Journal of Hazardous Materials 471 (2024) 134317

Contents lists available at ScienceDirect

Journal of Hazardous Materials

journal homepage: www.elsevier.com/locate/jhazmat

Research Article

Hidden danger: The long-term effect of ultrafine particles on mortality and its sociodemographic disparities in New York State

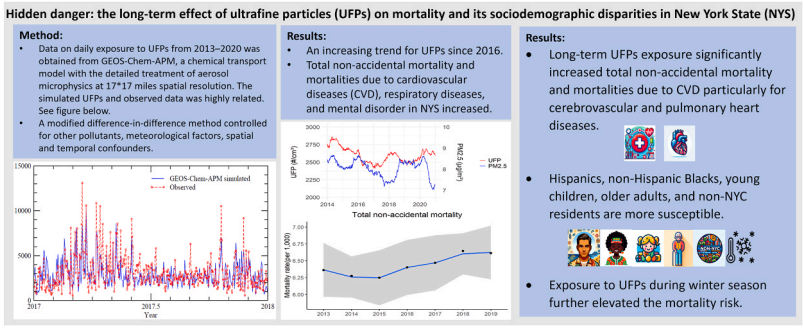
Quan Qi [a], Fangqun Yu [b], Arshad A. Nair [b], Sam S.S. Lau [c,d], Gan Luo [b], Imran Mithu [e], Wangjian Zhang [f], Sean Li [g], Shao Lin [h,i,*]

[a] Department of Economics, University at Albany, State University of New York, Albany, NY, USA
[b] Atmospheric Sciences Research Center, University at Albany, State University of New York, Albany, NY, USA
[c] Research Centre for Environment and Human Health & College of International Education, School of Continuing Education, Hong Kong Baptist University, Hong Kong, China
[d] Institute of Bioresource and Agriculture, Hong Kong Baptist University, Hong Kong, China
[e] Community, Environment and Policy Division, Mel and Enid Zuckerman College of Public Health, University of Arizona, Tucson, AZ, USA
[f] Department of Medical Statistics, School of Public Health, Sun Yat-sen University, Guangzhou, China
[g] Rausser College of Natural Resources, University of California, Berkeley, CA, USA
[h] Department of Epidemiology and Biostatistics, School of Public Health, University at Albany, State University of New York, Rensselaer, NY, USA
[i] Department of Environmental Health Sciences, School of Public Health, University at Albany, State University of New York, Rensselaer, NY, USA

HIGHLIGHTS

- Long-term UFPs exposure significantly increased total non-accidental mortality.
- Mortalities for cardiovascular diseases were associated with UFP exposure.
- Hispanics and non-Hispanic Blacks experienced higher UFP-related mortalities.
- Young children, older adults, and non-NYC residents had higher UFP-Mortality risks.
- Exposure to UFPs during winter season further elevated the mortality risk.

GRAPHICAL ABSTRACT



ARTICLE INFO

Keywords:
Ultrafine particles
Total non-accidental and cause-specific mortality
Long-term effect
Demographic disparity
Difference-in-difference

ABSTRACT

Although previous studies have shown increased health risks of particulate matters, few have evaluated the long-term health impacts of ultrafine particles (UFPs or $PM_{0.1}$, $\leq$ 0.1 μm in diameter). This study assessed the association between long-term exposure to UFPs and mortality in New York State (NYS), including total non-accidental and cause-specific mortalities, sociodemographic disparities and seasonal trends. Collecting data from a comprehensive chemical transport model and NYS Vital Records, we used the interquartile range (IQR) and high-level UFPs (≥75 % percentile) as indicators to link with mortalities. Our modified difference-in-difference model controlled for other pollutants, meteorological factors, spatial and temporal confounders. The findings indicate that long-term UFPs exposure significantly increases the risk of non-accidental mortality

* Correspondence to: Department of Environmental Health Sciences, Associate Director of Global Health Research, School of Public Health, University at Albany, State University of New York, One University Place, Rm 212d, Rensselaer, NY 12144, USA.
E-mail address: slin@albany.edu (S. Lin).

https://doi.org/10.1016/j.jhazmat.2024.134317
Received 22 December 2023; Received in revised form 9 April 2024; Accepted 14 April 2024
Available online 15 April 2024

(RR=1.10, 95 % CI: 1.05, 1.17), cardiovascular mortality (RR=1.11, 95 % CI: 1.05, 1.18) particularly for cerebrovascular (RR=1.21, 95 % CI: 1.10, 1.35) and pulmonary heart diseases (RR=1.33, 95 % CI: 1.13, 1.57), and respiratory mortality (borderline significance, RR=1.09, 95 % CI: 1.00, 1.18). Hispanics (RR=1.13, 95 % CI: 1.00, 1.29) and non-Hispanic Blacks (RR=1.40, 95 % CI: 1.16, 1.68) experienced significantly higher mortality risk after exposure to UFPs, compared to non-Hispanic Whites. Children under five, older adults, non-NYC residents, and winter seasons are more susceptible to UFPs' effects.

1. Introduction

Despite a significant reduction in air pollution, 100,000 people die each year in the United States (US) and 6.7 million worldwide due to anthropogenic emissions of air pollutants [1,2] Short-term exposure to PM_{10} and $PM_{2.5}$ is significantly associated with all-cause mortality and results in specific mortality. [3–6] It is therefore important to consider the adverse effect of long-term exposure to air pollution as residential exposure to these pollutants occurs on a daily and is cumulative. Prior studies have shown the adverse health effects of a long-term exposure to PM_{10} and $PM_{2.5}$. [7–9] However, socioeconomic status (SES) could modify the effect of exposure to particulate matter. [10,11] In addition, subcomponents of $PM_{2.5}$ that are smaller and present in large numbers in the air pose greater health risks than $PM_{2.5.}$ [12] However, the health effects of ultrafine particle (UFP, or $PM_{0.1}$) exposure are not yet well understood, although in recent years there are increasing numbers of research studies [13–15].

UFPs are airborne particles less than 100 nm in aerodynamic diameter. [16] There are multiple sources of UFPs, including natural gas combustion, wood burning, food cooking, diesel/gasoline, motor oil burning, sea spray, shipping oil burning, and product created by nanotechnology. [17,18] Toxicological research has demonstrated that exposure to UFPs could cause autonomic modulation of the heart, atherosclerotic lesions, functional deterioration of the heart, lung inflammation, and systemic inflammation. [19,20] UFPs can directly reach the brain through the olfactory bulb and easily bypass the blood-brain barrier, although some controversies exist. [21] Furthermore, toxicological studies in human subjects have proved that UFPs are responsible for cardiac changes in young adults, increased heart rate, altered peripheral leukocyte distribution, and purine oxidation of DNA. [22–24] A number of epidemiological studies found that morbidities of cardiovascular diseases (CVD) and respiratory diseases were associated with short-term exposure to UFPs. [14,25] Some studies assessed the effects of long-term exposure to UFPs and found that increased risks of CVD, such as chronic heart failure, hypertension, and myocardial infarction were associated with long-term UFP exposure, [15,26,27] while other studies presented mixed findings regarding mortality. [28–30]

Unfortunately, little research has been conducted on exposure to UFPs and their impact on health due to a lack of regulated UPF data. Most studies assessed the short-term effects instead of the long-term impacts, and the majority of the literature evaluated how UFPs affected either total mortality or one specific cause of mortality, rather than both. [14,31] Lastly, there have been a scarcity of prior studies examining variations in the associations between UFP exposure and mortality stratified by demographics and seasonality.

This study aims to fill the knowledge gaps described above by assessing the associations between long-term UFPs exposure and both total non-accidental mortality and cause-specific mortality in New York State (NYS), including mortality due to CVD, respiratory diseases, mental disorders, and nervous system diseases. This study also evaluates the variations in the associations between UFPs exposure and mortality by sociodemographic status and seasonal patterns.

2. Methods

2.1. Study design

A modified difference-in-differences (DID) approach was used to examine the UFP-mortality relationship on a yearly basis. The DID approach has been traditionally used to conduct policy evaluations by comparing outcomes in two groups (with and without the policy) and in two time periods (pre-/post-policy). [32] The modified DID approach in this study extends the comparison beyond two spatial or temporal units. [11] Every sample is compared to itself in a given location at a different time which allows the study design to control for time-invariant spatial confounders and temporal trends using year dummy variables. This modified DID approach has previously been used to estimate the effect of $PM_{2.5}$ exposure on mortality, [11,33,34] as well as for other environmental factors. [35] This study was approved by the Institutional Review Board at the University at Albany, State University of New York (17 ×189), New York State Bureau of Vital Records (# 2021–0914) and New York City Department of Health and Mental Hygiene.

2.2. Exposure data source and definition

Data on daily exposure to UFPs from 2013–2020 was obtained from GEOS-Chem-APM, a chemical transport model with the detailed treatment of aerosol microphysics necessary for quantifying aerosol properties. [36] Driven by meteorological observations taken by NASA's Global Modeling Assimilation Office (GMAO) and up-to-date emission inventories, [37,38] this model has been extensively applied, developed, and tested using measurements of the atmospheric physical and chemical state and quantifies aerosol properties such as size distribution, numbers, and mass. [36,39–41] Further details on GEOS-Chem-APM and its advantages for quantifying aerosol exposure and use in air pollutant exposure assessment can be found in Nair et al. [42].

The model is run on the North American nested-grid configuration at an hourly temporal resolution and 0.25°× 0.3125° (~17-mile) spatial resolution. Data is output for UFPs, other critical pollutants and meteorological factors over NYS in the near surface layer where UFP exposure occurs. We then aggregated those factors to the county subdivision level on a yearly basis and used two metrics of UFPs in our analyses:1) an interquartile range (*IQR* = 75th percentile – 25th percentile) change; and 2) a dichotomous alternative with the cutoff point as the 75th percentile. County subdivisions in NYS are known as minor civil divisions. They are the primary subcounty governmental or administrative units and have legal boundaries and names as well as governmental functions or administrative purposes specified by State law. Our sample includes a total of 1010 county subdivisions, each averaging 27,015 acres in size and 19,457 residents in population.

2.3. Outcome data source and definition

Mortality data for all of NYS from 2013–2019 was obtained by integrating the Upstate NY and New York City (NYC) Vital Records. Both Upstate NY and NYC data include date of death, age at death, manner of death, cause of death, and demographic information (sex, age, birthplace, race, ethnicity). Upstate NY data includes addresses down to the

ZIP code level, and NYC data includes addresses down to the community district level, which includes 59 community districts that serve local and citywide planning and services, with district sizes ranging from 900–15,000 acres and populations varying from about 50,000 to ≥ 200,000 residents.

We consider annual total non-accidental mortality with ICD 10 codes A00-R99 and subsets of four major cause-specific mortalities: respiratory mortality: J00-J99; CVD: I00-I99; mental disorders: F01-F99; and diseases of nervous system: G00-G99.

2.4. Statistical analysis and potential confounders

We estimate the long-term UFP exposure effect on total mortality and the five cause-specific mortalities at the county subdivision level on a yearly basis using a modified DID method following Wang et al. [11]. For the mortality data in Upstate NY, we geocoded street-level residences and identified the corresponding county subdivision. Where this was not possible due to insufficient address information, we calculated the intersecting areas between ZIP code area and county subdivision polygons, assigning ZIP codes to the subdivision with the largest intersection area. For NYC data, each community district corresponds directly to a county subdivision (referred to as a borough in NYC). NYC consists of five boroughs: Manhattan, Bronx, Brooklyn, Queens, and Staten Island. We matched NYC residents to their respective county subdivisions based on community district data. UFP exposures were estimated at the county subdivision level as the geometric mean weighted by the fractional area of overlap with the model grid. We used a multi-pollutant model to control for multi-pollutants (O_3, NH_3, SO_2), meteorological factors (summer temperature and its standard deviation, winter temperature and its standard deviation, and relative humidity (RH)), and county subdivision/year dummy variables in the model. These variables are potential confounders in this study as they are risk factors of mortality and also related to air pollution according to previous studies [11,43–45]. Our model can be expressed in the following manner:

$$\ln\left(E\left(Y_{c,t}\right)\right) = \beta_0 + \beta_1 UFPs_{c,t} + \beta_2 Z_c + \beta_3 U_t + \beta_4 O_{3c,t} + \beta_5 NH_{3c,t} + \beta_6 SO_{2c,t} + \beta_7 Tsummer_{c,t} + \beta_8 SD\_Tsummer_{c,t} + \beta_9 Twinter_{c,t} + \beta_{10} SD\_Twinter_{c,t} + \beta_{11} RH_{c,t} + \ln(P_{c,t})$$

where $Y_{c,t}$ represents the number of deaths in county subdivision c in year t, Z_c is a dummy variable for each location *c*, capturing geographic confounders consistent over time but distinct across areas, U_t is a time-specific dummy variable accounting for the general temporal confounders across regions, and $\ln(P_c)$ denotes the log-transformed population of each location, serving as the offset term.

To assess whether these effects differ by demographic characteristics, we conducted stratified analysis for sex, age (<5, 5–17, 18–44, 45–64, ≥65 years), and race-ethnicity (Hispanic and Non-Hispanic White, Black, and Others). The stratified analyses by NYC (versus non-NYC) and seasonality were performed to examine the differences in the UFP-mortality relationship.

3. Results

In Fig. 1A, we show the temporal trends of $PM_{2.5}$ and UFPs in NYS from 2013 to 2020 on a moving-average yearly basis. From 2014 to mid-2017, UFP concentrations declined. This was followed by an increase until mid-2018, and a subsequent rise beginning in 2019, whereas $PM_{2.5}$ levels began to decrease from 2019 onwards. Fig. 1B illustrates the monthly variation of UFPs and $PM_{2.5}$, suggesting higher UFPs concentrations from February to April and increased $PM_{2.5}$ concentrations from June to September. Detailed descriptive statistics are shown in Table S1 and Fig. S1.

The temporal trends of the non-accidental mortality rate and other major cause-specific mortality rates in NYS from 2013 to 2019 are illustrated in Fig. 2. The number of non-accidental deaths per 1000 people increased from 6.36 in 2013 to 6.61 in 2019, representing an approximate 4 % increase, of which the rise in CVD mortality accounted for approximately one-third. Additionally, there were slight increases in mortality rates caused by respiratory diseases, mental disorders, and the nervous system from 2013 to 2019.

Tables 1 and 2 illustrate the associations between UFPs and mortality. For total non-accidental mortality (883,725 cases), UFPs were associated with an excess risk (ER_{IQR}) of 3.67 (95 % CI: −0.65, 8.18) and a significant risk ratio (RR) of 1.10 (95 % CI: 1.05, 1.17). SO_2 demonstrated a strong effect with an ER_{IQR} of 5.06 (95 % CI: 1.69, 8.53) and an RR of 1.11 (95 % CI: 1.03, 1.20), while other confounders showed either negative or null impacts (Table 1). In Table 2, CVD mortality was found to have a high RR of 1.11 (95 % CI: 1.05, 1.18), while respiratory diseases showed an RR of 1.09 (95 % CI: 1.00, 1.18) at the bottom line. No significant effects were noted for mortality due to mental disorders or nervous system diseases.

Given that we observed a positive association with CVD overall, we further examined the long-term effects on the major subtypes within this category (Table 3). Table S2 shows the specific subtypes and ICD10 codes. Mortality due to cerebrovascular diseases showed a notable excess risk (ER_{IQR}=16.80, 95 % CI: 7.08, 27.40) and a higher risk ratio (RR=1.21, 95 % CI: 1.10, 1.35). Chronic rheumatic heart diseases presented a high ER_{IQR} of 26.26 (95 % CI: 2.25, 55.92), while pulmonary heart disease had an increased RR of 1.33 (95 % CI: 1.13, 1.57).

Table 4 shows the effects of UFPs on total non-accidental mortality

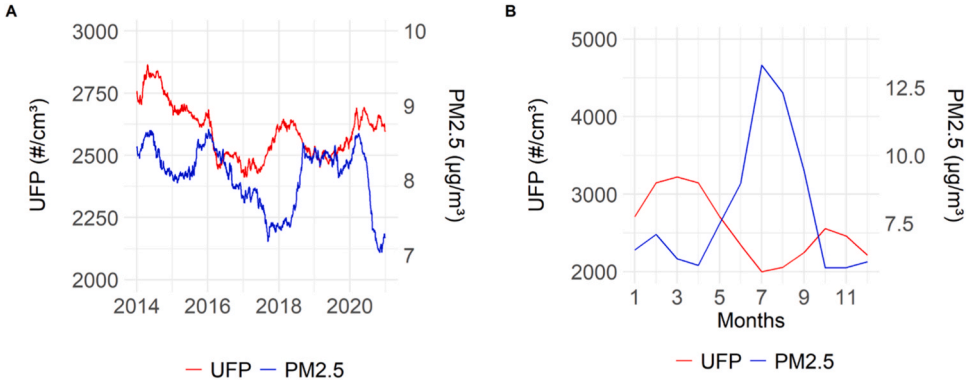


Fig. 1. The annual temporal trend (Panel A) and monthly variation (Panel B) for $PM_{2.5}$ and UFPs across county subdivisions in New York State, 2013–2020. Note: We illustrate the annual temporal trends of UFPs and $PM_{2.5}$ concentrations by employing a moving annual average, calculated as the mean of daily exposure values within a rolling one-year period to mitigate the impact of short-term fluctuations. We calculated the monthly variation by averaging UFP and $PM_{2.5}$ concentrations across all years by months.

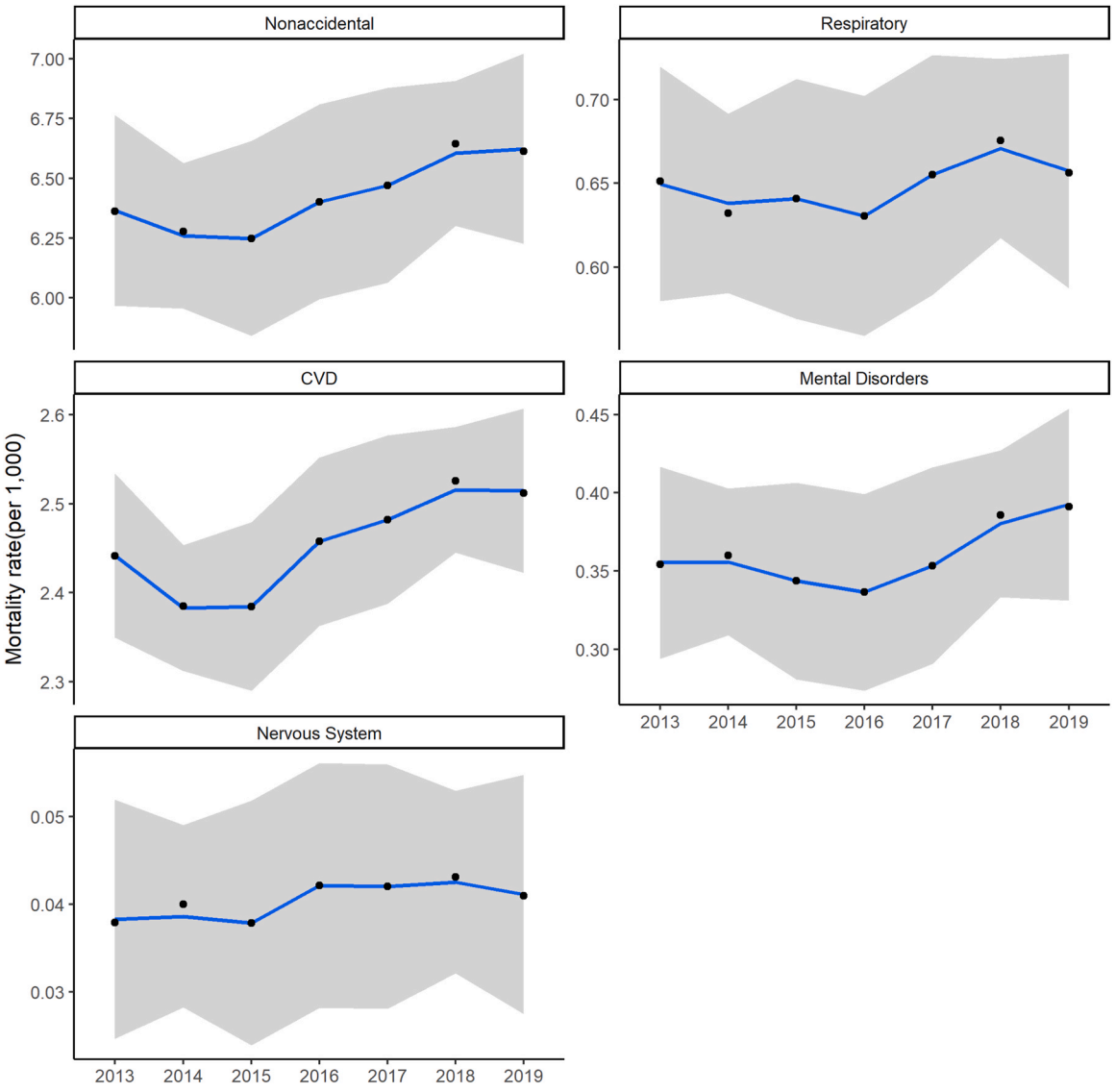


Fig. 2. The temporal trend for non-accidental mortality and cause-specific mortality in New York State, 2013–2019.

Table 1
The association (excess risk and risk ratio) between UFPs, other pollutants, meteorological factors and total non-accidental mortality in New York State, 2013–2019 (Cases=883,725).

Exposure	IQR	ER_{IQR} % (95 % CI)[a]	RR (95 % CI)[b]
UFP	834.0	3.67 (−0.65, 8.18)	1.10 (1.05, 1.17)
O_3	1.9	-3.21 (−5.99, −0.35)	0.98 (0.97, 1.00)
NH_3	0.5	-6.87 (−12.73, −0.63)	0.90 (0.80, 1.01)
SO_2	0.4	5.06 (1.69, 8.53)	1.11 (1.03, 1.20)
Summer temperature	1.6	-2.05 (−6.80, 2.94)	0.99 (0.96, 1.03)
Winter temperature	2.4	-0.32 (−7.84, 7.80)	1.00 (0.96, 1.03)
Relative humidity	4.8	-4.03 (−10.03, 2.37)	0.99 (0.98, 1.01)

[a] ER_{IQR} % (95 % CI) was calculated treating the corresponding exposure as a continuous variable in the DID model.

[b] RR (95 % CI) was calculated treating UFPs as a dichotomous variable with the cutoff point is 75th percentile in the DID model.

among different demographic subgroups and seasons. We found that UFPs increased the mortality rate among females (ER_{IQR}=5.32, 95 % CI: 0.89, 9.94; RR=1.09, 95 % CI: 1.03, 1.15) and males exposed to high levels of UFPs (RR=1.12, 95 % CI: 1.05, 1.19). Hispanics (ER_{IQR}=13.01, 95 % CI: 5.10, 21.51; RR=1.13, 95 % CI: 1.00, 1.29) and non-Hispanic

Table 2
The association [a] (excess risk and risk ratio) between ultrafine particles and total non-accidental mortality, cause-specific mortality in New York State, 2013–2019 (N = 7063).

Mortality	Cases	ER_{IQR} % (95 % CI)[b]	RR (95 % CI)[c]
Total non-accidental	883,725	3.67 (−0.65, 8.18)	1.10 (1.05, 1.17)
Respiratory diseases	89,161	1.65 (−5.01, 8.76)	1.09 (1.00, 1.18)
Cardiovascular diseases	337,436	4.39 (−0.19, 9.19)	1.11 (1.05, 1.18)
Mental disorders	49,546	3.31 (−3.56, 10.68)	1.06 (0.98, 1.14)
Diseases of the nervous system	5578	5.27 (−8.82, 21.52)	0.94 (0.78, 1.13)

[a] NH_3, O_3, SO_2, summer temperature and its deviation, winter temperature and its deviation, relative humidity, time trends that are similar across county subdivision, and time-invariant factors are controlled.

[b] The interquartile range (IQR) for total non-accidental mortality and the major cause-specific mortalities of UFP is 834.0 #/cm^3. ER_{IQR} % (95 % CI) was calculated treating UFPs as a continuous variable in the DID model.

[c] RR(95 % CI) was calculated treating UFPs as a dichotomous variable with the cutoff point is 75th percentile in the DID model.

Table 3
The association [a] (excess risk and risk ratio) between ultrafine particles and mortality due to main CVD subtypes in New York State, 2013–2019.

Mortality	Cases	ER_{IQR} % (95 % CI)[b]	RR (95 % CI)[c]
Cerebrovascular	37,819	16.80 (7.08, 27.40)	1.21 (1.10, 1.35)
Hypertensive	40,624	-3.87 (−11.08, 3.93)	1.03 (0.92, 1.15)
Ischemic heart diseases	189,966	4.10 (−0.71, 9.14)	1.05 (0.98, 1.13)
Acute rheumatic fever	13	NA[d]	NA
Chronic rheumatic heart	933	26.26 (2.25, 55.92)	1.02 (0.81, 1.28)
Pulmonary heart disease	4788	5.15 (−9.02, 21.53)	1.33 (1.13, 1.57)

[a] NH_3, O_3, SO_2, summer temperature and its deviation, winter temperature and its deviation, relative humidity, time trends that are similar across county subdivision, and time-invariant factors are controlled.
[b] The interquartile range (IQR) of UFP is 834.0 #/cm^3. ER_{IQR} % (95 % CI) was calculated treating UFPs as a continuous variable in the DID model.
[c] RR(95 % CI) was calculated treating UFPs as a dichotomous variable with the cutoff point is 75th percentile in the DID model.
[d] NA means that models cannot be fitted due to substantial zero mortality rate in county subdivisions, which is a result of small number of deaths.

Table 4
Effects [a] (excess risk and risk ratio) of ultrafine particles on total non-accidental mortality among different demographic subgroups and seasons in New York State, 2013–2019.

Group	Cases	IQR	ER_{IQR} (%)[b]	RR[c]
Gender				
Male	460,328	834.2	1.94 (−2.78, 6.89)	1.12 (1.05, 1.19)
Female	423,362	833.6	5.32 (0.89, 9.94)	1.09 (1.03, 1.15)
Race/Ethnicity				
Hispanics	81,964	852.4	13.01 (5.10, 21.51)	1.13 (1.00, 1.29)
Non-Hispanic White	488,728	830.1	29.29 (18.43, 41.16)	1.09 (0.98, 1.20)
Non-Hispanic Black	118,164	952.8	-8.72 (−19.17, 3.08)	1.40 (1.16, 1.68)
Non-Hispanic Others	34,462	848.8	4.71 (−6.88, 17.74)	1.20 (1.00, 1.43)
Age				
Age<5	6691	833.1	11.28 (−1.90, 26.24)	1.20 (1.05, 1.38)
5-17	1395	834.1	-8.12 (−23.32, 10.11)	0.65 (0.52, 0.81)
18-44	22,181	833.8	3.87 (−5.52, 14.20)	1.11 (0.98, 1.25)
45-64	149,417	833.3	1.82 (−3.60, 7.55)	1.14 (1.07, 1.22)
Age≥ 65	703,841	833.3	5.29 (0.73, 10.06)	1.11 (1.05, 1.17)
Urbanicity				
NYC	334,416	1301.3	-36.07 (−48.66, −20.39)	0.98 (0.95, 1.01)
Non-NYC	549,309	827.6	21.33 (14.35, 28.73)	1.10 (1.04, 1.17)
Season				
Spring	224,622	1146.5	0.17 (−2.90, 3.34)	0.93 (0.87, 0.98)
Summer	206,697	778.1	-2.05 (−4.50, 0.47)	1.03 (0.97, 1.09)
Fall	215,348	820.9	-3.06 (−5.35, −0.70)	1.05 (0.99, 1.10)
Winter	237,058	860.4	8.18 (3.86, 12.68)	1.04 (1.00, 1.09)

[a] NH_3, O_3, SO_2, summer temperature and its deviation, winter temperature and its deviation, relative humidity, time trends that are similar across county subdivision, and time-invariant factors are controlled.
[b] ER_{IQR} % (95 % CI) was calculated treating UFPs as a continuous variable in the DID model.
[c] RR (95 % CI) was calculated treating UFPs as a dichotomous variable with the cutoff point is 75th percentile in the DID model.

Blacks (RR=1.40, 95 % CI: 1.16, 1.68) showed heightened risks. For children under five, high UFP exposure was most pronounced (RR=1.20, 95 % CI: 1.05, 1.38), while those over 65 also had significant correlations (ER_{IQR}=5.29, 95 % CI: 0.73, 10.06; RR= 1.11, 95 % CI: 1.05, 1.17). Non-NYC residents had increased mortality associated with UFPs (ER_{IQR}=21.33, 95 % CI: 14.35, 28.73; RR= 1.10, 95 % CI: 1.04, 1.17) although the IQR of UFPs in NYC was higher, and winter showed increased effects (ER_{IQR}=8.18, 95 % CI: 3.86, 12.68; RR=1.04, 95 % CI: 1.00, 1.09).

4. Discussion

4.1. Temporal trend for UFPs and mortality

Our study suggested an increase trend in overall UFPs concentration in NYS from 2013–2020, but a declining trend in $PM_{2.5}$ concentration. Similarly, Chen et al. observed an increase of UFPs concentration from 2012–2019 in Rochester, NY [46] and Nair et al. indicated a moderate increase of UFPs from 2017 to 2020 in NYS. [42] Craft et al. also reported that air quality initiatives in NYS have resulted in a reduced $PM_{2.5}$ concentration for decades. [47] On the other hand, studies by Saha et al. [48] in the US and Garcia-Marlès et al. [49] in urban Europe have reported small declines in UFP concentrations over similar time frames. In terms of health outcomes, we found that total non-accidental mortality, respiratory, CVD, and mental disorders mortalities have continuously increased in NYS since 2013. The death rate also showed an increasing trend across the US from 2010–2017 [50] of which, CVD-related mortality accounts for nearly one-third. [51].

4.2. Long-term UFPs exposure and total non-accidental mortality

Long-term UFPs exposure was associated with a 10 % increase in total non-accidental mortality. To date, our study represents one of the few attempts to investigate the relationship between long-term UFPs exposure and mortality by controlling for critical pollutants, meteorological factors and unobservable confounders. A previous study by Ostro et al. [29] constructed a cohort of women from the California Teachers Study from January 2001 to July 2007, and found no significant association between long-term exposure to UFP mass concentrations and all-cause mortality, with a hazard ratio of 1.01 (95 % CI: 0.98, 1.05) per IQR increase. Using data from the US National Health Interview Survey on people aged 18–85, Pond et al. [28] reported that each IQR increment in long-term UFPs exposure was associated with a 2–3 % increase in all-cause mortality; however, they used a single-year estimate of UFPs as a proxy. A recent study by Bouma et al. found that total mortality increased by 1.2 % related to UFPs exposure. [30] The differences between our findings and previous research could be due to the different methodologies used, unique study population or geographic areas, objective or interviewed data collected, different units of UFP measurement, or incompatible time periods.

4.3. Association between UFPs and cause-specific mortality

Significant positive associations were observed between UFPs exposure and mortality rates for CVD (11 % increase) and for respiratory diseases (borderline significance). Previous studies also found the positive relationship between UFPs and CVD mortality. For instance, in a large national cohort study conducted in the US with UFPs measured only using a single-year estimate, an IQR increase in long-term UFPs exposure was associated with a 4 % increase in cardiopulmonary mortality (95 % CI: 1.02, 1.06). [28] Similar to our study, Ostro et al. observed a significant association between long-term UFP exposure and mortality from ischemic heart disease in California, with a hazard ratio of 1.10 (95 % CI: 1.02, 1.18); [29] however, they found no significant increase in respiratory disease mortality related to UFP exposure. Schwarz et al. suggested that UFP exposure was associated with an

increase of 4.46 % for respiratory mortality whereas no clear associations for CVD or natural mortality in three German cities [52].

For specific cause mortality, our study found that high UFP exposure was related to increased mortality risks for cerebrovascular and pulmonary heart diseases. Some epidemiological studies indirectly support our findings by demonstrating a positive relationship between UFP exposure and CVD morbidity. Lin et al. found a 0.1–0.3 % increase in CVD-related hospital admission and a 0.7–1.1 % increase in the subtypes of cerebrovascular disease, hypertension, and ischemic heart diseases.

4.4. Disparities by demographics and seasons

Our study showed variations in elevated non-accidental mortality associated with long-term exposure to high levels of UFPs across various demographic groups, i.e., higher risks were found among Hispanics, non-Hispanic Blacks, children under five, older adults, and non-NYC residents. Since our study is the first study to investigate sociodemographic differences in the relationships between UFPs and mortality, direct comparisons with previous research are not available. Nevertheless, we can indirectly compare our findings with studies on $PM_{2.5}$ in terms of demographic differences in health effects.

Our study found that both genders showed significantly increased RRs after long-term exposure to UFP although the ER_{IQR} for females was much higher than that for males. Villeneuve et al. [53] and Ostro et al. [29] showed significant increases in overall mortality due to $PM_{2.5}$ exposure in Canadian women and female Californian teachers, respectively, while Liu et al. [54] identified a higher susceptibility to $PM_{2.5}$ in Chinese males. However, Puett et al. [55] found no association in the US male cohort. This difference could be attributed to variations in physiology, lifestyle factors, or occupational exposures. More in-depth research is therefore warranted to validate our findings.

We found that Hispanics had an increased risk in mortality rate in relation to an IQR increase in UFPs or high UFPs concentrations and non-Hispanic Blacks experienced the highest increase in mortality following long-term exposure to high levels of UFPs. Similarly, previous studies have reported that minority population may be more susceptible to the adverse effect of air pollution. [56] Factors such as residential segregation, SES, and health care access may all contribute to this disparity. Specifically, Black or low-income populations are more likely to reside in areas with high emissions, pollution, and toxic substances. [57] Mikati et al. found that the $PM_{2.5}$ burden on the average Black population in the US was 1.54 times higher than that of the overall US residents. [58] Another study observed that long-term exposure to increased levels of $PM_{2.5}$ significantly increased overall mortality rates in census tracts with a higher proportion of Black residents in New Jersey. [11] The higher association in the Black/Hispanic subpopulation may be attributed to unmeasured confounding, higher susceptibility, or increased exposure, such as smoking, occupational exposures, or near traffic pollution gradients, etc.

Additionally, our study suggested that children under five and older adults were particularly vulnerable to the effects of UFPs exposure, which is consistent with the understanding that these age groups have weaker immune systems and higher susceptibility to respiratory and CVD illnesses. [59] As people age, many adults often encounter diminishing organ function [60,61] and a rising incidence of coexisting health conditions [62,63], which may hinder their capacity to adapt and respond to airborne contaminants. Due to factors such as a heightened metabolic rate relative to their body weight, a larger surface-to-volume body ratio, intense activity patterns, and a constrained ability to adapt, children are often more susceptible to environmental threats such as airborne contaminants than healthy adults. [64,65] Woodruff et al. found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with overall postneonatal mortality, with an odds ratio of 1.07 (95 % CI: 0.93–1.24). [66] A large previous studies have also reported a significant association between long-term $PM_{2.5}$ exposure and increased mortality in older adults [7,56,67,68].

Moreover, this study indicated that non-NYC residents have greater health risks related to high UFPs exposure when compared to NYC residents. Due to economic and political influences, social infrastructure, environmental protection resources, and healthcare services are likely to be better in highly urbanized areas in comparison to rural areas. Similar to this study, Han et al. found a stronger association between $PM_{2.5}$ and mortality in areas with lower levels of urbanization [45].

This study also reported seasonal differences in the impact of UFP exposure on mortality rates, with the highest excess risk observed during winter months. A possible explanation is that lower temperatures tend to facilitate the formation of a greater number of UFPs and induce a slower atmospheric dispersion rate. [14] Prior studies have identified an inverse relationship between temperature and outdoor UFPs exposure. [14,69] Lin et al. observed that in NYS, the impact of UFP on CVD-related hospital admissions during fall and winter was twice as significant compared to spring and summer. [14] They also found that the influence of UFPs was more pronounced at lower temperatures. On the other hand, the adverse effect of UFPs during cold months may also be attributed to increased indoor exposure to UFPs from heating systems and reduced ventilation. Weichenthal et al. noted that during winter in Canada, the use of electric furnaces and indoor humidifiers significantly elevated the average indoor UFP exposure [69].

4.5. Strengths and limitations

This study is one of the few studies investigating the long-term health effect of UFPs exposure on mortality rather than the short-term effect by most prior research. In addition, our study not only evaluated the total mortality, but also cause-specific mortality in relation to UFP exposure. This study stands as one of the larger-scale analyses conducted in the US to explore the long-term effects of UFPs on mortality, with its substantial dataset encompassing 883,725 deaths in NYS, one of the nation's largest states. It covers a diverse demographic, including individuals of all age groups and a multitude of racial/ethnic populations. Additionally, in our DID model, every sample was compared to itself in a given location at a different time, allowing us to control time-invariant spatial disparities and uniform temporal trends across locations. Furthermore, this study provides new evidence of the modifying effects of demographics, urbanicity, and seasonal factors on the UFPs-mortality relationship. In terms of impacts, our findings point to the possible severest impact of UFP on human mortality and the critical need for monitoring and regulating UFP levels in the US. The disparities by demographics on UFP-mortality relationship may help public health agencies target high-risk populations.

Although our study has certain limitations, we have addressed them accordingly. First, UFP measurement data is usually not available due to the lack of regular monitoring sites for UFPs. The accuracy of our simulated UFP data using the GEOS-Chem-APM model in this study needs further validation, because the resolution scale of 17 miles × 17 miles may be too coarse, resulting in the possibility of exposure misclassification. As described in our previous paper, [14] the GEOS-Chem-APM model applied in our study has been employed in multiple global research studies, and the modeling results have been validated and evaluated against a large set of land-, ship-, aircraft-, and satellite-based measurements. [36,39] However, the larger-scale spatial resolution may indeed cause us to miss highly localized suddenly increased UFP concentrations or be difficult to distinguish near highway from urban background exposures within 17 miles of radius. Further studies are needed to generate higher resolution (down to ~ 1 mile or below) of UFP data which could be used to assess both local sources and background exposure on human health. Due to this concern, we have additionally validated the model simulated UFP against those observed at a monitoring site in the Pinnacle State Park in NYS (R^2 =0.65, see Fig. S2). Furthermore, to validate the association between exposure and health, our previous study used UFP monitoring data from two urban locales (Queens and Rochester) to link with CVD hospitalization data

and showed an increased risk of hospitalizations (0.3 %−0.7 %), with similar results identified using our modeled data (0.1 %−0.9 %), thus showing our findings to be robust. [14] Second, this paper relies solely on mortality data, which reflects the most severe outcomes; however, it is important to note that there are additional factors to consider as other studies have confirmed the positive association between UFPs and multiple morbidities such as CVD and respiratory diseases. [14,70] Third, there could have been a number of unobservable factors that confounded our results. Although we controlled for known confounders, such as temperature, RH, NH_3, O_3, and SO_2, there may be other variables such as the changes of population demographics or composition, SES, housing envelope protection, occupational exposures, and time activity patterns that could influence our results. These variables can be considered time-invariant in the study location over the period of study, which are controlled for by design under the modified DID method. In this method, we compared the UFP-mortality association within the same county subdivision (unit of our analysis) during the study period (2013–2019). We used demographic variables as the proxy indicators for SES because SES variables such as income or smoking etc. are not available through mortality data. In addition, we confirmed that there were no significant changes in the population composition of NYS or the median income within county subdivisions during our study period. All suggest that substantial bias due to the confounding effects caused by demographic changes over time seems unlikely. Finally, we examined the association between UFP and accidental death which is not biologically related to UFP and serves as a negative control. We found an insignificant association between UFP and accidental falls (ICD 10: W19) with the RR of 1.08 (95 % CI: 0.91, 1.29) that further supports our findings.

5. Conclusion

We observed increased temporal trends for both UFPs and total mortality and mortalities due to CVD, respiratory diseases, and mental disorder in NYS. Long-term UFP exposure was associated with increases in total non-accidental mortality and CVD mortality (particulatly for cerebrovascular and pulmonary heart diseases). Hispanics, non-Hispanic Blacks, children under five, older adults, non-NYC residents were more susceptible to the adverse effects of UFPs, and the risks were higher during winter months.

CRediT authorship contribution statement

Wangjian Zhang: Methodology. **Sean Li:** Investigation. **Shao Lin:** Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition, Data curation, Conceptualization. **Quan Qi:** Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Methodology, Formal analysis, Data curation. **Fangqun Yu:** Supervision, Resources, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization. **Arshad A. Nair:** Writing – original draft, Methodology, Data curation. **Sam S. S. Lau:** Writing – review & editing. **Gan Luo:** Methodology. **Imran Mithu:** Writing – original draft.

Environmental Implication

Ultrafine particles (UFPs) are airborne particles less than 100 nm in aerodynamic diameter. The small size allows them to enter the body through lung easily and reach the most distal regions within hours. Compared to larger particles, the large surface area to volume ratio enables UFPs to absorb greater amounts of hazardous metal and organic compounds per unit. Our findings address the severest impact of UFPs on human mortality and the critical need for monitoring and regulating UFP levels in the US. The disparities by demographics on UFP-mortality relationship may help public health agencies target high-risk populations.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Shao Lin and Fangqun Yu reports financial support was provided by New York State Energy Research and Development Authority (NYSERDA) and National Institute on Aging of the National Institutes of Health. If there are other authors, they 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.

Acknowledgements

This work was supported and funded by the New York State Energy Research and Development Authority (NYSERDA, contract no. 137487) and the National Institute on Aging of the National Institutes of Health under award number 1R01AG070949-01A1. We thank New York State Bureau of Vital Records (# 2021–0914) and New York City Department of Health and Mental Hygiene for providing the comprehensive mortality data. We also thank University at Albany Atmospheric Science Research Center's Air Quality Monitoring Products team (ASRC) for making their measurement data publicly available (http://atmoschem.asrc.cestm.albany.edu/~aqm/). This paper is solely the responsibility of the authors, and it is not associated with any of those organizations. We thank Dr. Ola Kalu for her help in initially preparing the results section and Dr. Han Liu for providing useful discussions.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jhazmat.2024.134317.

References

[1] Thakrar, S.K., Balasubramanian, S., Adams, P.J., Azevedo, I.M.L., Muller, N.Z., Pandis, S.N., et al., 2020. Reducing mortality from air pollution in the united states by targeting specific emission sources. Environ Sci Technol Lett 7 (9), 639–645. https://doi.org/10.1021/ACS.ESTLETT.0C00424.
[2] Ambient (outdoor) air pollution. Accessed October 5, 2023. https://www.who.int/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health.
[3] Atkinson, R.W., Kang, S., Anderson, H.R., Mills, I.C., Walton, H.A., 2014. Epidemiological time series studies of PM 2.5 and daily mortality and hospital admissions: a systematic review and meta-analysis. Thorax. https://doi.org/10.1136/thoraxjnl-2013-204492 (Published online).
[4] Janssen, N.A.H., Fischer, P., Marra, M., Ameling, C., Cassee, F.R., 2013. Short-term effects of PM2.5, PM10 and PM2.5–10 on daily mortality in the Netherlands. Sci Total Environ 463-464, 20–26. https://doi.org/10.1016/J.SCITOTENV.2013.05.062.
[5] Shi, L., Zanobetti, A., Kloog, I., Coull, B.A., Koutrakis, P., Melly, S.J., et al., 2016. Low-concentration PM2.5 and mortality: estimating acute and chronic effects in a population-based study. Environ Health Perspect 124 (1), 46. https://doi.org/10.1289/EHP.1409111.
[6] Wang, X., Ran, S., Xia, H., Shi, H., Wu, G., Zhang, Z., et al., 2023. Ambient air pollution associated with incident asthma, subsequent cardiovascular disease and death: a trajectory analysis of a national cohort. J Hazard Mater 460, 132372. https://doi.org/10.1016/J.JHAZMAT.2023.132372.
[7] Pun, V.C., Kazemiparkouhi, F., Manjourides, J., Suh, H.H., 2017. Long-term PM2.5 exposure and respiratory, cancer, and cardiovascular mortality in older US adults. Am J Epidemiol 186 (8), 961. https://doi.org/10.1093/AJE/KWX166.
[8] Vodonos, A., Awad, Y.A., Schwartz, J., 2018. The concentration-response between long-term PM 2.5 exposure and mortality; A meta-regression approach. Environ Res 166, 677–689. https://doi.org/10.1016/j.envres.2018.06.021.
[9] Stafoggia, M., Oftedal, B., Chen, J., Rodopoulou, S., Renzi, M., Atkinson, R.W., et al., 2022. Long-term exposure to low ambient air pollution concentrations and mortality among 28 million people: results from seven large European cohorts within the ELAPSE project. Lancet Planet Heal 6 (1), e9–e18. https://doi.org/10.1016/S2542-5196(21)00277-1.
[10] Kioumourtzoglou, M.A., Schwartz, J., James, P., Dominici, F., Zanobetti, A., 2016. PM2.5 and mortality in 207 US cities: Modification by temperature and city

characteristics. Epidemiology 27 (2), 221. https://doi.org/10.1097/EDE.0000000000000422.
[11] Wang, Y., Kloog, I., Coull, B.A., Kosheleva, A., Zanobetti, A., Schwartz, J.D., 2016. Estimating causal effects of long-term PM2.5 exposure on mortality in New Jersey. Environ Health Perspect 124 (8), 1182–1188. https://doi.org/10.1289/EHP.1409671.
[12] Schraufnagel, D.E., 2020. The health effects of ultrafine particles. Exp Mol Med 52 (3), 311–317. https://doi.org/10.1038/s12276-020-0403-3.
[13] Ohlwein, S., Kappeler, R., Kutlar Joss, M., Künzli, N., Hoffmann, B., 2019. Health effects of ultrafine particles: a systematic literature review update of epidemiological evidence. Int J Public Health 64 (4), 547–559. https://doi.org/10.1007/s00038-019-01202-7.
[14] Lin, S., Ryan, I., Paul, S., Deng, X., Zhang, W., Luo, G., et al., 2022. Particle surface area, ultrafine particle number concentration, and cardiovascular hospitalizations. Environ Pollut 310, 119795. https://doi.org/10.1016/J.ENVPOL.2022.119795.
[15] Bai, L., Chen, H., Hatzopoulou, M., Jerrett, M., Kwong, J.C., Burnett, R.T., et al., 2018. Exposure to ambient ultrafine particles and nitrogen dioxide and incident hypertension and diabetes. Epidemiology 29 (3), 323–332. https://doi.org/10.1097/EDE.0000000000000798.
[16] Li, N., Georas, S., Alexis, N., Fritz, P., Xia, T., Williams, M.A., et al., 2016. A work group report on ultrafine particles (American Academy of Allergy, Asthma & Immunology): why ambient ultrafine and engineered nanoparticles should receive special attention for possible adverse health outcomes in human subjects. J Allergy Clin Immunol 138 (2), 386–396. https://doi.org/10.1016/J.JACI.2016.02.023.
[17] Xue, W., Xue, J., Mousavi, A., Sioutas, C., Kleeman, M.J., 2020. Positive matrix factorization of ultrafine particle mass (PM0.1) at three sites in California. Sci Total Environ 715, 136902. https://doi.org/10.1016/J.SCITOTENV.2020.136902.
[18] Yu, X., Venecek, M., Kumar, A., Hu, J., Tanrikulu, S., Soon, S.T., et al., 2019. Regional sources of airborne ultrafine particle number and mass concentrations in California. Atmos Chem Phys 19 (23), 14677–14702. https://doi.org/10.5194/ACP-19-14677-2019.
[19] Zareba, W., Couderc, J.P., Oberdorster, G., Chalupa, D., Cox, C., Huang, L.S., et al., 2009. ECG parameters and exposure to carbon ultrafine particles in young healthy subjects. Inhal Toxicol 21 (3), 223. https://doi.org/10.1080/08958370802492407.
[20] Araujo, J.A., Barajas, B., Kleinman, M., Wang, X., Bennett, B.J., Gong, K.W., et al., 2008. Ambient particulate pollutants in the ultrafine range promote early atherosclerosis and systemic oxidative stress. Circ Res 102 (5), 589–596. https://doi.org/10.1161/CIRCRESAHA.107.164970.
[21] Wang, B., Feng, W.Y., Wang, M., Shi, J.W., Zhang, F., Ouyang, H., et al., 2007. Transport of intranasally instilled fine Fe2O3 particles into the brain: Micro-distribution, chemical states, and histopathological observation. Biol Trace Elem Res 118 (3), 233–243. https://doi.org/10.1007/S12011-007-0028-6/FIGURES/5.
[22] Samet, J.M., Rappold, A., Graff, D., Cascio, W.E., Berntsen, J.H., Huang, Y.C.T., et al., 2009. Concentrated ambient ultrafine particle exposure induces cardiac changes in young healthy volunteers. Am J Respir Crit Care Med 179 (11), 1034–1042. https://doi.org/10.1164/RCCM.200807-1043OC.
[23] Frampton, M.W., Stewart, J.C., Oberdörster, G., Morrow, P.E., Chalupa, D., Pietropaoli, A.P., et al., 2006. Inhalation of ultrafine particles alters blood leukocyte expression of adhesion molecules in humans. Environ Health Perspect 114 (1), 51. https://doi.org/10.1289/EHP.7962.
[24] Vinzents, P.S., Møller, P., Sørensen, M., Knudsen, L.E., Hertel, O., Jensen, F.P., et al., 2005. Personal exposure to ultrafine particles and oxidative DNA damage. Environ Health Perspect 113 (11), 1485. https://doi.org/10.1289/EHP.7562.
[25] Traboulsi, H., Guerrina, N., Iu, M., Maysinger, D., Ariya, P., Baglole, C.J., 2017. Inhaled pollutants: the molecular scene behind respiratory and systemic diseases associated with ultrafine particulate matter. Int J Mol Sci 18 (2). https://doi.org/10.3390/IJMS18020243.
[26] Bai, L., Weichenthal, S., Kwong, J.C., Burnett, R.T., Hatzopoulou, M., Jerrett, M., et al., 2019. Associations of long-term exposure to ultrafine particles and nitrogen dioxide with increased incidence of congestive heart failure and acute myocardial infarction. Am J Epidemiol 188 (1), 151–159. https://doi.org/10.1093/AJE/KWY194.
[27] Downward, G.S., van Nunen, E.J.H.M., Kerckhoffs, J., Vineis, P., Brunekreef, B., Boer, J.M.A., et al., 2018. Long-term exposure to ultrafine particles and incidence of cardiovascular and cerebrovascular disease in a prospective study of a Dutch cohort. Environ Health Perspect 126 (12). https://doi.org/10.1289/EHP3047.
[28] Pond, Z.A., Saha, P.K., Coleman, C.J., Presto, A.A., Robinson, A.L., Arden Pope, C., 2022. Mortality risk and long-term exposure to ultrafine particles and primary fine particle components in a national U.S. Cohort. Environ Int 167. https://doi.org/10.1016/J.ENVINT.2022.107439.
[29] Ostro, B., Hu, J., Goldberg, D., Reynolds, P., Hertz, A., Bernstein, L., et al., 2015. Associations of mortality with long-term exposures to fine and ultrafine particles, species and sources: results from the California teachers study cohort. Environ Health Perspect 123 (6), 549. https://doi.org/10.1289/EHP.1408565.
[30] Bouma, F., Janssen, N.A., Wesseling, J., van Ratingen, S., Strak, M., Kerckhoffs, J., et al., 2023. Long-term exposure to ultrafine particles and natural and cause-specific mortality. Environ Int 175 (November 2022), 107960. https://doi.org/10.1016/j.envint.2023.107960.
[31] Kettunen, J., Lanki, T., Tiittanen, P., Aalto, P.P., Koskentalo, T., Kulmala, M., et al., 2007. Associations of fine and ultrafine particulate air pollution with stroke mortality in an area of low air pollution levels. Stroke 38, 918–922. https://doi.org/10.1161/01.STR.0000257999.49706.3b.
[32] Card, D., Krueger, A.B., 1994. Minimum wages and employment: a case study of the fast-food industry in New Jersey and Pennsylvania. Am Econ Rev 84 (4), 772–793.
[33] Fan, Z., Li, Y., Wei, J., Chen, G., Wang, R., Xu, R., et al., 2023. Long-term exposure to fine particulate matter and site-specific cancer mortality: a difference-in-differences analysis in Jiangsu province, China. Environ Res 222, 115405. https://doi.org/10.1016/J.ENVRES.2023.115405.
[34] Yitshak-Sade, M., Kloog, I., Zanobetti, A., Schwartz, J.D., 2019. Estimating the causal effect of annual PM2.5 exposure on mortality rates in the Northeastern and mid-Atlantic states. Environ Epidemiol 3 (4). https://doi.org/10.1097/EE9.0000000000000052.
[35] Hu, J., Zhou, M., Qin, M., Tong, S., Hou, Z., Xu, Y., et al., 2022. Long-term exposure to ambient temperature and mortality risk in China: A nationwide study using the difference-in-differences design. Environ Pollut 292, 118392. https://doi.org/10.1016/J.ENVPOL.2021.118392.
[36] Yu, F., Luo, G., 2009. Simulation of particle size distribution with a global aerosol model: contribution of nucleation to aerosol and CCN number concentrations. Atmos Chem Phys 9 (20), 7691–7710. https://doi.org/10.5194/ACP-9-7691-2009.
[37] Guenther, A., Karl, T., Harley, P., Wiedinmyer, C., Palmer, P.I., Geron, C., 2006. Estimates of global terrestrial isoprene emissions using MEGAN (model of emissions of gases and aerosols from nature). Atmos Chem Phys 6 (11), 3181–3210. https://doi.org/10.5194/ACP-6-3181-2006.
[38] Bond, T.C., Bhardwaj, E., Dong, R., Jogani, R., Jung, S., Roden, C., et al., 2007. Historical emissions of black and organic carbon aerosol from energy-related combustion, 1850–2000. Glob Biogeochem Cycles 21 (2). https://doi.org/10.1029/2006GB002840.
[39] Williamson, C.J., Kupc, A., Axisa, D., Bilsback, K.R., Bui, T.P., Campuzano-Jost, P., et al., 2019. A large source of cloud condensation nuclei from new particle formation in the tropics. Nature 574 (7778), 399–403. https://doi.org/10.1038/s41586-019-1638-9.
[40] Nair, A.A., Yu, F., Campuzano-Jost, P., DeMott, P.J., Levin, E.J.T., Jimenez, J.L., et al., 2021. Machine learning uncovers aerosol size information from chemistry and meteorology to quantify potential cloud-forming particles. Geophys Res Lett 48 (21). https://doi.org/10.1029/2021GL094133.
[41] Yu, F., Luo, G., Nair, A.A., Tsigaridis, K., Bauer, S.E., 2022. Use of machine learning to reduce uncertainties in particle number concentration and aerosol indirect radiative forcing predicted by climate models. Geophys Res Lett 49 (16). https://doi.org/10.1029/2022GL098551.
[42] Nair, A.A., Lin, S., Luo, G., Ryan, I., Qi, Q., Deng, X., et al., 2023. Environmental exposure disparities in ultrafine particles and PM2.5 by urbanicity and socio-demographics in New York state, 2013–2020. Environ Res 239, 117246. https://doi.org/10.1016/J.ENVRES.2023.117246.
[43] Wang, S., Wu, G., Du, Z., Wu, W., Ju, X., Yimaer, W., et al., 2023. The causal links between long-term exposure to major PM2.5 components and the burden of tuberculosis in China. Sci Total Environ 870, 161745. https://doi.org/10.1016/J.SCITOTENV.2023.161745.
[44] Renzi, M., Forastiere, F., Schwartz, J., Davoli, M., Michelozzi, P., Stafoggia, M., 2019. Long-term PM10 exposure and cause-specific mortality in the latium region (Italy): a difference-in-differences approach. Environ Health Perspect 127 (6). https://doi.org/10.1289/EHP3759.
[45] Han, C., Xu, R., Gao, C.X., Yu, W., Zhang, Y., Han, K., et al., 2021. Socioeconomic disparity in the association between long-term exposure to PM2.5 and mortality in 2640 Chinese counties. Environ Int 146, 106241. https://doi.org/10.1016/J.ENVINT.2020.106241.
[46] Chen, Y., Masiol, M., Squizzato, S., Chalupa, D.C., Zíková, N., Pokorná, P., et al., 2022. Long-term trends of ultrafine and fine particle number concentrations in New York State: apportioning between emissions and dispersion. Environ Pollut 310, 119797. https://doi.org/10.1016/j.envpol.2022.119797.
[47] Croft, D.P., Utell, M.J., Liu, H., Lin, S., Hopke, P.K., Thurston, S.W., et al., 2024. 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 Heal. https://doi.org/10.1007/s11869-024-01505-6.
[48] Saha, P.K., Hankey, S., Marshall, J.D., Robinson, A.L., Presto, A.A., 2021. High-spatial-resolution estimates of ultrafine particle concentrations across the continental United States. Environ Sci Technol 55, 10320–10331. https://doi.org/10.1021/acs.est.1c03237.
[49] Garcia-Marlès, M., Lara, R., Reche, C., Pérez, N., Tobías, A., Savadkoohi, M., et al., 2024. Inter-annual trends of ultrafine particles in urban Europe. Environ Int. https://doi.org/10.1016/j.envint.2024.108510.
[50] Woolf, S.H., Schoomaker, H., 2019. Life expectancy and mortality rates in the United States, 1959-2017. JAMA - J Am Med Assoc 322 (20), 1996–2016. https://doi.org/10.1001/jama.2019.16932.
[51] Acosta, E., Mehta, N., Myrskylä, M., Ebeling, M., 2022. Drug overdose mortality, for instance, has increased exponentially since. J Gerontol B Psychol Sci Soc Sci 77 (S2), 148–157. https://doi.org/10.1093/geronb/gbac032.
[52] Schwarz, M., Schneider, A., Cyrys, J., Bastian, S., Breitner, S., Peters, A., 2023. Impact of ambient ultrafine particles on cause-specific mortality in three German cities. Am J Respir Crit Care Med 207 (10), 1334–1344. https://doi.org/10.1164/RCCM.202209-1837OC.
[53] Villeneuve, P.J., Weichenthal, S.A., Crouse, D., Miller, A.B., To, T., Martin, R.V., et al., 2015. Long-term exposure to fine particulate matter air pollution and mortality among Canadian women. Epidemiology 26 (4), 536–545. https://doi.org/10.1097/EDE.0000000000000294.
[54] Liu, L., Luo, S., Zhang, Y., Yang, Z., Zhou, P., Mo, S., et al., 2022. Longitudinal impacts of PM2.5 constituents on adult mortality in China. Environ Sci Technol 56 (11), 7224–7233. https://doi.org/10.1021/ACS.EST.1C04152.
[55] Puett, R.C., Hart, J.E., Suh, H., Mittleman, M., Laden, F., 2011. Particulate matter exposures, mortality, and cardiovascular disease in the health professionals follow-

up study. Environ Health Perspect 119 (8), 1130–1135. https://doi.org/10.1289/EHP.1002921.
[56] Franklin, M., Zeka, A., Schwartz, J., 2007. Association between PM2.5 and all-cause and specific-cause mortality in 27 US communities. J Expo Sci Environ Epidemiol 17 (3), 279–287. https://doi.org/10.1038/sj.jes.7500530.
[57] Collins, M.B., Munoz, I., Jaja, J., 2016. The role of local narratives in emerging climate governance linking "toxic outliers" to environmental justice communities. Environ Res Lett 11, 015004. https://doi.org/10.1088/1748-9326/11/1/015004.
[58] Mikati, I., Benson, A.F., Luben, T.J., Sacks, J.D., Richmond-Bryant, J., 2018. Disparities in distribution of particulate matter emission sources by race and poverty status. Am J Public Health 108 (4), 480–485. https://doi.org/10.2105/AJPH.2017.304297.
[59] Huang, W., Cao, J., Tao, Y., Dai, L., Lu, S.E., Hou, B., et al., 2012. Seasonal variation of chemical species associated with short-term mortality effects of PM2.5 in Xi'an, a central city in China. Am J Epidemiol 175 (6), 556. https://doi.org/10.1093/AJE/KWR342.
[60] Gillooly, M., Lamb, D., 1993. Airspace size in lungs of lifelong non-smokers: effect of age and sex. Thorax 48 (1), 39. https://doi.org/10.1136/THX.48.1.39.
[61] Ho, J.C., Chan, K.N., Hu, W.H., Lam, W.K., Zheng, L., Tipoe, G.L., et al., 2001. The effect of aging on nasal mucociliary clearance, beat frequency, and ultrastructure of respiratory cilia. Am J Respir Crit Care Med 163 (4), 983–988. https://doi.org/10.1164/AJRCCM.163.4.9909121.
[62] Gibson, A., Walsh, J., Brown, L.M., 2018. A perfect storm: challenges encountered by family caregivers of persons with Alzheimer's disease during natural disasters. J Gerontol Soc Work 61 (7), 775–789. https://doi.org/10.1080/01634372.2018.1474158.
[63] Rafiey, H., Momtaz, Y.A., Alipour, F., Khankeh, H., Ahmadi, S., Khoshnami, M.S., et al., 2016. Are older people more vulnerable to long-term impacts of disasters? Clin Inter Aging 11, 1791–1795. https://doi.org/10.2147/CIA.S122122.
[64] Stanberry, L.R., Thomson, M.C., James, W., 2018. Prioritizing the needs of children in a changing climate. PLoS Med 15 (7). https://doi.org/10.1371/JOURNAL.PMED.1002627.
[65] ter Mors, S., Hensen, J.L.M., Loomans, M.G.L.C., Boerstra, A.C., 2011. Adaptive thermal comfort in primary school classrooms: creating and validating PMV-based comfort charts. Build Environ 46 (12), 2454–2461. https://doi.org/10.1016/J.BUILDENV.2011.05.025.
[66] Woodruff, T.J., Parker, J.D., Schoendorf, K.C., 2006. Fine particulate matter (PM2.5) air pollution and selected causes of postneonatal infant mortality in California. Environ Health Perspect 114 (5), 786. https://doi.org/10.1289/EHP.8484.
[67] Di, Q., Wang, Y., Zanobetti, A., Wang, Y., Koutrakis, P., Choirat, C., et al., 2017. Air Pollution and Mortality in the Medicare Population. N Engl J Med 376 (26), 2513–2522. https://doi.org/10.1056/nejmoa1702747.
[68] Kazemiparkouhi, F., Honda, T., Eum, K.Do, Wang, B., Manjourides, J., Suh, H.H., 2022. The impact of Long-Term PM2.5 constituents and their sources on specific causes of death in a US Medicare cohort. Environ Int 159, 106988. https://doi.org/10.1016/J.ENVINT.2021.106988.
[69] Weichenthal, S., Dufresne, A., Infante-Rivard, C., Joseph, L., 2006. Indoor ultrafine particle exposures and home heating systems: a cross-sectional survey of Canadian homes during the winter months. J Expo Sci Environ Epidemiol 2007 17 (3), 288–297. https://doi.org/10.1038/sj.jes.7500534.
[70] Wu, A.H., Fruin, S., Larson, T.V., Tseng, C.C., Wu, J., Yang, J., et al., 2021. Association between airport-related ultrafine particles and risk of malignant brain cancer: a multiethnic cohort study. Cancer Res 81, 4360–4369. https://doi.org/10.1158/0008-5472.CAN-21-1138.