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Breakthroughs in Epidemiology

The association between long-term exposure to $PM_{2.5}$ constituents and diabetes incidence and blood glucose levels among World Trade Center Health Program general responders

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Abstract

Exposure to fine particulate matter ($PM_{2.5}$) is associated with cardiometabolic risk among World Trade Center Health Program general responders, but current studies focus mainly on $PM_{2.5}$ mass. We studied the associations between annual source-apportioned $PM_{2.5}$ exposures, and self-reported diabetes or repeated blood glucose measurements among general responders enrolled between 2003 and 2019 (n = 34 764), residing in Tri-state area. We used non-negative matrix factorization to attribute $PM_{2.5}$ component to sources (ie, biomass burning, oil combustion, metal industry, other industries, and motor vehicles). We used multivariable mixed-effect models to estimate the simultaneous associations of the source-apportioned $PM_{2.5}$ exposures with the outcomes. We found (% change [95% CIs]) an interquartile range increase in $PM_{2.5}$ attributed to metal industry sources (0.42 $\mu g/m^3$) to be associated with an 8.35% (1.39%, 15.79%) higher risk of diabetes and a 1.31% (0.80%, 1.82%) increase in glucose levels. Source-specific associations with glucose were modified by sex, showing larger associations with biomass burning (1.08% [0.32%, 1.85%] per 0.44 $\mu g/m^3$) and motor vehicle (1.34% [0.76%, 1.93%] per 0.92 $\mu g/m^3$) pollution among women, and larger associations with oil-combustion (0.68% [0.03%, 1.34%] per 1.74 $\mu g/m^3$) pollution among men. These findings can inform policies and interventions targeting emissions from these specific sources, particularly for general responders with a history of extreme air pollution exposures.

Key words: $PM_{2.5}$chemical components; World Trade Center; diabetes; glucose.

Introduction

Diabetes is a chronic metabolic disorder in which higher-than-optimal blood glucose level is a common early symptom.[1] Both diabetes and high blood glucose are considered leading causes of global burden of diseases and premature death, posing a major threat to global public health.[2] Additionally, diabetes is a major contributor to long-term health complications. It exacerbates endothelial dysfunction, oxidative stress, and abnormal platelet function, thereby increasing the risk for cardiovascular diseases (CVD) such as coronary artery disease, heart failure, and stroke by 2- to 4-fold.[3] Therefore, it is crucial to identify modifiable exposures associated with increased cardiometabolic disease (CMD) risk.

Metabolic diseases develop through a complex interplay of factors, including lifestyle factors—such as physical activity, smoking, diet, and stress[4]—as well as environmental exposures—such as greenness, temperature, and air pollution.[5] Numerous studies have linked fine particulate matter ($PM_{2.5}$) to an elevated risk of developing type 2 diabetes and elevated glucose levels. Nevertheless, the epidemiological evidence is still sparse, with great heterogeneity between regions and studies, partially due to the different composition and sources of $PM_{2.5}$ exposure.[6] Several reviews concluded that higher $PM_{2.5}$ exposure is associated with poorer glycemic outcomes. For example, the pooled effect estimates from 11 studies in high-income countries showed that a 10 $\mu g/m^3$ increase in annual $PM_{2.5}$ exposure was positively associated with diabetes (OR = 1.09, 95% CI, 1.05-1.13).[6] Another review summarizing 17 studies reported that the same increase in $PM_{2.5}$ exposure was associated with an increased fasting blood glucose of 0.23 mmol/L (95% CI, 0.01-0.45).[7]

The composition of $PM_{2.5}$ is complex, and the physical structure, chemical composition, and source of the component mixture are diverse.[8] A few studies have documented the links

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between several components and sources of $PM_{2.5}$ and various cardiometabolic health-related outcomes.[8] A study conducted in 26 U.S. communities reported that particles from industrial combustion sources and traffic may, on average, have greater toxicity and result in higher diabetes admission rates.[9] Another study[10] found that elevated fasting blood glucose levels are associated with $PM_{2.5}$ exposure from coal combustion, industrial sources, and vehicle emissions. However, the reported results are still mixed, and evidence for the combined effects of components appointed to different sources is very limited, especially among vulnerable populations.

The World Trade Center Health Program (WTCHP) general responder cohort (GRC) includes a unique population with a history of exposure to an extreme air pollution event,[11] alongside 23 years of cohort data, including clinical laboratory results and residential information. A 2025 study found several WTC-related exposures to be associated with higher risks of developing diabetes and worsening glucose trajectories among general responders.[12] In addition to the cardiometabolic consequences of WTC-exposures, a 2023 study investigated the associations with later-in-life $PM_{2.5}$ exposure and found an increase of 0.78 mg/dL (95% CI, 0.30-1.26) in blood glucose levels associated with an interquartile range (IQR) increase in $PM_{2.5}$ averaged 6 months before the study visit. These associations were more pronounced among people with diabetes. Glucose increases attributed to air pollution exposure, such as those reported in the study, are often small. However, the continuous and ubiquitous nature of exposure can translate into adverse health outcomes when affecting large populations. Further, higher glucose levels, even within the normoglycemic range, were found to increase the risk of developing type 2 diabetes,[13] cerebrovascular, cardiovascular, and overall mortality.[14,15] To our knowledge, no studies have investigated the diabetes and glycemic risk associated with source-apportioned $PM_{2.5}$ exposure years after the 9/11 attack.

In this study, we examine the simultaneous associations of annual source-apportioned $PM_{2.5}$ exposures with long-term diabetes incidence and blood glucose levels among WTCHP general respondents. Additionally, since several studies reported higher vulnerability and chronic disease risks among women in the WTCHP GRC,[16,17] we also studied the modification of these associations by sex.

Methods

Study population

The study population includes 34 764 WTCHP general responders in the Tri-State area (ie, New York, New Jersey, and Connecticut) from 2003 to 2019. The WTCHP GRC data includes monitoring visits of responders who took part in rescue, recovery, and clean-up tasks following the September 11, 2001, attack on the WTC. Details regarding eligibility criteria are described elsewhere.[18] An exposure assessment questionnaire was administered at the initial monitoring visit. Sociodemographic information was collected at the first visit and address histories were updated at each monitoring visit starting 2012. Clinical chemistry laboratory tests were performed at each monitoring visit. The data includes laboratory results from Mount Sinai, NYU, Stony Brook (SUNY), and the Northwell Clinical Centers of Excellence. A medical questionnaire was administered during all monitoring visits (typically offered annually), including self-reported information on the diagnosis date of various chronic conditions. The responders were asked about numerous conditions (including diabetes) and whether their physician had ever told them they had the condition.

Sub-cohort for diabetes analysis

We constructed a sub-cohort including annual follow-up observations to analyze the long-term associations between the pollutants and diabetes risk. Responders were followed annually until disease occurrence, death, or last documented visit (whichever came first). We began the observation period in 2003 to allow for a potential induction period. Responders reported been diagnosed with diabetes during or prior to this induction period (2.09%) were excluded. Diabetes was assigned to responders based on self-reports or with the presence of at least two blood glucose test results higher than 200 mg/dL.[19] This conservative threshold was chosen to account for the possibility that the responders were not fasting before the blood test, although they were instructed to do so.

Sub-cohort for glucose analysis

We included repeated measurements of blood glucose (n = 121 246) obtained during the monitoring visits from 26 756 members of the WTCHP GRC. We excluded glucose values lower than <50 mg/dL and higher than >600 mg/dL to avoid bias due to outcome outliers (0.41%).

Exposure

We obtained $PM_{2.5}$ mass estimates from prediction models using extreme gradient boosting (XGBoost) and satellite-derived aerosol optical depth modeling to predict daily $PM_{2.5}$ on a 1 × 1-km^2 resolution covering the contiguous United States.[20] We aggregated the daily values to create annual mean predictions. The model is validated using $PM_{2.5}$ data from the U.S. Environmental Protection Agency Air Quality System precomputed daily summary files and has a root mean square error of 3.11 $\mu g/m^3$.

We additionally estimated five source-apportioned $PM_{2.5}$ exposures. Exposures were obtained using annual mean predictions of 14 $PM_{2.5}$ components from national super-learned models (bromine, Br; calcium, Ca; copper, Cu; iron, Fe; potassium, K; nickel, Ni; lead, Pb; silicon, Si; vanadium, V; zinc, Zn; elemental carbon, EC; nitrate, NO_3; organic carbon, OC; and sulfate, SO_4).[21] The models were estimated from validated ensemble machine learning models (SVM) at a 50 × 50-m grid cell level in urban areas and at a 1 × 1-km level in rural areas across the United States.[22] The estimates incorporated predictions from multiple machine-learning models including random forest, stochastic gradient boosting, extreme gradient boosting (XGB), cubist, and K-nearest neighbors models. Predictors included satellite observations, meteorology data and novel land use covariates. The CV R^2 ranged from 0.80 to 0.96 across different constituents. We used non-negative matrix factorization (NMF) to attribute annual estimates of $PM_{2.5}$ chemical components to factors and transformed the factor values into source-apportioned $PM_{2.5}$ exposures.

The best model factorization fit, calculation of the factors, and conversion to source-apportioned $PM_{2.5}$ exposures can be found in Krasnov et al.[23] In brief, we labeled the first source as biomass burning based on high loading of K and Br; the second as oil combustion based on high loading of V and Ni; the third as metal-industry based on high loading of Pb; the fourth as other industries based on high loading of SO_4; and the fifth as motor vehicles and resuspension of dust (MVRD) based on high loadings of EC, Fe, Cu, Ca, and Zn.[24]

Annual exposures were estimated and linked at the residential address level. Address history has been collected in the WTCHP GRC since 2012. Using updated addresses and reported moving dates, we constructed a longitudinal dataset with annual

observations, each linked to the most recently reported geocoded address; for years prior to 2012, we assigned the closest reported address. Among movers (15.9%), if a responder moved mid-year, the average value of both locations was calculated.

Covariates

We adjusted the models for individual characteristics, including age, sex (male and female), race (Black, White, other, and unknown), and education (elementary school, high school, college, other, and unknown). We further adjusted the model for occupation during the 9/11 (protective services yes/no) and the length of time spent on site. Occupation was included as a covariate since high-stress jobs on site has been associated both with WTC exposures (higher air pollution exposure) and an increased CVD risk.[25] The variables representing the length of time on site were selected based on our previous analysis that showed increased glucose levels associated with the number of hours spent on site during September 2001, and an elevated diabetes risk associated with the number of days spent on site during the nine months following the attack (October 2001-June 2002).[12]

We also adjusted the models for census tract-level socioeconomic variables used as a proxy for sociodemographic neighborhood adjustment: percentage below poverty line (annual income < $11 484), percentage of Black population, percentage with no high school diploma, and median household income at the baseline visit. These measures were linked to the census tract corresponding to each responder's residential address. Finally, we adjusted the models for annual average temperature exposure, using temperature estimates from a high-resolution geospatial model.[26]

Statistical analysis

We used mixed-effect Poisson survival with the Anderson–Gill formulation to study the associations with diabetes and generalized additive linear models to assess the associations with repeated measures of log-transformed blood glucose levels. First, we evaluated the associations with $PM_{2.5}$ mass. Then, we evaluated the simultaneous associations with the five source-apportioned exposures using multi-exposure models. To assess the role of sex as a potential modifier, we repeated the models including interaction terms with sex and estimated sex-specific associations in stratified analyses. All models included a random intercept for each responder to account for repeated measures, and penalized spline for the year of testing to control for the time trend.

Sensitivity analysis

About 23% of the responders did not have blood glucose results. Therefore, as a sensitivity analysis, we repeated the glucose outcome models, including inverse probability weights (IPW) to ensure our results are not biased due to selection bias. We estimated the probability of having glucose information using a logistic regression including sociodemographic predictors (age, sex, race, education, and occupation) and census tract-level socioeconomic variables at the baseline visit (percentage below poverty line, percentage of Black population, percentage with no high school diploma, and median household income).

Results

We included 34 764 responders, of whom 12.59% reported having diabetes. The average age in the first year of follow-up was about 47 years, 86.62% were males, and 51.56% were White. The average blood glucose level was 101.99 mg/dL. Overall, 121 246 glucose blood tests were drawn between 2003 and 2019 from 26 747 WTCPH cohort participants (76.9%). Comparing responders with and without blood glucose results available, we found younger age, a larger proportion of racial minorities, lower education levels, and a larger proportion of females among those who had a glucose test. Responders with glucose tests resided in neighborhoods of lower socioeconomic status on average. Finally, diabetes was slightly more common in this group (Table 1).

The average $PM_{2.5}$ exposure was 9.22 $\mu g/m^3$, and the IQR was 4.06. Among the source-apportioned exposures, oil-combustion and industrial $PM_{2.5}$ contributed the highest levels, averaging 1.30 and 1.36 $\mu g/m^3$, respectively. In contrast, $PM_{2.5}$ from metal industry sources was the lowest, averaging 0.36 $\mu g/m^3$ (Table 2). This is also supported by Figure S1, which shows a high correlation between $PM_{2.5}$ mass, oil-combustion sources (r = 0.70), and industrial pollution (r = 0.60). The correlations among the source-apportioned exposures were low to moderate, with the highest correlation observed between metal industry and biomass burning (r = −0.49).

We found an IQR increase in annual $PM_{2.5}$ exposure from metal industry sources (0.42 $\mu g/m^3$) to be associated with higher risk of diabetes (8.35% increase; 95% CI, 1.49%-15.79%) and a 1.31% increase in blood glucose levels (95% CI, 0.80%-1.82%). We did not observe associations with other source-apportioned exposures or total $PM_{2.5}$ mass. The associations with glucose remained very similar after incorporating IPWs, and the overall inference did not change (Table 3).

Several of the observed associations with glucose were modified by sex. The association with total $PM_{2.5}$ was statistically significantly modified by sex (P = .002). However, total $PM_{2.5}$ was not associated with glucose in either of the groups. We found larger associations with biomass burning among women (1.08%; 95% CI, 0.32%-1.85%) compared to men (–0.45%; 95% CI, –1.03% to 0.16%). Similarly, associations with motor vehicle and resuspended dust pollution were stronger among women (1.34%; 95% CI, 0.76%-1.93%) than men (–0.04%; 95% CI, –0.33% to 0.24%). In contrast, oil-combustion pollution was more strongly associated with glucose among men (0.68%; 95% CI, 0.03%-1.34%) compared to women (–0.18%; 95% CI, –1.50% to 1.15%) (Figure 1; Table S1).

The associations with diabetes were not modified by sex in both models. However, the interaction with biomass burning exposure was marginally significant (P value = .050), showing an elevated diabetes risk associated with biomass burning pollution among women (12.89%, 95% CI, 1.03%-26.15%) but not men (−1.40%, 95% CI, −6.83% to 4.33%). All estimates in this stratified analysis presented relatively wide confidence intervals (Table S2).

Discussion

We found elevated diabetes risk and blood glucose levels associated with metal industry sourced $PM_{2.5}$ pollution among WTCHP responders followed for more than two decades. Additionally, our results highlight differential vulnerability affecting glycemic changes among men and women. The associations with biomass burning and motor vehicle sources were larger among women, while the association with oil combustion pollution was stronger among men. These elevated risks were independent of WTC-exposures, occupation in September 2001, and other individual and area-level confounders.

Our study highlights a specific association between $PM_{2.5}$ pollution from metal industry sources and poorer glycemic outcomes. The metal industry represents sources containing Pb, K, and Cu that can be found in building paint, ceramics, pipes, and plumbing

Table 1. Population characteristics 2003-2019 (N = 34 764).

Variable	N (%)/(mean (SD))	People without glucose values (N = 8008)	People with glucose values (N = 26 756)
Sociodemographic characteristics, *n* (%)			
Age at cohort entry, mean (SD)	47.59 (10.15)	49.04 (10.12)	45.66 (9.62)
Race			
Black	3629 (8.69%)	946 (5.84%)	2453 (10.68%)
White	21 540 (51.56%)	8186 (57.66%)	12 370 (53.85%)
Other	4701 (11.25%)	1018 (6.29%)	3468 (15.10%)
Unknown	11 906 (28.50%)	5583 (34.49%)	4680 (20.37%)
Highest education level			
Elementary school	2788 (6.67%)	762 (4.71%)	1877 (8.17%)
High school	8055 (19.28%)	2866 (17.70%)	4835 (21.05%)
College	10 779 (25.80%)	4245 (26.22%)	6046 (26.32%)
Other[a]	17 023 (40.75%)	1472 (9.10%)	838 (3.6%)
Unknown	3131 (7.5%)	6846 (42.28%)	9375 (40.81%)
Sex			
Male	36 186 (86.62%)	14 321 (88.39%)	19 547 (85.09%)
Female	5590 (13.38%)	1879 (11.61%)	3424 (14.91%)
Neighborhood characteristics, mean (SD)			
% Black population	0.88% (1.82%)	0.57% (1.3%)	1.08% (2.02%)
% Under the poverty line	9.42% (9.33%)	6.95% (7.26%)	11.16% (10.25%)
% With no high school education	4.19% (4.27%)	3.14% (3.41%)	4.96% (4.69%)
Median household income	78 996 (34784)	90 571.40 (33684.67)	68 200.63 (30760.23)
Outcomes			
Diabetes, *n* (%)	5149 (12.59%)	99 (0.69%)	253 (1.10%)
Glucose (mmol/L), mean (SD)	(-)	(-)	101.99 (36.33)

[a]Professional school/BA/BS/associate degree.

materials, as well as manufacturing processes, transportation, and coal-fired power plant emissions.[27-30] Similar to our study, a study from Canada[31] found elevated diabetes risks associated with various industrial sources that were not specified. Elevated diabetes risks were also found to be associated with cement[32] and plastic industries.[33] Additionally, a study focusing on occupational metal exposures found higher prevalence rates of diabetes among mining/production and smelting/refining workers compared to office workers.[34] Finally, a study in China found that among various constituents, increases in $PM_{2.5}$ from coal combustion, industrial sources, and vehicle emissions were associated with higher blood glucose.[10] The underlying mechanism of metal exposure in the pathogenesis of glucose elevation and diabetes is not yet fully understood. It was suggested that some metals might damage insulin function (by damaging the pancreatic β-cells), induce hyperglycemia, and increase plasma glucose levels,[35] resulting in diabetes.[36]

Table 2. Summary statistics of exposures derived from the prediction models.

Variable	Mean (SD)	IQR
Total $PM_{2.5}$ ($\mu g/m^3$)	9.22 (2.49)	4.06
Source apportioned $PM_{2.5}$ ($\mu g/m^3$)		
Biomass burning sources	1.08 (0.40)	0.44
Oil combustion sources	1.30 (1.21)	1.74
Metal industry sources	0.36 (0.32)	0.42
Industry sources	1.36 (0.92)	1.35
MVRD sources	1.13 (0.64)	0.92
Mean temperature (°C)	12.76 (2.54)	12.93

Abbreviations: MVRD, motor vehicle and resuspension of dust; $PM_{2.5}$, fine particulate matter.

Although we did not find an increased glycemic risk associated with total $PM_{2.5}$, most current studies did find $PM_{2.5}$ mass as an important risk factor. A 2025 systematic umbrella review found $PM_{2.5}$ to be statistically significantly associated with increased risk of developing type 2 diabetes, reporting an odds ratio of 1.12 (95% CI, 1.09-1.15).[37] A U.S. analysis also showed annual $PM_{2.5}$ exposure to be associated with an increased diabetes risk. The lack of association in our current analysis may possibly be related to the limited variability of $PM_{2.5}$ mass across the Tri-state area, compared to the wider exposure distribution in larger geographic areas. Importantly, we did identify effects of specific $PM_{2.5}$ sources that were masked when pooling sources into the aggregated $PM_{2.5}$ mass measure, highlighting the value of our source-specific approach. Additionally, a previous analysis among WTCHP general responders did show increases in glucose levels associated with $PM_{2.5}$ exposure averaged 6-months before the study visit.[38] The difference in findings likely reflects the distinct exposure windows, as our analysis focused on long-term effects.

Our findings show differential vulnerability to specific pollution sources among men and women. Air pollution has been previously associated with glucose and reported to be stronger among women than men.[39,40] We found women to be more vulnerable to biomass burning and motor vehicle pollution. A study in China also found exposure to biomass fuel to be associated with high fasting blood glucose impairment in women. However, this study did not include a comparison with men.[41] A U.S. study found that living near roadways was associated with higher glucose levels, highlighting larger observed effects women (β = 5.16 mg/dL; 95% CI, 1.48-8.84) compared with men (β = 0.14 mg/dL; 95% CI, −3.04 to 3.33).[42]

Our finding of sex-related differences is consistent with current literature, which shows higher vulnerability and chronic disease risks among women in the WTCHP GRC.[16,17] The increased vulnerability among women may be related to differences in behavior,

Table 3. The association between $PM_{2.5}$ mass and source-apportioned exposures and the outcomes studied.

	Exposure	IQR	Diabetes % change (95% CI)	Glucose (mmol/L) % change (95% CI)	
				No IPW	IPW
Model 1	Total $PM_{2.5}$ ($\mu g/m^3$)	4.06	−6.66% (−16.33; 4.12)	−0.45% (−0.99; 0.09)	−0.50% (−1.03; 0.03)
Model 2	Biomass burning sources	0.45	0.40% (−4.85; 5.95)	−0.21% (−0.56; 0.13)	−0.20% (−0.55; 0.13)
	Oil combustion sources	1.79	4.66% (−6.58; 17.26)	0.54% (−0.05; 1.14)	0.58% (−0.00; 1.16)
	Metal industry sources	0.41	8.35% (1.39; 15.79)	1.31% (0.80; 1.82)	1.25% (0.75; 1.76)
	Industry sources	1.46	5.33% (−3.18; 14.59)	0.36% (−0.05; 0.78)	0.33% (−0.07; 0.74)
	MVRD sources	0.92	5.32% (−0.09; 11.03)	0.17% (−0.09; 0.44)	0.11% (−0.11; 0.40)

Abbreviations: MVRD, motor vehicle and resuspension of dust.
We used mixed effect Poisson survival with the Anderson–Gill formulation to study the associations with diabetes and generalized additive linear models to assess the associations with repeated measures of log-transformed blood glucose levels.
Model 1 assesses the associations with total $PM_{2.5}$ mass, and model 2 simultaneously assesses the associations with the 5 source-apportioned $PM_{2.5}$ exposures. Models were adjusted for age, sex, race, census tract-level socioeconomic variables, mean temperature, year, occupation, and time spent on site during and after the 9/11 attack. We used a random intercept per responder and penalized spline for the year of testing to control for the time trend. Results are represented for IQR increases in exposure.
Because 23% of the responders did not have blood glucose measurements, we performed a sensitivity analysis, where we repeated the glucose outcome models, including inverse probability weights (IPW). We estimated the probability of having glucose information using a logistic regression, adjusting for individual and area-level confounders.

lifestyle, occupation, and physiology.[43] In addition, women may be more vulnerable to air pollution exposure due to pregnancy-related metabolic changes,[44] which can have long-lasting effects. Finally, in our previous study[12] we found consistently higher CMD risk associated with exposures linked to intense stress (ie, witnessing horror and the loss of someone) that may trigger Post-Traumatic Stress Disorder (PTSD) and depression.[45,46] PTSD is a well-established risk factor for CVD,[47] and studies in the WTCHP cohort show that women have a higher prevalence of PTSD and depression than men, which may contribute to their greater vulnerability to CMD[48] and help explain the increased susceptibility observed in our study.

Interestingly, oil combustion pollution was associated with elevated glucose levels among men, but not women. Oil combustion pollution is more prevalent in northeastern states in the U.S.,[49] and specifically in New York City, which uses a substantial amount of oil for space-heating.[50] Oil combustion pollution was found to be associated with other health outcomes, including atherosclerotic CVD mortality[49] and stroke.[51] It was also associated with impaired glucose tolerance among pregnant women.[52] The increased vulnerability observed among men in our study may be related to unique properties of the WTCHP cohort, including occupational factors and differences in exposure patterns.

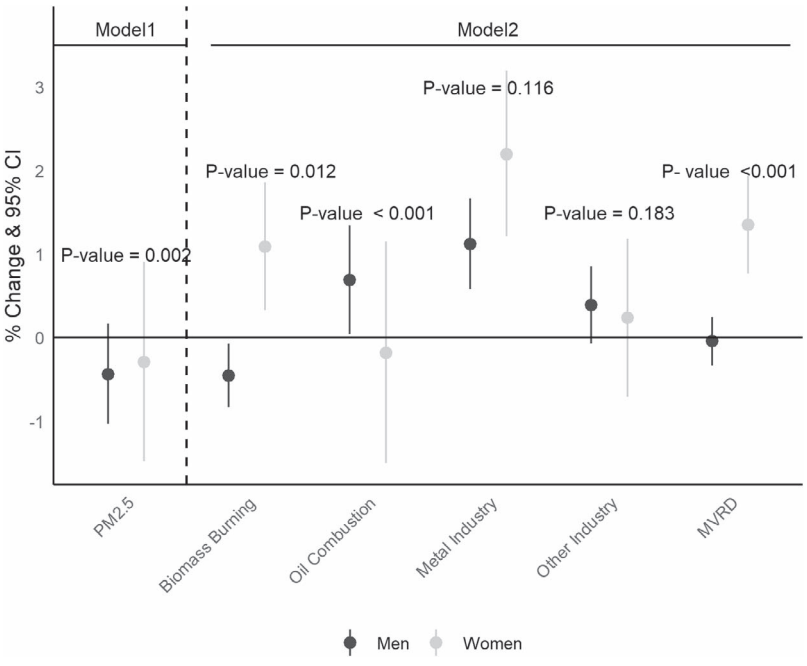

Figure 1. The association between PM2.5 mass and source-apportioned exposures and glucose, by sex. $PM_{2.5}$ = fine particulate matter; MVRD = motor vehicle and resuspension of dust. We used generalized additive linear models to assess the associations with repeated measures of log-transformed blood glucose levels. Model 1 assesses the associations with $PM_{2.5}$ mass, and model 2 simultaneously assesses the associations with the 5 source-apportioned $PM_{2.5}$ exposures. Models were adjusted for age, sex, race, census tract-level socioeconomic variables, mean temperature, year, occupation, and time spent on site during and after the 9/11 attack. We used a random intercept per responder and penalized spline for the year of testing to control for the time trend. Results are represented for IQR increases in exposure and the interaction term is specified above each set of sex stratified results.

Our findings are highly significant, especially for WTCHP responders, a unique population exposed to an extreme air pollution event linked to numerous adverse health outcomes.[46] A 2025 study found increased diabetes risk associated with higher WTC exposures among responders.[12] Therefore, it is especially important to investigate the health effects of environmental exposures on glycemic status later in life among this potentially vulnerable cohort. Moreover, our findings suggest that female responders may be especially susceptible to certain pollution sources, highlighting the need for targeted monitoring and early detection of CMDs in this subgroup.

Our findings should be interpreted in the context of several limitations. First, the total $PM_{2.5}$ mass and its composition in the tri-state area may not be generalizable to all rural and urban areas. However, our findings remain important to millions of people residing in similar large metropolitan areas. Second, although we use a comprehensive modeling approach to estimate $PM_{2.5}$ components, measurement error may still be possible. However, this error is minimized due to the high spatiotemporal resolution of our exposures. Additionally, our NMF model successfully reduced the data dimensionality, allowing us to incorporate the various $PM_{2.5}$ components tested in one model. Finally, the use of self-reported outcome definitions might be subject to recall bias when responders do not remember the exact year of diagnosis or the type of diagnosed diabetes. However, the similar exposure associations found with self-reported outcomes and clinical biomarkers supports the robustness of our findings. Information on subtypes of diabetes was not collected during monitoring visits. However, since the data recorded incidence among mid-aged participants, and incident cases before 2003 were excluded, type 1 diabetes is highly unlikely.

Conclusions

We found greater cardiometabolic risks (both glucose levels and diabetes risk) associated with $PM_{2.5}$ from metal industry sources among the overall WTCHP responders. Furthermore, elevated glucose levels were also linked to biomass burning and motor vehicle pollution among women, and to oil-combustion pollution among men. These findings can inform policies and regulations aimed at reducing emissions from these sources. This has important implications for the general public, who are exposed daily to pollution from traffic, heating, and building emissions. It is particularly relevant for populations that may be more vulnerable to environmental health effects, such as the WTCHP responder population, who experienced a massive air pollution event more than two decades ago; an event with lasting health consequences to this day.

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Study investigators, conference presentations, preprint publication information, thanks.

Supplementary material

Supplementary material is available at the *American Journal of Epidemiology* online.

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Conflict of interest

The authors have no conflicts of interest to disclose.

Data availability

The WTC cohort data was obtained from the World Trade Center Health Program (WTCHP) General Responder Cohort (GRC) data center. It includes protected health information with identifiers of individual patients. Per our data use agreement, data supplied for projects with IRB approval shall not be reused or redisclosed. Investigators wishing to request data can contact the WTCHP GRC data center directly.

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