

Prenatal Exposure to Wildfire PM_{2.5} and Risk of ASD and ADHD in Children Enrolled in Medicaid

Stefania Papatheodorou, MD, PhD; Hayon Michelle Choi, PhD; Krista F. Huybrechts, MS, PhD; Sonia Hernandez-Diaz, MD, DrPH; Yang Liu, PhD; Danlu Zhang, MPH; Matthew Shupler, PhD; Michael Leung, PhD; Wanyu Huang, PhD; Xinye Qiu, PhD; Yaguang Wei, PhD; Christopher J. McDougle, MD; Evgenia Papatheodorou, MD; Joel D. Schwartz, PhD; Brent A. Coull, PhD; Marc G. Weisskopf, PhD, ScD; Antonella Zanobetti, PhD

[+ Supplemental content](#)

IMPORTANCE Wildfires are an increasing source of fine particulate matter (PM_{2.5}), yet evidence on prenatal wildfire smoke exposure and long-term neurodevelopmental outcomes remains limited.

OBJECTIVE To examine whether prenatal exposure to wildfire-related PM_{2.5} is associated with risk of autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) in children.

DESIGN, SETTING, AND PARTICIPANTS This was a population-based cohort study of the US nationwide Medicaid population using Medicaid Analytic Extract (MAX) data from 2007 to 2014 with follow-up through childhood. Included were mother-child pairs with continuous enrollment and stable residential zip code during pregnancy.

EXPOSURE Weekly wildfire smoke-attributable PM_{2.5} at 1-km² resolution, aggregated to the zip code level across pregnancy.

MAIN OUTCOMES AND MEASURES Incident ASD and ADHD identified using validated claims-based algorithms. Associations were estimated using Cox proportional-hazards models with distributed lag nonlinear models.

RESULTS A total of 893 212 mother-child pairs (mother mean [SD] age, 24.7 [5.9] years) were included in this analysis. Children entered the cohort at birth (452 218 [50.6%] male). Mean (SD) observed follow-up was 6.3 (2.4) years for children diagnosed with ASD and 7.1 (1.9) years for children diagnosed with ADHD. Among 893 212 children, 8357 were diagnosed with ASD and 22 302 with ADHD. The cumulative incidence until age 8 years was 1.9% for ASD and 10.4% for ADHD. An IQR increase in cumulative wildfire PM_{2.5} exposure (0.75 µg/m³) during pregnancy was associated with increased risk of ASD (hazard ratio [HR], 1.30; 95% CI, 1.07-1.59) but not ADHD (HR, 1.03; 95% CI, 0.91-1.17). Associations for ASD were strongest during midpregnancy to late gestation compared with early gestation. Results were robust to sensitivity analyses.

CONCLUSIONS AND RELEVANCE Results of this cohort study show that prenatal exposure to wildfire-related PM_{2.5} may be associated with increased risk of ASD but not ADHD. These findings highlight the potential neurodevelopmental impacts of wildfire smoke and underscore the need for strategies to reduce exposure during pregnancy as wildfire activity intensifies.

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Author Affiliations: Author affiliations are listed at the end of this article.

Corresponding Author: Stefania Papatheodorou MD, PhD, Department of Biostatistics and Epidemiology, Rutgers School of Public Health, 683 Hoes Ln W, Piscataway, NJ 08854 (sp2599@sph.rutgers.edu).

Wildfires have emerged as an increasing threat to human health, especially among vulnerable populations,¹ and are increasing in size and severity due to rising temperatures, drying patterns, and human activities.^{2,3} Smoke from wildfires comprises a complex mixture of fine particulate matter (PM_{2.5}) and gaseous pollutants.⁴ Wildfire PM_{2.5} has been shown to be more toxic than other sources of PM_{2.5} due to its distinct chemical composition, smaller particle size, and frequent coexposure to environmental stressors such as elevated temperatures.⁵

Wildfire PM_{2.5} exposure has been increasingly linked to adverse neuropsychiatric outcomes across the life course,^{6–9} including respiratory, behavioral, and mental health effects in children and adolescents.^{10–14} Prenatal exposure has also been associated with low birth weight and preterm birth,^{15–17} which are associated with neurodevelopment. Two recent studies^{18,19} from California examined the association between wildfire PM_{2.5} and the risk of autism spectrum disorder (ASD), with 1 study¹⁸ showing increased risk in the third trimester with longer duration of wildfire PM_{2.5} exposure, whereas the other study¹⁹ did not identify a sensitive window of exposure and showed increased risks only at very high concentrations and at environments with lower levels of nonwildfire-related PM_{2.5}. Previous studies have linked ambient PM_{2.5} exposure with both ASD²⁰ and ADHD,^{21–23} potentially through shared mechanisms involving oxidative stress, inflammation, and neuroinflammation.²⁴

ASD and attention-deficit/hyperactivity disorder (ADHD) are significant public health concerns with a rising prevalence in the US.²⁵ As wildfires become more frequent and severe, and evidence accumulates on the health implications of wildfire PM_{2.5}, there is a pressing need to examine its potential role in early brain development and subsequent neurodevelopmental outcomes. This study seeks to address this important gap by examining whether exposure to wildfire PM_{2.5} during pregnancy is associated with an increased risk of ASD and ADHD in children using a large, population-based national US Medicaid cohort.

Methods

Study Design and Participants

The study protocol was approved by the institutional review boards at the Harvard T.H. Chan School of Public Health, Mass General Brigham, and Rutgers School of Public Health, which waived the requirement for informed consent. We conducted a population-based cohort study of live births in the 2007 to 2014 Medicaid Analytic Extract (MAX), which covers more than 40% of US births.^{26,27} MAX contains demographic, enrollment, medical claims, and outpatient prescription data. The cohort included women aged 12 to 55 years who were continuously enrolled from 3 months before the last menstrual period (LMP) through 1 month after delivery and their liveborn children. These enrollment requirements were applied to ensure adequate ascertainment of maternal characteristics and prenatal exposures and to establish follow-up of the children. Mothers and infants were linked using a deterministic

Key Points

Question Is prenatal exposure to wildfire-related fine particulate matter (PM_{2.5}) associated with risk of autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) in children?

Findings In this population-based cohort study of 893 212 mother-child pairs enrolled in US Medicaid, higher prenatal wildfire PM_{2.5} exposure was associated with an increase in ASD risk, whereas no meaningful association was observed for ADHD; associations for ASD were strongest with exposure to PM_{2.5} during midpregnancy to late pregnancy.

Meaning This study found that prenatal exposure to wildfire-related air pollution may be associated with an increased risk of ASD, highlighting the importance of reducing maternal exposure during pregnancy as wildfire activity intensifies.

algorithm based on the Medicaid case number, which identifies family units, and concordance between infant birth dates and maternal delivery windows.²⁶ The proportion match is approximately 60%, and the proportion of correct linkages is around 99% based on a validation study.²⁸ Gestational age was estimated from maternal and infant diagnostic and procedure codes using algorithms with high accuracy,²⁹ and the LMP was calculated from the delivery date and gestational age. We included pregnancies conceived on or after January 1, 2007. Follow-up data were available through December 31, 2018, providing at least 4 years of potential follow-up for children born in 2014. Children were followed up from birth until the first ASD or ADHD diagnosis, loss of Medicaid enrollment, death, or the end of the study period, whichever occurred first. To reduce exposure misclassification due to residential mobility, we restricted the cohort to women who had the same residential zip code at LMP and at delivery. The study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines.

Neurodevelopmental Disorders

ASD and ADHD were identified using validated claims-based algorithms incorporating diagnostic codes in both *International Classification of Diseases, Ninth (ICD-9)* and *Tenth (ICD-10) Revision*, and age criteria. These algorithms have high positive predictive values and yield prevalence estimates consistent with national data (eTable 1 in Supplement 1).^{30,31}

Wildfire PM_{2.5} Exposure Assessment

We assigned daily wildfire smoke-attributable PM_{2.5} concentrations at 1 km² spatial resolution across the contiguous US for the study period, aggregated to the weekly residential zip code level, using a previously validated machine-learning framework.³² Wildfire-specific PM_{2.5} was estimated by subtracting background concentrations from total PM_{2.5} concentrations that incorporated fire emissions, using a dual random forest modeling approach. The modeling framework demonstrated high spatiotemporal performance (annual R² approximately 0.90 via cross-validation), capturing regional and seasonal variability in wildfire-related PM_{2.5} across both urban and rural settings.

Covariates

Covariates were selected a priori based on a directed acyclic graph (eFigure 1 in Supplement 1). At the individual level, models adjusted for maternal age, maternal race and ethnicity, parity, prenatal folate dispensing, substance abuse, smoking, alcohol abuse, poor nutrition, and season of conception, categorized as spring (March-May), summer (June-August), fall (September-November), and winter (December-February). Maternal race and ethnicity were obtained from MAX enrollment records and included Asian or Other Pacific Islander, Black or African American, Hispanic or Latino, White, and unknown. Race and ethnicity were included as covariates because environmental exposures and access to neurodevelopmental evaluation and diagnosis may vary across racial and ethnic groups and not because they may be biological risk factors. Substance abuse, smoking, alcohol abuse, and poor nutrition were each modeled as binary indicators (yes or no), as used in previous studies,^{33,34} with a detailed definition shown in eTable 2 in Supplement 1. Although some of these characteristics may not be causally associated with wildfire PM_{2.5} exposure, they were retained in the models as covariates because of their established associations with ASD and ADHD risk.

We incorporated zip code-level socioeconomic indicators, including median household income, percentage of adults without a high school diploma, percentage of owner-occupied housing units, percentage Black, percentage Hispanic, and population density, derived from the 2000 to 2014 American Community Survey and the 2000 and 2010 US Census. County-level average body mass index was obtained from the Behavioral Risk Factor Surveillance System as a contextual indicator of population health and obesity prevalence that may capture regional differences associated with maternal metabolic health and neurodevelopmental risk. To approximate health care access, we calculated the annual distance from the centroid of each Zip Code Tabulation Area to the nearest hospital using data from the Dartmouth Atlas of Health Care.³⁵ Models additionally adjusted for urbanicity in 3 categories: urban areas (≥50 000 people), urban clusters (2500-49 999 people), and rural areas (<2500 people) based on US Census definitions.³⁶ All models were adjusted for background PM_{2.5} derived from the same model as wildfire PM_{2.5} and for average pregnancy temperature.

Although individual-level maternal comorbidities (eg, gestational diabetes, hypertensive disorders of pregnancy) and gestational age at birth were available, we did not adjust for these variables because they may lie on the causal pathway between wildfire PM_{2.5} exposure and ASD or ADHD.

Statistical Analysis

We fit stratified Cox models that allowed the baseline hazard to vary flexibly by birth year and county of residence, accounting for temporal and geographic heterogeneity in outcome incidence and exposure. The primary distributed lag models included exposures during gestational weeks 1 to 33. Because distributed lag nonlinear models require complete exposure information over the lag period, we selected week 33 as a maximum lag to capture many preterm births while covering most of gestation. For the exposure and lag response for wildfire

PM_{2.5}, we used a natural spline constraint, tested *df* from 2 to 6, and chose the combination that minimized the Akaike information criterion in the fully adjusted model. Therefore, we specified the exposure-response relationship using a natural spline with 3 *df* for wildfire PM_{2.5}, and the lag-response relationship using 2 *df*. We accounted for all covariates described in the previous section and computed robust standard errors, using zip code as the cluster index. Hazard ratios (HRs) were expressed per 1-IQR increase in cumulative wildfire PM_{2.5} across pregnancy.

Effect modification by child sex, race and ethnicity, and US Census region (eFigure 2 in Supplement 1) was evaluated using stratified analyses.^{37,38}

Sensitivity Analyses

We conducted sensitivity analyses using alternative lag windows (28 and 37 weeks), restricting outcomes to diagnoses after ages 3 and 5 years, and specifying linear exposure-response functions. We also evaluated minimally adjusted models keeping age, race, season of conception, median household income, urbanicity, and stratification by birth year and county, to address the noncollapsibility of the HRs and applied inverse probability of censoring weights to assess potential bias due to loss to follow-up.³⁹ To account for potential within-mother correlation among women contributing more than 1 pregnancy, we repeated the analyses using robust SEs clustered at the maternal level. We also examined whether the transition from ICD-9 to ICD-10 affected the results by performing stratified analysis for diagnoses before and after October 1, 2015.

To assess the robustness of the observed associations to potential unmeasured confounding, we calculated the evidence for causality (E-values).⁴⁰ E-values quantify the minimum strength of association that an unmeasured confounder would need to have with both the exposure and outcome, conditional on the measured covariates, to explain away the observed effect estimate.

We used SAS, version 9.3 (SAS Institute Inc) for data extraction, and R, version 4.2.0 (R Project for Statistical Computing) for Linux-gnu for analyses. Our interpretation of the results focused on the consistency of estimates across the main and sensitivity analyses, the strength of the adjusted HR, and its precision, as reflected in the width of the 95% CI, rather than on statistical significance at the *P* < .05 level.

Results

Study Population

The final analytic cohort included 893 212 mother-child pairs (mother mean [SD] age, 24.7 [5.9] years). Children entered the cohort at birth (994 [49.4%] female; 452 218 [50.6%] male). Mean (SD) observed follow-up was 6.3 (2.4) years for children diagnosed with ASD and 7.1 (1.9) years for children diagnosed with ADHD (eFigure 3 in Supplement 1). Maternal race and ethnicity were obtained from MAX enrollment records and included the following: 36 793 Asian or Other Pacific Islander (4.1%), 272 612 Black or African American (30.5%), 189 989

Table 1. Characteristics of the Study Population

Characteristic	Total	Wildfire smoke PM _{2.5}	
		Low (≤0.43 µg/m ³)	High (>0.43 µg/m ³)
Population			
Person, No.	893 212	446 606	446 606
Individual covariates			
Sex of children, No. (%)			
Female	440 994 (49.4)	220 311 (49.3)	220 683 (49.4)
Male	452 218 (50.6)	226 295 (50.6)	225 923 (50.6)
Race or ethnic group, No. (%)			
Asian or Other Pacific Islander	36 793 (4.1)	11 600 (2.6)	25 193 (5.6)
Black or African American	272 612 (30.5)	126 554 (28.3)	146 058 (32.7)
Hispanic or Latino	189 989 (21.3)	105 353 (23.6)	84 636 (19.0)
White	362 579 (40.6)	174 692 (39.1)	187 887 (42.1)
Unknown	31 239 (3.5)	14 814 (3.3)	16 425 (3.7)
Maternal age at delivery, mean (SD)	25.4 (6.0)	25.9 (6.1)	24.9 (5.8)
US Census region, No. (%)			
Northeast	191 946 (21.5)	131 658 (29.5)	60 288 (13.5)
Midwest	269 681 (30.2)	148 928 (33.3)	120 753 (27.0)
South	231 750 (25.9)	89 321 (20.0)	142 429 (31.9)
West	199 835 (22.4)	76 699 (17.2)	123 136 (27.6)
Ecological variables			
Median household income, mean (SD), \$ thousand	45 (16)	47 (17)	44 (15)
Did not complete high school, mean (SD), %	33 (15)	34 (16)	32 (15)
Living below the federal poverty line, mean (SD), %	14 (9)	14 (9)	13 (8)
Nearest hospital, mean (SD), km	5.3 (6.5)	4.4 (5.4)	6.1 (7.4)
Urbanicity, No. (%)			
Urban area	736 523 (82.5)	395 323 (88.5)	341 200 (76.4)
Urban cluster	131 226 (14.7)	42 881 (9.6)	88 345 (19.8)
Rural	25 458 (2.9)	8398 (1.9)	17 060 (3.8)
PM _{2.5} exposures, µg/m ³			
Wildfire PM _{2.5} , mean (SD)	0.55 (0.47)	0.27 (0.12)	0.83 (0.52)
Nonwildfire-related PM _{2.5} , mean (SD)	9.42 (2.88)	9.12 (3.36)	9.73 (2.27)

Abbreviation: PM_{2.5}, fine particulate matter

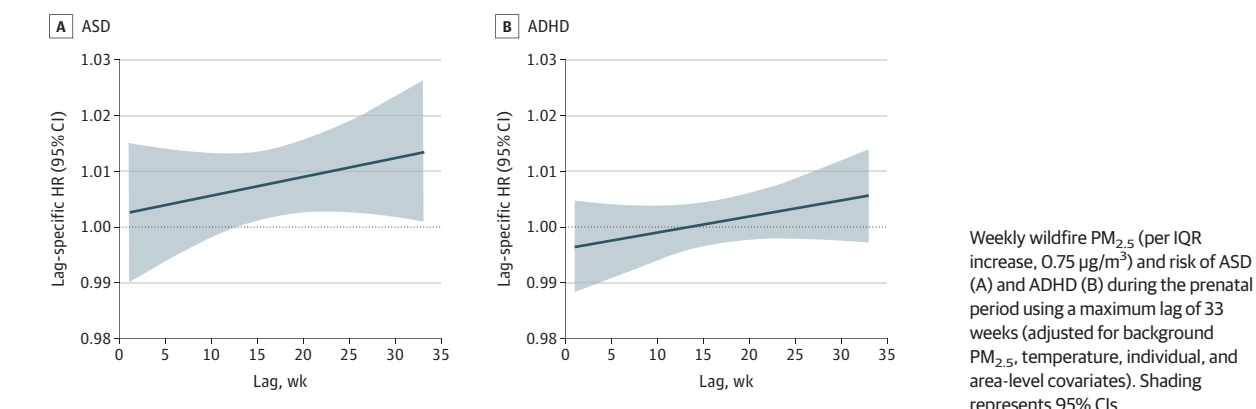
Hispanic or Latino (21.3%), 362 579 White (40.6%), and 31 239 unknown (3.5%). A total of 8357 children were diagnosed with ASD and 22 302 with ADHD. The cumulative incidence by age 8 years was 1.9% for ASD and 10.4% for ADHD, which is consistent with previous reports from the same cohort and with national statistics.³⁰ Participant characteristics were generally similar across wildfire PM_{2.5} exposure groups, although some sociodemographic and geographic differences were observed (Table 1). Compared with the lower-exposure group (PM_{2.5} ≤ 0.43 µg/m³), participants in the higher-exposure group (PM_{2.5} > 0.43 µg/m³) had slightly lower mean household income (mean [SD], \$44 000 [\$15 000] vs \$47 000 [\$17 000]), were more likely to live farther from a hospital (mean [SD], 6.1 [7.4] km vs 4.4 [5.4] km), and in rural (17 060 [3.8%] vs 8398 [1.9%]) or urban-cluster (88 345 [19.8%] vs 42 881 [9.6%]) areas but resided in areas with slightly lower proportions of residents who had not completed high school (mean [SD], 32% [15%] vs 34% [16%]) and who lived below the poverty level (mean [SD], 13% [8%] vs 14% [9%]). Overall, these differences did not indicate a consistent socioeconomic gradient across exposure groups. Wildfire and nonwildfire PM_{2.5} were moderately correlated (Pearson *r* = 0.37). The mean pregnancy

Table 2. Mean Annual Concentrations of Wildfire and Nonwildfire Fine Particulate Matter (PM_{2.5})

Year	Mean (SD), µg/m ³			Wildfire PM _{2.5} , %
	Total PM _{2.5}	Nonwildfire	Wildland fire	
2007	10.6 (3.7)	9.8 (3.5)	0.8 (0.8)	7.5
2008	10.4 (3.7)	9.7 (3.2)	0.7 (0.7)	6.7
2009	10.2 (3.3)	9.8 (3.0)	0.5 (0.4)	4.9
2010	9.9 (3.0)	9.4 (2.8)	0.5 (0.4)	5.1
2011	9.8 (2.9)	9.3 (2.9)	0.5 (0.4)	5.1
2012	9.6 (2.8)	9.1 (2.7)	0.5 (0.4)	5.2
2013	9.7 (2.9)	9.2 (2.8)	0.5 (0.4)	5.2
2014	9.1 (3.0)	8.7 (2.8)	0.4 (0.4)	4.4

concentrations of total, nonwildfire, and wildfire PM_{2.5} by year are shown in Table 2. Total and nonwildfire PM_{2.5} declined over the study period, whereas wildfire PM_{2.5} varied by year and declined from 0.8 µg/m³ in 2007 to 0.4 µg/m³ in 2014. Wildfire PM_{2.5} contributed 4.4% to 7.5% of total PM_{2.5}. The geographical distribution of the pregnancy-average wildfire PM_{2.5} by year is shown in eFigure 4 in Supplement 1.

Figure 1. Line Graphs Showing Lag-Response Relationship Between Weekly Wildfire Fine Particulate Matter (PM_{2.5}) and Risk of Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD)



Main Outcomes of Wildfire PM_{2.5}

Figure 1 presents the lag-specific associations between prenatal wildfire PM_{2.5} exposure and the risk of ASD and ADHD. An IQR increase in cumulative wildfire PM_{2.5} (0.75 µg/m³) was associated with a 30% higher risk of ASD (HR, 1.30; 95% CI, 1.07-1.59), whereas the increase for ADHD was not meaningful (HR, 1.03; 95% CI, 0.91-1.17). For both outcomes, the lag-response curves suggest a gradual increase in risk from early to late pregnancy, with the strongest point estimates observed during midgestation to late gestation, particularly after approximately 15 weeks. The corresponding exposure-response curves are shown in eFigure 5 in [Supplement 1](#). Background PM_{2.5} was associated with a modest increase in ASD risk (HR, 1.10; 95% CI, 1.02-1.19) but not ADHD (HR, 1.00; 95% CI, 0.90-1.10), with effect sizes smaller than those observed for wildfire PM_{2.5}, with no effect modification by US region (eTable 3 in [Supplement 1](#)).

Outcome Modification

Sex-stratified analyses did not provide evidence of outcome modification for ASD, with similar cumulative estimates observed in male (HR, 1.31; 95% CI, 1.04-1.64) and female (HR 1.31; 95% CI, 0.88-1.98) children. However, lag-response patterns differed, with male children showing elevated risks across most of pregnancy, whereas increases among female children were more apparent during midpregnancy to late pregnancy (**Figure 2**). Likewise, there was no evidence of outcome modification by sex for ADHD, with estimates close to the null in both male (HR, 1.00; 95% CI, 0.86-1.16) and female (HR, 1.11; 95% CI, 0.88-1.41) children.

Race-stratified analyses similarly did not indicate meaningful outcome modification (**Figure 3**). For ASD, point estimates were elevated across all racial and ethnic groups. For ADHD, estimates were generally near the null across groups, with the largest but imprecise estimate observed among Asian or Other Pacific Islander children. Regional analyses suggested heterogeneity, with larger point estimates observed in the South for both ASD (HR, 1.41; 95% CI, 0.82-2.42) and ADHD (HR, 1.32; 95% CI, 0.89-2.24), although CIs overlapped across regions.

Sensitivity Analyses

Sensitivity analyses using alternative lag windows, age restrictions, a parsimonious model, and a linear exposure-response specification produced results generally consistent with the primary analyses (eFigures 6-9 in [Supplement 1](#)). Analyses using inverse probability of censoring weights produced estimates similar to the primary analysis (ASD: HR, 1.29; 95% CI, 1.03-1.65; ADHD: 1.01; 95% CI, 0.87-1.17), suggesting that loss to follow-up did not meaningfully bias results. Mother-level clustering (ASD: cumulative HR, 1.29; 95% CI, 1.05-1.63; ADHD: cumulative HR, 1.03; 95% CI, 0.89-1.20) and ICD-9 to ICD-10 transition did not affect the results (eTable 4 in [Supplement 1](#)).

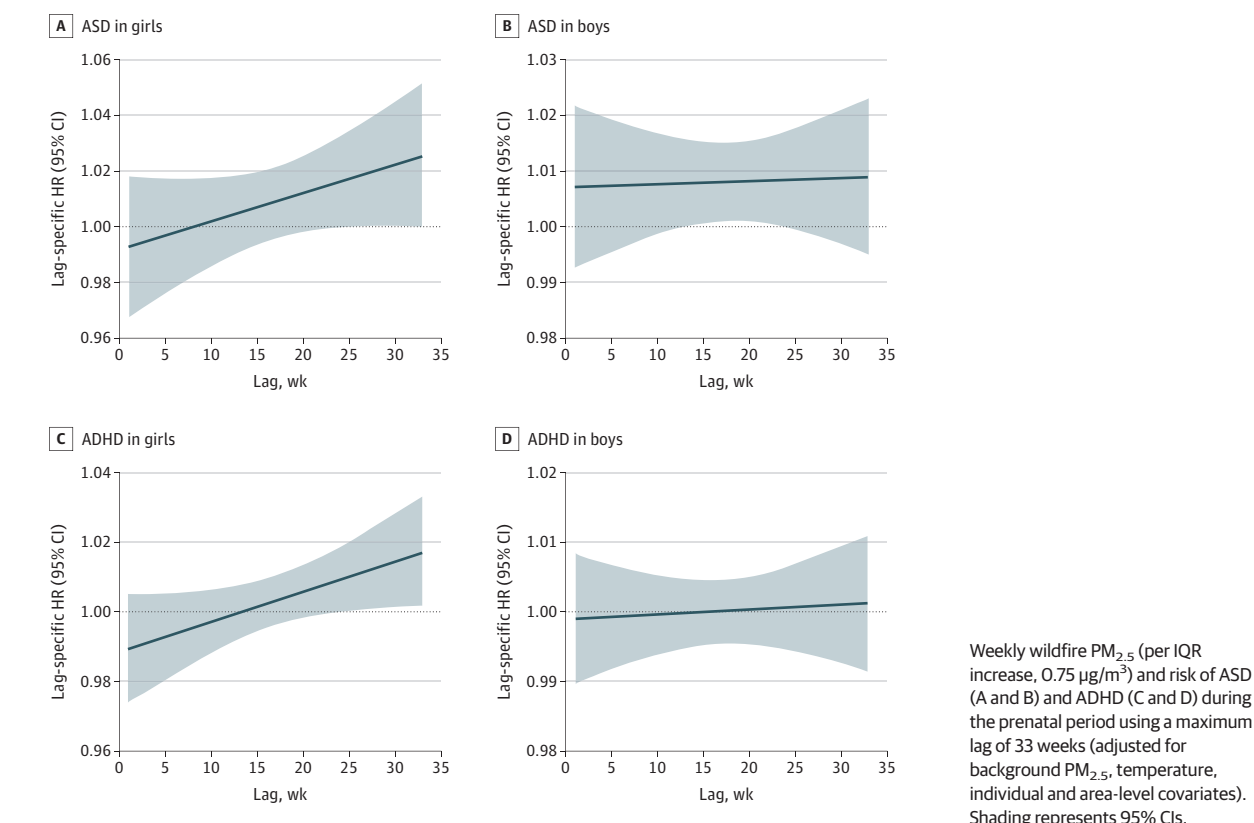
In the E-value analysis, an unmeasured confounder would need to be associated with both prenatal wildfire smoke PM_{2.5} exposure and ASD with risk ratios of at least 1.92 each, conditional on the measured covariates, to fully explain the observed association.

Discussion

In this large national cohort of Medicaid-enrolled mother-child pairs, prenatal exposure to wildfire-related PM_{2.5} was associated with increased risk of ASD, whereas associations with ADHD were not meaningfully elevated. Using distributed lag models, we identified stronger associations during midpregnancy to late pregnancy, suggesting the presence of sensitive developmental windows. These findings extend prior work by examining wildfire-specific PM_{2.5} in a geographically diverse population.

Prior studies evaluating wildfire PM_{2.5} and ASD have been limited and geographically restricted in California, with inconsistent findings. Luglio et al¹⁸ reported increased ASD risk associated with more than 10 wildfire smoke days during the third trimester among nonmovers (HR, 1.22; 95% CI, 1.04-1.44) in a cohort of 245 902 births between 2006 and 2014. In contrast, O'Sharkey et al¹⁹ using linked statewide birth and ASD registry data from 2001 to 2019 (8.6 million births), found largely null associations for pregnancy-average and trimester-

Figure 2. Line Graphs Showing Lag-Response Relationship Between Wildfire Fine Particulate Matter (PM_{2.5}) and Risk of Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) Stratified by Sex



specific wildfire PM_{2.5}, although elevated risks were observed at very high wildfire smoke levels in areas with low background PM_{2.5}. Differences between these studies and our findings may reflect variation in exposure assessment, exposure contrasts, statistical modeling approaches, and study populations.

The stronger association observed for ASD than for ADHD may reflect differences in the developmental timing and underlying neurobiology of the 2 disorders.^{41,42} ASD has strong prenatal neurodevelopmental origins, with disruptions in rapid brain growth, synaptogenesis, and maturation of neural circuits, occurring during late fetal development, whereas ADHD may reflect a broader developmental trajectory influenced by both prenatal and postnatal factors.⁴³⁻⁴⁹ Experimental evidence further supports persistent neurobehavioral and molecular effects of prenatal wildfire smoke exposure.^{50,51} Nevertheless, differences between the ASD and ADHD estimates should be interpreted cautiously, particularly given the limited epidemiologic evidence specifically examining prenatal wildfire PM_{2.5} and ADHD, and these findings require replication in other populations.

Heterogeneity between regions may reflect differences in wildfire characteristics and PM_{2.5} composition, climatic conditions and co-occurring heat, background air pollution, socioeconomic factors, health care access, and diagnostic practices.^{52,53}

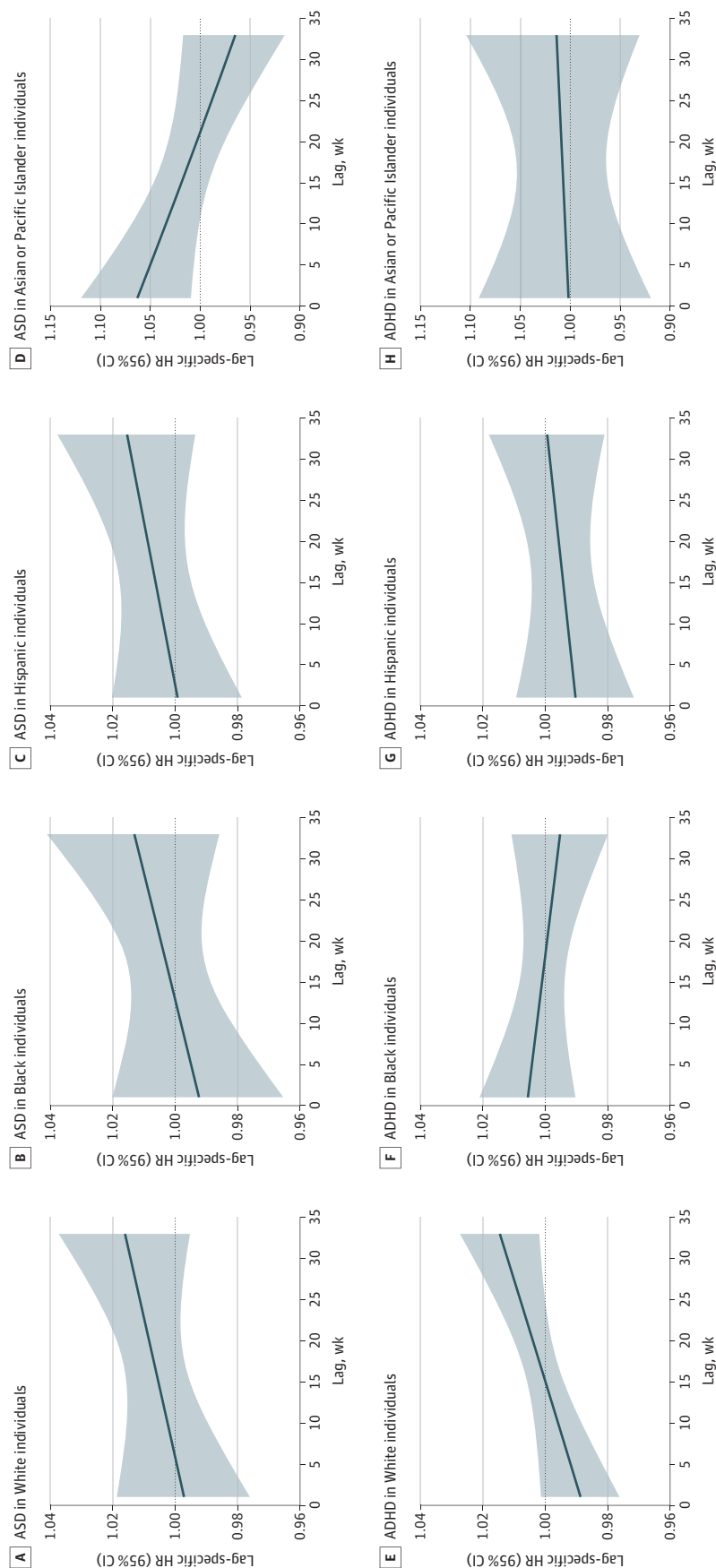
The nonlinear association between wildfire PM_{2.5} and ASD and ADHD is consistent with previous research on wildfire PM_{2.5} exposure and other mental health⁵⁴ or other health outcomes.^{55,56} Attenuation at higher exposure levels may reflect behavioral adaptations during severe smoke events, exposure misclassification, reduced precision at the upper end of the exposure distribution, or selection related to early pregnancy loss.⁵⁷

Strengths and Limitations

This study has several strengths. The large, national cohort of more than 890 000 mother-child pairs provided substantial statistical power and geographic diversity, including populations often underrepresented in environmental epidemiology. Exposure assessment relied on a validated high-resolution model, and the use of distributed lag models enabled evaluation of sensitive windows across gestation. Findings were robust across multiple sensitivity analyses, including alternative lag structures, outcome definitions, and adjustment strategies, as well as methods accounting for potential informative censoring.

Some limitations should also be considered. Wildfire PM_{2.5} was assigned at the zip code level without information on time-activity patterns or indoor mitigation behaviors, and weekly estimates may not capture short-term exposure peaks. However, area-level measures may be less susceptible to confounding by individual characteristics than highly personalized

Figure 3. Line Graphs Showing Lag-Response Relationship Between Wildfire Fine Particulate Matter (PM_{2.5}) and Risk of Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) Stratified by Race



covariates). Shading represents 95% CIs.

Weekly wildfire PM_{2.5} (per IQR increase, 0.75 µg/m³) and risk of ASD (A-D) and ADHD (E-H) during the prenatal period using a maximum lag of 33 weeks (adjusted for background PM_{2.5}, temperature, individual and area-level

exposure metrics.^{58,59} Although we restricted the cohort to individuals with stable zip codes during pregnancy, residential mobility within zip codes or between recorded time points could result in additional exposure misclassification. Outcomes were identified from administrative claims, although validated algorithms were used. Distance to the nearest hospital may not adequately capture access to specialists diagnosing ASD and ADHD, potentially contributing to geographic differences in outcome ascertainment. Residual confounding is also possible; however, an unmeasured confounder would need to be associated with both wildfire PM_{2.5} and ASD by risk ratios of at least 1.92, conditional on measured covariates, to explain away the observed association. Although the exposure distributions observed in our cohort may not fully reflect those experienced by more recent populations, our findings may become increasingly relevant to public health as the number of pregnancies affected by wildfire smoke grows. Studies using more recent birth cohorts are needed to determine whether these associations persist under contemporary, potentially higher wildfire-smoke exposure conditions. Although Medicaid-reimbursed dispensing may capture a substantial proportion of folate-containing product use in this low-income population, supplements obtained over the counter or paid for outside Medicaid were not recorded. Although linkage and enrollment requirements

reduce the eligible sample, previous evaluations of the MAX pregnancy cohort found minimal differences in key clinical and demographic characteristics between pregnant women included and those excluded due to eligibility requirements.^{60,61} Because the study population consisted of Medicaid beneficiaries, findings may not be fully generalizable to populations with different socioeconomic or health care characteristics, if these were effect modifiers of the biologic effects this study aims to assess.

Conclusions

As wildfire activity continues to intensify, understanding its potential impact on early-life health is increasingly important. Findings of this cohort study suggest that prenatal exposure to wildfire-related PM_{2.5} may be associated with an increased risk of ASD, particularly during midpregnancy- to late pregnancy. Efforts to reduce exposure during pregnancy, including improved air quality forecasting and strategies to limit indoor smoke infiltration, may be important for protecting maternal and child health. Further research is needed to clarify biological mechanisms and to evaluate the combined effects of wildfire smoke and other environmental and social stressors on neurodevelopment.

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Author Affiliations: Department of Biostatistics and Epidemiology, Rutgers School of Public Health, Piscataway, New Jersey (S. Papatheodorou); Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (S. Papatheodorou, Hernandez-Diaz, Shupler, Schwartz, Weisskopf); Korea Advanced Institute for Science and Technology, Daejeon, South Korea (Choi); Division of Pharmacoepidemiology and Pharmacoeconomics, Brigham and Women's Hospital, Boston, Massachusetts (Huybrechts); Gangarosa Department of Environmental Health, Rollins School of Public Health, Emory University, Atlanta, Georgia (Liu); Department of Biostatistics and Bioinformatics, Rollins School of Public Health, Emory University, Atlanta, Georgia (Zhang); The Nature Conservancy, Arlington, Virginia (Shupler); Department of Environmental Health, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (Leung, Huang, Wei, Schwartz, Weisskopf, Zanobetti); CardioVascular Institute, Beth Israel Deaconess Medical Center, Boston, Massachusetts (Qiu); Department of Medicine, Harvard Medical School, Boston, Massachusetts (Qiu); Department of Environmental Medicine and Climate Science, Icahn School of Medicine at Mount Sinai, New York, New York (Wei); Lurie Center for Autism, Massachusetts General Hospital, Lexington (McDougle); Department of Psychiatry, Harvard Medical School, Boston, Massachusetts (McDougle); University Hospital of Ioannina, Greece (E. Papatheodorou); Department of Biostatistics, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (Coull).

Author Contributions: Dr S. Papatheodorou had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: S. Papatheodorou, Choi, Leung, E. Papatheodorou, Schwartz, Coull.

Acquisition, analysis, or interpretation of data: S. Papatheodorou, Choi, Huybrechts, Hernandez Diaz, Liu, Zhang, Shupler, Leung, Huang, Qiu, Wei, McDougle, E. Papatheodorou, Schwartz, Weisskopf, Zanobetti.

Drafting of the manuscript: S. Papatheodorou, Choi, E. Papatheodorou.

Critical review of the manuscript for important intellectual content: All authors.

Statistical analysis: S. Papatheodorou, Choi, Leung, Schwartz, Coull.

Obtained funding: S. Papatheodorou.

Administrative, technical, or material support: Huybrechts, Liu, Zhang, Qiu, Wei, E. Papatheodorou.

Supervision: Hernandez Diaz, McDougle, E. Papatheodorou, Weisskopf, Zanobetti.

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