope to overall non-external cause mortality, excluding investigation of specific individual causes of death. The primary objective of this analysis was to address uncertainties associated with the concentration-response functions being used in health impact assessment for future policy planning, which generally focuses on non-external cause mortality. An expanded analysis of major specific underlying causes of deaths (e.g., cardiovascular, respiratory, etc.) would be useful for understanding causal mechanisms, which we are conducting separately. Second, we did not consider the chemical constituents of $PM_{2.5}$, as some other studies have done. The every-3rd-day sampling frequency of $PM_{2.5}$ and its late start year (2003) would further weaken statistical power to characterize any changes in the short-term air pollution effects. We are studying $PM_{2.5}$ constituents separately for other research questions. Third, changes in the distribution of age of death and underlying demographics of the city's population during the study period could have also impacted the short-term relationship between air pollution and mortality over the 30-year period. We characterized the breakdown of deaths by age group and major cause in the supplemental material, but we did not conduct time-series analysis by age group nor by cause because the size and precision of the estimated risks for all deaths in the five-year time windows did not support further stratification with sufficient statistical power. This lack of stratification limits the interpretation of our findings. However, we are conducting a separate analysis by race/ethnicity, which is often found to be an important effect modifier of air pollution effects. Finally, the applicability of our findings may be limited to other cities that have similar emission sources and compositions (traffic and buildings). However, in the context of climate change mitigation, most major U.S. and European cities face the same challenge of reducing fossil fuel combustion, and our approach and findings, if not the numerical results, will be useful in both prioritizing and evaluating efforts.

Conclusions

Our analysis of changes in the short-term effects of three regulated air pollutants over extended periods (30 years for NO_2 and O_3 and 20 years for $PM_{2.5}$) in New York City

found that estimated risks fluctuated but did not show increasing or decreasing trends. Substantial declines in the levels of NO_2 and $PM_{2.5}$ during the study period with unchanged PER for NO_2 and $PM_{2.5}$ imply a decline in attributable excess deaths. Therefore, our findings also demonstrate the benefits of emission regulation policies. Further reductions in NO_2 and $PM_{2.5}$ levels will lead to imprecision in assessment of risk estimates, particularly for $PM_{2.5}$, for a typical length of time-series studies (e.g., five years). However, given the relatively unchanged risk estimates, the average estimates over the study period should suffice as input to health impact assessment tools for future policy planning. Given the relatively robust risk estimates for NO_2 and NO_2 contributions to the formation of O_3 and $PM_{2.5}$ constituents, NO_2 will be a good progress indicator of greenhouse gas reduction policy goals set by New York City and State in the coming decade.

Abbreviations

COPD	chronic obstructive pulmonary disease
CR	concentration-response
DLNM	distributed lag non-linear model
ED	emergency department
HIA	health impact assessment
IQR	interquartile range
NO_2	nitrogen dioxide
NYC	New York City
NYS	New York State
O_3	ozone
PER	percent excess risk
PM	particulate matter
$PM_{2.5}$	particulate matter 2.5 microns in size
TRI	Total Risk ndex
U.S.	United States

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12940-025-01171-w.

Supplementary Material 1

Acknowledgements

We thank our NYSERDA project officer Dr. Ellen Burkhard. Any opinions expressed in this article do not necessarily reflect those of NYSERDA or the State of New York.

Author contributions

R.G. was lead data analyst and author for the manuscript. A.S.C also performed some data analysis and contributed to the manuscript. K.I. produced some figures and contributed to the manuscript. M.P. prepared the pollutant data and edited the manuscript. S.J. provided guidance on policy impacts of pollution trends and edited the manuscript. All authors read and approved the final manuscript.

Funding

This work was supported by New York State Energy Research and Development Authority (NYSERDA) agreement number 156225 and the City of New York tax levy funds.

Data availability

Air pollution and weather data that we used in this analysis are publicly available from the sources that we cited. Mortality data that we analyzed were obtained from the Office of Vital Statistics, New York City Department of Health and Mental Hygiene under our approved Institutional Review Board protocol for this study, but the Data Use Agreement does not allow us to distribute the data. However, researchers can request research-oriented mortality data from the Office of Vital Statistics. Information on requesting research-oriented data can be found here: https://www.nyc.gov/site/doh/data/data-sets/vital-statistics-data.page.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of NYC Department of Health and Mental Hygiene.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

Received: 30 December 2024 / Accepted: 21 March 2025
Published online: 13 June 2025

References

1. Brauer M, Freedman G, Frostad J, et al. Ambient air pollution exposure Estimation for the global burden of disease 2013. Environ Sci Technol. 2016;50(1):79–88. https://doi.org/10.1021/acs.est.5b03709
2. Cohen AJ, Brauer M, Burnett R, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the global burden of diseases study 2015. Lancet. 2017;389(10082):1907–18. https://doi.org/10.1016/s0140-6736(17)30505-6.
3. EPA US. Environmental benefits mapping and analysis program - community edition (BenMAP-CE). 2024.
4. EPA US. Co-benefits risk assessment health impacts screening and mapping tool (COBRA). 2024.
5. Kheirbek I, Haney J, Douglas S, Ito K, Caputo S Jr., Matte T. The public health benefits of reducing fine particulate matter through conversion to cleaner heating fuels in New York City. Environ Sci Technol. 2014;48(23):13573–82. https://doi.org/10.1021/es503587p
6. Kheirbek I, Haney J, Douglas S, Ito K, Matte T. The contribution of motor vehicle emissions to ambient fine particulate matter public health impacts in New York City: a health burden assessment. Environ Health. 2016;15(1):89. https://doi.org/10.1186/s12940-016-0172-6
7. Johnson S, Haney J, Cairone L, Huskey C, Kheirbek I. Assessing air quality and public health benefits of New York City's climate action plans. Environ Sci Technol. 2020;54(16):9804–13. https://doi.org/10.1021/acs.est.0c00694
8. Shukla K, Seppanen C, Naess B et al. Zip code-level estimation of air quality and health risk due to particulate matter pollution in New York City. Environ Sci Technol. 2022;56(11):7119–30. https://doi.org/10.1021/acs.est.1c07325
9. Pitiranggon M, Johnson S, Haney J, Eisl H, Ito K. Long-term trends in local and transported PM2.5 pollution in New York City. Atmos Environ. 2021;248(S5):118238. https://doi.org/10.1016/j.atmosenv.2021.118238.
10. Clancy L, Goodman P, Sinclair H, Dockery DW. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. Lancet. 2002;360(9341):1210–4. https://doi.org/10.1016/S0140-6736(02)11281-5.
11. Hedley AJ, Wong CM, Thach TQ, Ma S, Lam TH, Anderson HR. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. Lancet. 2002;360(9346):1646–52. https://doi.org/10.1016/s0140-6736(02)11612-6.
12. Burns J, Boogaard H, Polus S, et al. Interventions to reduce ambient air pollution and their effects on health: an abridged Cochrane systematic review. Environ Int. 2020;135:105400. https://doi.org/10.1016/j.envint.2019.105400.
13. Shin HH, Stieb D, Burnett R, Takahara G, Jessiman B. Tracking National and regional spatial-temporal mortality risk associated with NO2 concentrations in Canada: A bayesian hierarchical two-level model. Risk Anal. 2012;32(3):513–30. https://doi.org/10.1111/j.1539-6924.2011.01684.x.
14. Choi Y, Kim H, Lee JT. Temporal variability of short term effects of PM. Sci Total Environ. 2018;644:122–8. https://doi.org/10.1016/j.scitotenv.2018.06.275.

15. Ye D, Klein M, Mulholland JA, et al. Estimating acute cardiovascular effects of ambient PM. Environ Health Perspect. 2018;126(2):027007. https://doi.org/10.1289/EHP2182.
16. Tzima K, Analitis A, Katsouyanni K, Samoli E. Has the risk of mortality related to short-term exposure to particles changed over the past years in Athens, Greece? Environ Int. 2018;113:306–12. https://doi.org/10.1016/j.envint.2018.01.002.
17. Meng YY, Yue D, Molitor J, Chen X, Su JG, Jerrett M. Reductions in NO. Environ Res. 2022;213:113600. https://doi.org/10.1016/j.envres.2022.113600.
18. Nishikawa H, Seposo XT, Madaniyazi L, et al. Long-term trends in mortality risk associated with short-term exposure to air pollution in 10 Japanese cities between 1977 and 2015. Environ Res. 2023;219:115108. https://doi.org/10.1016/j.envres.2022.115108.
19. Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 US urban communities, 1987–2000. JAMA. 2004;292(19):2372–8. https://doi.org/10.1001/jama.292.19.2372.
20. De Leon SF, Thurston GD, Ito K. Contribution of respiratory disease to nonrespiratory mortality associations with air pollution. Am J Respir Crit Care Med. 2003;167(8):1117–23. https://doi.org/10.1164/rccm.200205-409OC
21. Ito K, De Leon SF, Lippmann M. Associations between Ozone and daily mortality: analysis and meta-analysis. Epidemiol. 2005;16(4):446–57. https://doi.org/10.1097/01.ede.0000165821.90114.7f.
22. Ito K, Mathes R, Ross Z, Nádas A, Thurston G, Matte T. Fine particulate matter constituents associated with cardiovascular hospitalizations and mortality in New York City. Environ Health Perspect. 2011;119(4):467–73. https://doi.org/10.1289/ehp.1002667.
23. Lippmann M, Chen LC, Gordon T, Ito K, Thurston GD. National particle component toxicity (NPACT) initiative: integrated epidemiologic and toxicologic studies of the health effects of particulate matter components. Res Rep Health Eff Inst. 2013;(177):5–13.
24. Rattigan OV, Civerolo KL, Felton HD, Schwab JJ, Demerjian KL. Long term trends in New York: PM2.5 mass and particle components. Aerosol Air Qual Res. 2016;16(5):1191–205.
25. Blanchard CL, Shaw SL, Edgerton ES, Schwab JJ. Emission influences on air pollutant concentrations in New York State: II. PM2.5 organic and elemental carbon constituents. Atmos Environ: X. 2019;3:100039. https://doi.org/10.1016/j.aeaoa.2019.100039
26. Chen Y, Rich DQ, Masiol M, Hopke PK. Changes in ambient air pollutants in New York State from 2005 to 2019: effects of policy implementations and economic and technological changes. Atmos Environ. 2023;311:119996. https://doi.org/10.1016/j.atmosenv.2023.119996
27. Masiol M, Squizzato S, Rich DQ, Hopke PK. Long-term trends (2005–2016) of source apportioned PM2.5 across New York State. Atmos Environ. 2019;201:110–120. https://doi.org/10.1016/j.atmosenv.2018.12.038.
28. U.S. Environmental Protection Agency Air Quality System. Accessed April 23. 2023. https://www.epa.gov/outdoor-air-quality-data/download-daily-data
29. Schwartz J. The distributed lag between air pollution and daily deaths. Epidemiology. 2000;11(3).
30. Ito K, De Leon SF, Lippmann M. Associations between ozone and daily mortality: analysis and meta-analysis. Epidemiology. 2005;16(4):446–57.
31. Ito K, De Leon SF, Lippmann MCFJ. Associations between ozone and daily mortality: analysis and meta-analysis. Epidemiology. 2005;16(4):446–57.
32. Wood SN. Generalized additive models: an introduction with R. Hall/CRC; 2006.
33. Gasparrini A, Armstrong B, Kenward MG. Distributed lag non-linear models. Stat Med. 2010;29. https://doi.org/10.1002/sim.3940.
34. U.S. EPA. Integrated Science Assessment (ISA) for ozone and related photochemical oxidants (final report, Apr 2020). U.S. Environmental Protection Agency, Washington, DC, EPA/600/r-20/012, 2020.
35. U.S. EPA. Integrated Science Assessment (ISA) for oxides of nitrogen– health criteria (final report. Jan 2016). U.S. Environmental Protection Agency, Washington, DC, EPA/600/r-15/068, 2016.
36. U.S. EPA. Integrated Science Assessment (ISA) for particulate matter (final report. Dec 2019). U.S. Environmental Protection Agency, Washington, DC, EPA/600/r-19/188, 2019.
37. Davalos AD, Luben TJ, Herring AH, Sacks JD. Current approaches used in epidemiologic studies to examine short-term multipollutant air pollution exposures. Ann Epidemiol. 2017;27(2):145–153.e1. https://doi.org/10.1016/j.annepidem.2016.11.016.
38. Li W SK, Huynh M, Castro A, Gurr D, Kelley D, Kennedy J, et al. Summary of vital statistics, 2015. New York, NY: New York City Department of Health and Mental Hygiene, Bureau of Vital Statistics, 2017.
39. Madsen A, Thihalolipavan S, Maduro G, et al. An intervention to improve cause-of-death reporting in New York City hospitals, 2009–2010. Prev Chronic Dis. 2012;9:E157. https://doi.org/10.5888/pcd9.120071.
40. Ong P, Lovasi GS, Madsen A, Van Wye G, Demmer RT. Evaluating the effectiveness of New York City health policy initiatives in reducing cardiovascular disease mortality, 1990–2011. Am J Epidemiol. 2017;186(5):555–63. https://doi.org/10.1093/aje/kwx134.
41. Zhang W, Lin S, Hopke PK, et al. Triggering of cardiovascular hospital admissions by fine particle concentrations in New York State: before, during, and after implementation of multiple environmental policies and a recession. Environ Pollut. 2018;242(Pt B):1404–16. https://doi.org/10.1016/j.envpol.2018.08.030.
42. Yount CS, Utell MJ, Hopke PK, et al. Triggering of ST-elevation myocardial infarction by ultrafine particles in New York: changes following tier 3 vehicle introduction. Environ Res. 2023;216(Pt 1):114445. https://doi.org/10.1016/j.envres.2022.114445.
43. Croft DP, Utell MJ, Liu H, et al. Change in rate of healthcare encounters for respiratory infection from air pollution exposure after improved vehicle emissions standards in New York State. Air Qual Atmos Health. 2024;17(6):1267–80. https://doi.org/10.1007/s11869-024-01505-6
44. Lin S, Xue Y, Thandra S, et al. PM(2.5) and its components and respiratory disease healthcare encounters - unanticipated increased exposure-response relationships in recent years after environmental policies. Environ Pollut. 2024;360:124585. https://doi.org/10.1016/j.envpol.2024.124585
45. Rich DQ, Zhang W, Lin S, et al. Triggering of cardiovascular hospital admissions by source specific fine particle concentrations in urban centers of New York State. Environ Int. 2019;126:387–94. https://doi.org/10.1016/j.envint.2019.02.018.
46. Schwarz M, Peters A, Stafoggia M, et al. Temporal variations in the short-term effects of ambient air pollution on cardiovascular and respiratory mortality: A pooled analysis of 380 urban areas over a 22-year period. Lancet Planet Health. 2024;8(9):e657–65. https://doi.org/10.1016/S2542-5196(24)00168-2.
47. U.S. EPA. Supplement to the 2019 Integrated Science Assessment for particulate matter. (final report, 2022). U.S. Environmental Protection Agency, Washington, DC, EPA/635/r-22/028, 2022.
48. U.S. EPA. Integrated Science Assessment (ISA) for ozone and related photochemical oxidants (final report, Feb 2013). U.S. Environmental Protection Agency, Washington, DC, EPA/600/r-10/076f, 2013.
49. Orellano P, Reynoso J, Quaranta N, Bardach A, Ciapponi A. Short-term exposure to particulate matter (PM10 and PM2.5), nitrogen dioxide (NO2), and ozone (O3) and all-cause and cause-specific mortality: Systematic review and meta-analysis. Environ Int. 2020;142:105876. https://doi.org/10.1016/j.envint.2020.105876.
50. Wei Y, Wang Y, Wu X, et al. Causal effects of air pollution on mortality rate in Massachusetts. Am J Epidemiol. 2020;189(11):1316–23. https://doi.org/10.1093/aje/kwaa098.
51. Mills IC, Atkinson RW, Kang S, Walton H, Anderson HR. Quantitative systematic review of the associations between short-term exposure to nitrogen dioxide and mortality and hospital admissions. BMJ Open. 2015;5(5):e006946. https://doi.org/10.1136/bmjopen-2014-006946.
52. Wang M, Li H, Huang S, et al. Short-term exposure to nitrogen dioxide and mortality: A systematic review and meta-analysis. Environ Res. 2021;202:111766. https://doi.org/10.1016/j.envres.2021.111766.
53. Mills IC, Atkinson RW, Anderson HR, Maynard RL, Strachan DP. Distinguishing the associations between daily mortality and hospital admissions and nitrogen dioxide from those of particulate matter: A systematic review and meta-analysis. BMJ Open. 2016;6(7):e010751. https://doi.org/10.1136/bmjopen-2015-010751.
54. Meng X, Liu C, Chen R, et al. Short term associations of ambient nitrogen dioxide with daily total, cardiovascular, and respiratory mortality: multilocation analysis in 398 cities. BMJ. 2021;372:n534. https://doi.org/10.1136/bmj.n534.
55. Environmental Protection Agency US. 2021. Estimating PM2.5- and ozone-attributable health benefits. Technical support document (TSD) for the final revised cross-State air pollution rule update for the 2008 ozone season NAAQS. Docket id no. EPA-hq-oar-2020-0272. Office of Air and Radiation Research Triangle Park, North Carolina March 2021.
56. Pozzer A, Anenberg SC, Dey S, Haines A, Lelieveld J, Chowdhury S. Mortality attributable to ambient air pollution: A review of global estimates. Geohealth. 2023;7(1):e2022GH000711. https://doi.org/10.1029/2022GH000711.
57. NYS. New York State, the climate leadership and community protection act (Climate Act), 2019. Legislation article 75 climate change, environmental

conservation (env) Chap. 43-b. Sections 75–0107, statewide greenhouse gas emissions limits. https://www.Nysenate.Gov/legislation/laws/env/75-0107

58. The City of New York. New York City's roadmap to 80 X 50. 2014. The New York City Mayor's Office of Sustainability. https://www.Nyc.Gov/assets/sustainability/downloads/pdf/publications/new%20york%20city's%20roadmap%20to%2080%20x%2050_final.Pdf

Publisher's note