EHP Environmental Health Perspectives

pubs.acs.org/EnvHealthPerspectives

Article

Wildfire-Season Fine Particulate Matter Exposure and Associations with Influenza and Influenza-like-Illness Risk in the Western USA

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Cite This: *Environ. Health Perspect.* 2026, 134, 397–407

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ABSTRACT: **BACKGROUND:** Influenza remains a significant public health threat with pandemic potential. Understanding environmental factors influencing virus spread and severity is critical, particularly as wildfires become more frequent and intense. While temperature and humidity's roles in virus seasonality and persistence are well understood, the impacts of air pollution—especially wildfire-specific particulate matter ($PM_{2.5}$)—on respiratory infections are less explored. **OBJECTIVES:** This study aimed to investigate the association between wildfire $PM_{2.5}$ exposure and influenza or influenza-like illness (ILI) incidence. Specifically, we assessed (1) the long-term impact of $PM_{2.5}$ exposure during the preceding wildfire season on influenza/ILI risk in the following flu season, and (2) the effects of short-term $PM_{2.5}$ exposure during the active flu season. **METHODS:** We utilized ILI and influenza data from state health departments in six Western U.S. states (Arizona, Colorado, Montana, Nevada, Oregon, and Washington) from 2010 to 2019. We applied generalized linear distributed lag models to assess the impact of $PM_{2.5}$ exposure during the preceding wildfire season on influenza or ILI risk in the subsequent flu season, as well as the effect of short-term $PM_{2.5}$ exposure during the current flu season. **RESULTS:** Long-term exposure to wildfire $PM_{2.5}$ was associated with increased influenza risk in states with influenza data: Arizona ([Rate Ratio (RR) = 1.061 (1.026−1.100)]), Colorado [RR = 1.067 (1.056−1.078)], Montana [RR = 1.038 (1.013−1.063)], and Oregon [RR = 1.049 (1.041−1.057)], per 10 $\mu g/m^3$ $PM_{2.5}$ increase. However, the states with only ILI data did not follow this pattern, revealing no observed effect in Nevada [RR = 1.005 (0.920−1.097)] and a negative effect in Washington [RR = 0.884 (0.842−0.919)]. Similarly, but to a lesser degree, short-term $PM_{2.5}$ exposure effects were noted in states with only influenza data but not ILI data. **DISCUSSION:** Our findings underscore a positive association between wildfire-specific $PM_{2.5}$ and influenza risk in states with influenza data, suggesting a differential effect of $PM_{2.5}$ on respiratory infections. This study supports further investigation into the causative mechanisms behind these correlations, particularly considering the increasing frequency of wildfires and the resulting air quality impacts.

INTRODUCTION

Influenza remains a significant global public health threat, with the World Health Organization (WHO) stating that the question is not "if", but "when" a pandemic will occur.[1] While influenza (or flu) is a well-defined respiratory viral infection caused by the influenza virus, influenza-like illness (ILI) encompasses a broader category of respiratory infections that present with similar symptoms, such as fever, cough, and sore throat, but may be caused by a variety of other viruses or pathogens, including respiratory syncytial virus, adenovirus, and coronaviruses.[2] The WHO defines an illness as ILI if the patient has a fever greater than or equal to 37.8 °C and a cough, which began in the last 10 days.[3] In comparison, the Centers for Disease Control and Prevention (CDC) in the United States uses the same definition as WHO but with the additional condition of a sore throat.[2] However, because ILI can be caused by various pathogens, only laboratory testing can confirm whether a case is truly influenza. The distinction between influenza and ILI is crucial as the epidemiology, transmission dynamics, and health outcomes can differ significantly between influenza and other causes of ILI. For instance, while influenza is known for its seasonal epidemics and potential for causing pandemics, other ILI-causing pathogens may have different patterns of spread and severity.[4,5]

Globally, influenza impacts over a billion people each year, leading to a substantial number of illnesses and deaths.[6] Specifically, in the United States, the period from 2010 to 2020 witnessed millions of cases and tens of thousands of deaths attributed to influenza.[2,7] Influenza infection targets all demographic groups, with certain populations—children, the elderly, pregnant person, and individuals with chronic conditions—experiencing heightened vulnerability.[8] Beyond the well-known risk factors of most respiratory infections (indoor crowding, host immunity), climatic factors, such as temperature and humidity, also play an important role in influenza's emergence, spread, and severity.[9−12]

Among other environmental factors, air pollution—particularly fine particulate matter ($PM_{2.5}$) has garnered increasing

Accepted: May 16, 2025
Published: May 4, 2026

https://doi.org/10.1021/EHP.6c00326
Environ. Health Perspect. 2026, 134, 397–407

attention due to its profound impact on respiratory health and affecting overall well-being.[13,14] These particles, measuring 2.5 μm or smaller, can penetrate deep into the lungs, exacerbating existing respiratory and chronic health conditions.[15] $PM_{2.5}$ exposure has been linked not only to worsening chronic illnesses but also to premature deaths globally, emphasizing its far-reaching effects beyond just respiratory issues.[16] The relationship between $PM_{2.5}$ and viral infections is well documented, with multiple studies indicating an elevated risk of respiratory diseases like COVID-19 and influenza associated with short-term (less than 1 month) $PM_{2.5}$ exposure.[17−22] A recent systematic review and meta-analysis reported that increases in $PM_{2.5}$ levels were associated with up to 50% higher risk of influenza, highlighting the importance addressing air quality as a potential factor influencing respiratory infection outcomes.[23]

While ambient $PM_{2.5}$ levels have generally been declining in many parts of the world, sources of $PM_{2.5}$ from wildfire smoke are on the rise.[24,25] This shift has caused a surge in research on the health impacts of wildfire-derived $PM_{2.5}$. Importantly, $PM_{2.5}$ from wildfires differs in composition and toxicity from $PM_{2.5}$ emitted by other anthropogenic sources. Wildfire smoke contains higher levels of organic carbon, hazardous air pollutants, and reactive oxygen species, which may drive more severe biological responses. In fact, studies have found that wildfire-derived $PM_{2.5}$ can be more toxic per unit mass than $PM_{2.5}$ from other combustion sources.[26,27] Moreover, wildfire smoke is becoming the dominant source of $PM_{2.5}$ in many western U.S. states.[28] Mechanistically, exposure to wildfire smoke has been linked to delayed immune suppression and increased susceptibility to respiratory infections.[29−33] However, only a few localized studies focused on temperate regions of the world[34,35] have observed positive associations between wildfire smoke exposure and influenza during the following flu season using time series environmental health modeling. Here, we test this association and explore the relationship between wildfire-specific $PM_{2.5}$ exposure and influenza risk across a broader geographic and environmental scope, focusing on six wildfire-impacted western U.S. states: Arizona (AZ), Colorado (CO), Montana (MT), Nevada (NV), Oregon (OR), and Washington (WA) (Figure 1, Table S1).

MATERIALS AND METHODS

All analyses were performed with R software (version 4.2; R Development Core Team) including the 'MASS',[36] 'tsModel',[37] 'tidyverse',[38] 'Epi',[39] 'zoo',[40] 'splines'[41] packages.

Study Area and Influenza and Influenza-like-Illness Data Collection

We contacted state health departments across all 11 Western USA states, specifically targeting their infectious disease or influenza surveillance divisions. Ten states responded, and weekly influenza or influenza-like illness (ILI) data were acquired from eight states from 2010 to 2020 at either the county or regional level. Our final analysis included six states with county level data: AZ, CO, MT, NV, OR, and WA (see Figure 1). Laboratory-confirmed influenza case data were obtained from AZ, CO, MT, and OR, while ILI data were collected from NV and WA[42] (Table 1). In both NV and WA, ILI data were reported based on the CDC case definition for ILI, which includes "fever (≥100 °F [37.8 °C]) and cough and/or sore throat without a known cause other than influenza".[43] For a visual representation of the influenza model variability across these six states, refer to Table S2.

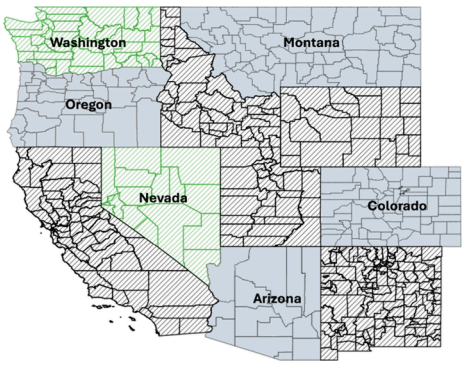

Figure 1. Six U.S. western states included in this study: Washington (WA), Oregon (OR), Nevada (NV), Arizona (AZ), Colorado (CO), and Montana (MT). Weekly laboratory-confirmed influenza data (solid gray) and influenza-like illness (ILI) data (green hashed) were collected at the county level from January 2010 to December 2019. States with gray hashing indicate regions that were contacted but ultimately not included in the study. We note that California and New Mexico provided data at a coarser level than county data and thus were excluded from our analysis.

Table 1. Summary of Influenza and Influenza-like-Illness (ILI) Cases Reported for Six Western U.S. States [Arizona (AZ), Colorado (CO), Montana (MT), Nevada (NV), Oregon (OR), Washington (WA)] from January 1, 2010, to December 31, 2019

Year	AZ	CO	MT	NV (ILI)	OR	WA (ILI)
2010	1152	383	874	9476	266	355
2011	8682	1007	4088	12252	677	1535
2012	5135	1016	3514	6116	893	2936
2013	10756	1794	11169	8863	1693	2423
2014	13387	2985	5651	8182	1558	1886
2015	11526	1336	4971	5979	2027	1701
2016	23989	2106	4828	6775	2226	1713
2017	23865	4550	8216	9803	5084	1072
2018	28002	4019	9778	8787	3471	126
2019	19167	3870	13320	8195	3980	9847
Total	144657	22930	76648	84428	22484	36701

Main Exposure Variables of Interest

$PM_{2.5}$ Exposure Assessment. We used daily $PM_{2.5}$ surface concentration estimates from a 1 km resolution data set (Swanson et al. 2022[49]) and extracted weekly county-level $PM_{2.5}$ using the "exactextract" package in R;.[44] Exposure data spanned from January 1, 2010, to December 31, 2019 (Table 2). Daily $PM_{2.5}$ data were averaged to the weekly level to match the weekly ILI and influenza case data reported by state health departments, ensuring temporal alignment between the exposure and outcome. We included both short- and long-term $PM_{2.5}$ exposure metrics to capture different biological and epidemiological pathways by which air pollution may influence influenza and ILI risk.

Short-Term $PM_{2.5}$ Exposure. This was defined as weekly averages during the same week as (lag 0) and up to 4 weeks prior to the influenza or ILI case week (lags 1−4). We also assessed cumulative exposure using summed $PM_{2.5}$ across multiple lag windows (e.g., weeks 0−1, 0−4). Short-term exposure was modeled to assess the acute effects of $PM_{2.5}$ on respiratory infection susceptibility, consistent with previous studies showing that recent exposures can impair immune response and exacerbate respiratory conditions.[22,45−47]

Long-Term Cumulative Wildfire Season $PM_{2.5}$ Exposure. This was defined as the cumulative concentration of $PM_{2.5}$ during each state's preceding wildfire season. This metric was included to

https://doi.org/10.1021/EHP.6c00326
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Table 2. Summary of Annual (All Weeks) and Cumulative Wildfire Season (May−Oct) $PM_{2.5}$ Levels ($\mu g/m^3$) Calculated from Weekly Averages for Six Western US States [Arizona (AZ), Colorado (CO), Montana (MT), Nevada (NV), Oregon (OR), Washington (WA)][a]

State	Mean	Med	SD	IQR	WF	State	Mean	Med	SD	IQR	WF
AZ						**NV**					
2010	4.13	3.84	1.65	2.22	127.83	2010	3.86	3.73	1.24	1.65	96.01
2011	5.03	4.56	2.37	3.10	161.84	2011	4.18	4.04	1.55	2.08	108.84
2012	5.35	5.20	2.66	4.07	174.68	2012	4.88	4.45	2.53	3.71	152.04
2013	4.30	3.91	2.13	3.29	132.99	2013	5.24	4.17	5.36	2.69	161.97
2014	3.86	3.55	1.72	2.29	111.95	2014	4.09	3.79	2.12	2.27	119.68
2015	3.39	3.09	1.50	1.85	99.27	2015	3.91	3.48	2.48	2.20	117.27
2016	3.74	3.64	1.39	1.86	120.82	2016	3.29	2.92	1.72	1.76	98.06
2017	5.09	4.69	2.11	2.78	165.75	2017	4.24	3.35	2.54	2.95	135.62
2018	4.59	4.16	2.16	2.87	143.67	2018	5.25	3.57	6.01	2.73	187.77
2019	3.52	3.40	1.23	1.80	109.15	2019	3.20	2.91	1.22	1.94	90.56
Total	4.30	3.88	1.89	2.61	134.79	Total	4.22	3.65	2.68	2.40	125.88
CO						**OR**					
2010	3.36	3.11	1.52	1.86	83.67	2010	3.37	3.15	1.39	1.67	58.80
2011	3.83	3.52	1.91	2.10	93.71	2011	3.68	3.23	1.92	2.07	66.92
2012	5.02	4.07	3.51	3.97	147.53	2012	4.38	3.18	3.73	3.10	108.19
2013	3.79	3.50	1.79	2.58	98.81	2013	4.72	3.94	2.90	2.87	98.72
2014	3.34	3.10	1.71	2.09	88.18	2014	4.26	3.72	2.25	2.73	90.54
2015	3.36	2.85	2.55	2.23	101.64	2015	4.74	3.74	4.43	2.29	113.43
2016	3.07	2.92	1.54	2.09	81.13	2016	3.30	3.25	1.40	1.87	59.05
2017	4.15	3.31	3.20	2.75	129.16	2017	5.66	3.13	8.98	2.62	183.12
2018	4.03	3.16	2.95	2.72	124.25	2018	5.22	3.34	6.64	2.82	149.16
2019	2.86	2.71	1.35	1.79	73.42	2019	3.56	3.39	1.36	1.65	54.66
Total	3.68	3.13	2.20	2.42	103.42	Total	4.29	3.30	3.50	2.37	98.69
MT						**WA**					
2010	3.21	3.08	1.38	1.81	55.53	2010	3.88	3.54	1.79	1.97	63.99
2011	3.50	3.14	1.68	2.18	72.97	2011	4.01	3.67	1.94	2.23	63.53
2012	5.36	3.51	5.44	4.11	150.44	2012	4.69	3.64	3.81	2.97	104.64
2013	3.89	3.28	2.08	2.28	88.11	2013	5.04	4.49	2.54	2.88	83.37
2014	3.74	3.22	2.35	2.02	86.08	2014	4.66	4.18	2.28	2.54	85.11
2015	4.47	2.90	6.62	1.86	133.64	2015	5.14	3.90	5.73	1.99	112.46
2016	2.99	2.64	1.72	1.66	60.65	2016	3.50	3.39	1.44	1.77	54.47
2017	5.25	2.99	6.51	2.37	171.02	2017	5.78	3.25	10.10	2.92	177.06
2018	4.38	2.81	5.13	2.03	131.00	2018	5.23	3.26	7.40	2.79	146.42
2019	2.74	2.50	1.31	1.55	50.17	2019	3.81	3.47	1.69	1.82	53.06
Total	3.95	3.04	3.42	2.19	100.90	Total	4.57	3.59	3.87	2.39	94.76

[a]Annual $PM_{2.5}$ data[49] show the mean, median (Med), standard deviation (SD), and interquartile range (IQR) for weekly average concentrations across all 52 weeks of each year. Wildfire season (WF) $PM_{2.5}$ represents the cumulative total $PM_{2.5}$ calculated by summing weekly average concentrations over the state-specific wildfire season. Total represents averages for all years 2010−2019.

assess the potential impact of sustained or repeated smoke exposure over several months. Chronic exposure may result in prolonged immune modulation, increased respiratory inflammation, or cumulative damage that could elevate susceptibility to respiratory infections, including influenza, during the subsequent flu season.[32,48] To estimate long-term wildfire-specific $PM_{2.5}$ exposure, we used a two-step approach that builds upon the methodology outlined by Landguth et al.[35] Landguth et al. relied on U.S. EPA Air Quality System (AQS) data tagged with wildfire smoke qualifier codes and annotations made by air quality specialists when ambient $PM_{2.5}$ concentrations were judged to be influenced by wildfire smoke based on supporting evidence (e.g., satellite imagery, fire incident reports) (Table S1). These tags allowed for the estimation of state-level wildfire-attributed $PM_{2.5}$ values during wildfire seasons.

In our study, we extended this approach by integrating high-resolution satellite-based data from the National Oceanic and Atmospheric Administration (NOAA) Hazard Mapping System (HMS) Fire and Smoke Product. HMS provides daily shapefiles of smoke plume polygons categorized by intensity: light, medium, or heavy, based on visual interpretation of satellite imagery.[30] We used these data to spatially identify smoke-affected areas across the six study states. We then overlaid these HMS smoke plumes with our 1 km gridded daily $PM_{2.5}$ surface concentration estimates (Swanson et al. 2022[49]) to assign wildfire smoke exposure at the county level.[49] Specifically, for each day and county, we determined whether any portion of the county overlapped with a HMS smoke plume. If an overlap occurred, we classified that day as a "smoke day" and included the corresponding county-level $PM_{2.5}$ value in the wildfire-specific cumulative exposure estimate. To reduce potential misclassification due to the inherent uncertainty in plume detection, especially for the "light" category, we conducted sensitivity checks excluding light plumes. However, because light plumes comprise a substantial proportion of smoke-affected days and are relevant for chronic low-level exposure, we opted to include all HMS plume categories (light, medium, and heavy) in the main analysis. This approach was consistent across states and years. County-level wildfire-specific $PM_{2.5}$ was calculated by summing daily $PM_{2.5}$ values on days with plume overlap during each state's defined wildfire season (based on average seasonal timing and smoke activity; see Table 2). Although wildfire activity is generally concentrated in the summer and fall, we initially

https://doi.org/10.1021/EHP.6c00326
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examined $PM_{2.5}$ data across the full calendar year to detect any unexpected trends. No meaningful seasonal $PM_{2.5}$ effects were observed outside of these periods, supporting our decision to limit long-term exposure calculations to wildfire seasons only.

Final models incorporated both short- and long-term $PM_{2.5}$ exposure metrics, but these were first evaluated in separate models to assess their individual contributions to the model fit. All exposure variables were added iteratively to ensure each improved model performance and interpretability before inclusion in the final structure.

Other Explanatory Variables of Interest

To determine the best model for assessing the impact of $PM_{2.5}$ exposure on influenza risk in each state, we evaluated each model based on residual deviance and Quasi-AIC (QAIC), with the lowest values indicating the best fit. Autocorrelation in influenza cases was modeled using a quasi-Poisson count model with 1, 2, and 3 week lags. A 1 week autocorrelation structure consistently performed bested and was selected for the final models.

Recognizing the strong association between climate factors and influenza risk, we examined the influence of temperature, precipitation, and humidity as well. Historical daily temperature, precipitation, and humidity data were extracted at the weekly and county levels for each of the six states using the Oregon PRISM data set[50] (Figures S1 and S2). Models were fitted to assess the effects of temperature, precipitation, and humidity separately, as well as their combined effects (precipitation × temperature × humidity). Additionally, we explored their interactions with $PM_{2.5}$, capturing how these climate factors might modify the relationship between $PM_{2.5}$ and health outcomes. Humidity did not improve the model fit or show consistent statistical significance in any of the six states. As such, it was excluded from final models and included only precipitation and temperature. We compared model performance using residual deviance and QAIC values to evaluate the relative importance of these factors and their interactions. To account for the cyclical seasonal patterns in influenza, we explored two methods for adjusting for seasonality: Fourier terms and natural splines. Fourier terms, including sine and cosine functions at frequencies of 52, 26, and 13 weeks, captured annual, biannual, and quarterly patterns of influenza incidence. Alternatively, natural splines allowed for nonlinear relationships between time (in weeks) and influenza rates. Incorporating these seasonality variables improved the model's ability to isolate the effects of other covariates, such as temperature and lagged influenza rates, thereby enhancing the accuracy of influenza risk estimates. The two seasonality adjustment methods were compared using residual deviance and QAIC values to determine the best representation of seasonality in the influenza risk models. For details on the final model selection, refer to Table S2.

Statistical Modeling

We utilized quasi-Poisson distributed lag models (DLMs) with an offset for the county population to investigate the relationship between influenza rate and various predictors. The quasi-Poisson approach was chosen to account for the overdispersion observed in the count data of influenza cases. Models were fit for each state individually, including county fixed effects to control for regional differences. Importantly, all of the models were developed iteratively. We began with a base model including only seasonal terms and autocorrelation structure, then added each covariate, climate variables, short-term $PM_{2.5}$ lags, and wildfire-season $PM_{2.5}$ exposure one at a time. This stepwise approach allowed us to assess how each variable improved the model fit using residual deviance and QAIC metrics. Separate models were initially constructed to examine the effects of short-term and long-term $PM_{2.5}$ independently. These were then compared with combined models incorporating both exposure types. To assess the independent effects of wildfire-season and short-term $PM_{2.5}$ exposure on influenza or ILI risk, we constructed separate models for each exposure type and then compared them to combined models. In the wildfire-season $PM_{2.5}$ models, we controlled for short-term $PM_{2.5}$ lags to ensure that the observed effects reflected long-term seasonal exposure rather than coincident short-term spikes. Likewise, in the short term $PM_{2.5}$ models, we adjusted for cumulative wildfire-season $PM_{2.5}$ exposure to account for the prior seasonal burden. This mutual adjustment approach allowed us to estimate the unique contribution of each exposure type while controlling for the potential confounding effects of the other. This was particularly important given the possibility that short-term peaks during the influenza season may coincide with or follow heavy wildfire exposure periods and failing to adjust for this could lead to misattribution of risk. Final model selection was based on improvement in fit, interpretability, and consistency with known influenza dynamics. This approach enabled us to examine not only the independent effects of each exposure type but also their joint contribution when modeled together. These modeling decisions directly inform the interpretation of our results and are reflected in the discussion of differential patterns across states.

Missing data for influenza/ILI outcomes and $PM_{2.5}$ exposure were rare. We had complete data for influenza/ILI outcomes during the flu season (May−October) for all states, with the exception of Montana, which occasionally reported case counts as "NR" (not reported). These "NR" values were infrequent, occurring in fewer than five county-week observations per week per year. County-week observations with missing case counts or missing exposure or covariate values were excluded from the regression models. Missingness was assumed to be random and not associated with exposure or outcome levels. No imputation was applied. Given the multiyear structure of the data set and the large number of weekly observations across counties, the small proportion of missing values had minimal impact on model estimates or stability.

The model form used for each state separately was

$$\log(\mu_{t,k}) = \log(\text{Population}_k) + \beta_0 + \sum_{s=0}^{6} F_i(t,k) + \beta_5 \text{Climate}_{t,k} + \beta_6 \log_{(\mu_{t-1,k})} + \sum_{s=0}^{L} \beta_s \text{PM\_lag}_{s,t,k} + \beta_7 \text{WildfireSeason}_k + \beta_8 \sum_{i=10}^{Y} \beta_i \text{County}_k$$

where t is the week index from, t = 1, 2, ..., 435 (corresponding to the period from January 3, 2010, to December 31, 2019 (i.e., rough start of pandemic timeline for Western U.S.), excluding weeks outside of the influenza season as defined by the Center for Disease Control and Prevention), k is the county index. The outcome variable $\mu_{t,k}$ is the expected influenza count in county k for week t. The natural log of county population, $\log(\text{Population}_k)$, is the population in county k for week t, included in the model as an offset to allow for an influenza rate response. B_i's are the model parameters. $F_i(t, k)$ represent the seasonal terms in the model where i = 1, 2, 3 corresponds to the sine functions and i = 4, 5, 6 for cosine functions, all applied at frequencies of 13, 26, and 52 weeks, respectively, for county k in week t. These seasonal terms capture seasonal patterns in influenza incidence. $\text{Climate}_{t,k}$ represents a climate variable, which includes temperature (°C), precipitation (mm), or their interaction, for county k in week t. Relative humidity (percent) was evaluated but excluded from the final models due to poor model fit. $\log(\mu_{t-1,k})$ is the autocorrelation term, representing the log of influenza cases from the previous week in county k. One-week autocorrelation was always used in the model, while two- and three-week autocorrelations were tested, but based on model diagnostics, the one-week fit best. Short-term $PM_{2.5}$ exposure was modeled as a continuous variable ($\mu g/m^3$) using weekly average concentrations that lagged from 0 to 4 weeks prior to the case week. In some models, cumulative exposure windows (e.g., lag 0−1, lag 0−4) were also tested. $\text{PM\_lag}_{s,t,k}$ represents lagged $PM_{2.5}$ exposure for county k, with s ranging from 0 to L weeks depending on the lag structure. WildfireSeason_k was calculated as the cumulative sum of daily $PM_{2.5}$ values across the wildfire season in the previous calendar year, for days when NOAA's Hazard Mapping System (HMS) smoke plumes overlapped with each county. This variable was also continuous and measured in $\mu g/m^3$. To isolate the independent effects of long- and short-term $PM_{2.5}$ exposure, all models that

https://doi.org/10.1021/EHP.6c00326
Environ. Health Perspect. 2026, 134, 397−407

Table 3. Rate Ratios with 95% Confidence Intervals (CI) and 2-Tailed *p*-Values for $PM_{2.5}$ Exposure during Wildfire Season, Stratified by State and Lag Period[a]

State	Rate Ratios, 95% CI	*p*-value	State	Rate Ratios, 95% CI	*p*-value
AZ			**NV (ILI)**		
WF	**1.061 (1.026, 1.100)**	**<0.001**	WF	1.005 (0.920, 1.097)	0.069
0 week lag	0.948 (0.906, 0.975)	0.004	0 week lag	0.924 (0.923, 1.068)	0.077
1 week lag	1.026 (0.958, 1.048)	0.343	1 week lag	0.930 (0.944, 1.069)	0.155
2 week lag	1.008 (0.974, 1.047)	0.210	2 week lag	0.999 (0.985, 1.067)	0.952
3 week lag	0.956 (0.955, 1.037)	0.027	3 week lag	0.991 (0.979, 1.053)	0.629
4 week lag	**1.067 (1.053, 1.128)**	**<0.001**	4 week lag	0.962 (0.957, 1.056)	0.117
CO			**OR**		
WF	**1.067 (1.056, 1.078)**	**<0.001**	WF	**1.049 (1.041, 1.057)**	**<0.001**
0 week lag	0.999 (0.976, 1.006)	0.976	0 week lag	0.988 (0.966, 1.011)	0.287
1 week lag	**1.047 (1.020, 1.057)**	**<0.001**	1 week lag	0.975 (0.952, 1.000)	0.034
2 week lag	0.995 (0.977, 1.008)	0.533	2 week lag	1.038 (0.998, 1.051)	0.008
3 week lag	1.006 (0.959, 1.014)	0.705	3 week lag	**1.037 (1.016, 1.045)**	**<0.001**
4 week lag	0.984 (0.949, 0.997)	0.182	4 week lag	**1.028 (1.007, 1.027)**	**<0.001**
MT			**WA (ILI)**		
WF	**1.038 (1.013, 1.063)**	**<0.001**	WF	0.884 (0.842, 0.919)	<0.001
0 week lag	1.152 (0.875, 1.272)	0.160	0 week lag	1.051 (0.985, 1.077)	0.024
1 week lag	0.877 (0.848, 1.099)	0.067	1 week lag	1.010 (0.884, 1.073)	0.844
2 week lag	0.989 (0.882, 1.049)	0.047	2 week lag	0.993 (0.943, 1.037)	0.778
3 week lag	0.973 (0.926, 1.106)	0.559	3 week lag	0.989 (0.935, 1.007)	0.553
4 week lag	1.026 (0.947, 1.083)	0.437	4 week lag	0.999 (0.993, 1.007)	0.993

[a]0-week lag corresponds to the same week as the influenza or ILI cases; 1-week, 2-week, 3-week, and 4-week lags correspond to the number of weeks preceding the onset of cases. Bolded values indicate statistically significant positive associations and where the *p*-values are below the 0.05 level. WF = Wildfire season $PM_{2.5}$, AZ = Arizona, CO = Colorado, MT = Montana, NV = Nevada, OR = Oregon, WA = Washington.

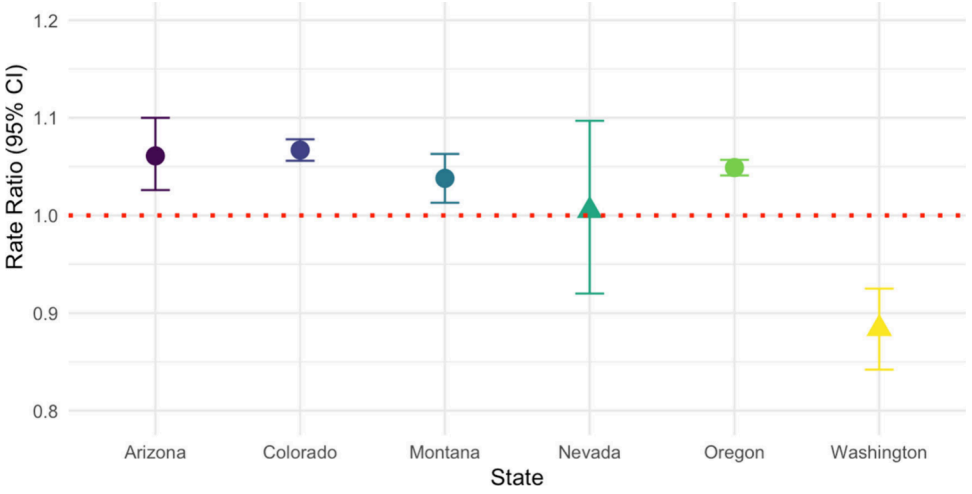

Figure 2. Rate ratios and 95% confidence intervals for each of the six states included in the analysis, representing the association between a 10 $\mu g/m^3$ increase in cumulative wildfire season $PM_{2.5}$ and the subsequent influenza or ILI risk. Circles indicate states with laboratory-confirmed influenza case data (Arizona, Colorado, Montana, and Oregon). Triangles indicate states with influenza-like illness (ILI) data (Nevada and Washington). The red dashed line at RR = 1.0 denotes the null value (no association). Each rate ratio reflects the estimated relative risk of influenza or ILI associated with a 10 $\mu g/m^3$ increase in cumulative wildfire-specific $PM_{2.5}$ during the defined wildfire season (e.g., May−October) in the year prior to the influenza season. These values are derived from the state-specific quasi-Poisson regression models adjusted for seasonality, weather, and other covariates. Numeric results shown in this figure are available in Table 3.

examined wildfire-season $PM_{2.5}$ included adjustment for short-term $PM_{2.5}$ lags and vice versa. This mutual control allowed us to distinguish the unique contribution of each exposure type while accounting for their potential temporal correlation. $\log(\text{County}_k)$ is a county-specific indicator variable for county k, to account for county-level fixed effects. All model covariates were added iteratively, beginning with a base model, including seasonality and autocorrelation, followed by stepwise inclusion of climate variables, short-term $PM_{2.5}$, and wildfire-season $PM_{2.5}$. Final model selection was based on improvement in residual deviance and QAIC. See Table S2 for specific state combinations. The terms in the model are all variables regularly associated with influenza dynamics as seen in Imai et al.[51]

Validation

Each model underwent a thorough validation process to ensure its robustness and accuracy. We began by examining the residuals vs fitted values plot to check for any discernible patterns that might indicate a poor fit or heteroskedasticity. We also analyzed the autocorrelation function (ACF) of the residuals to detect any remaining autocorrelation that might not have been fully accounted for in the model. These ACF plots revealed that the autocorrelation

https://doi.org/10.1021/EHP.6c00326
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term in our model was not sufficiently strong to account for all of the autocorrelation present in the data. To address this issue and improve the reliability of our estimates, we implemented a stratified bootstrap technique. The bootstrap was stratified by state and influenza season to preserve temporal structure and regional variation in both $PM_{2.5}$ exposure and influenza dynamics. This approach helped correct inflated standard errors due to autocorrelation and ensured a more accurate inference.

RESULTS

A total of 404,948 influenza and ILI cases were reported across six Western U.S. states: AZ, CO, MT, NV, OR, and WA, between January 1, 2010, and December 31, 2019 (Table S1). AZ, CO, MT, and OR reported laboratory-confirmed influenza case counts, while NV and WA reported ILI cases, which reflect total surveillance data and are not specific to the influenza virus alone. Case totals varied by year and state with the highest number of influenza cases observed in AZ and ILI cases in NV. $PM_{2.5}$ exposure data, summarized in Table 2, included annual and wildfire season means across all states over the same 10-year period. Average wildfire season $PM_{2.5}$ concentrations ranged from approximately 95 to 135 $\mu g/m^3$ across states and years, reflecting seasonal and regional variability in smoke exposure. Wildfire seasons were generally characterized by higher $PM_{2.5}$ levels compared to annual averages, highlighting the contribution of smoke events to the overall air quality. The states that reported laboratory-confirmed influenza cases (AZ, CO, MT, and OR) showed a positive correlation between a 10 $\mu g/m^3$ increase in the cumulative wildfire season $PM_{2.5}$ representing total exposure across the entire preceding wildfire season and influenza risk. For states that reported influenza-like-illness (ILI) NV showed no relationship while WA showed a negative relationship between ILI risk and wildfire season $PM_{2.5}$. A short-term association between $PM_{2.5}$ exposure and influenza risk was observed in AZ, CO, and OR. Temperature, precipitation, and humidity were also used in the analysis, but no effects were found for laboratory-confirmed influenza or ILI. The results are described in more detail next and see Table 3 and Figure 2.

Long-Term Wildfire Season $PM_{2.5}$ Exposure Effects on the Following Influenza Season (Table 3 and Figure 2)

Our analysis revealed that four out of the six states examined (AZ, CO, XMT, and OR) experienced an increased risk of laboratory-confirmed influenza during the influenza season following a 10 $\mu g/m^3$ increase in cumulative wildfire-season $PM_{2.5}$: AZ (RR = 1.061; 95% CI: 1.026, 1.100; $p < 0.001$), CO (RR = 1.067; 95% CI: 1.056, 1.078; $p < 0.001$), MT (RR = 1.038; 95% CI: 1.013, 1.063; $p < 0.001$), and OR (RR = 1.049; 95% CI: 1.041, 1.057; $p < 0.001$). In contrast, NV and WA, the two states reporting ILI counts, exhibited different patterns. NV did not reveal any relationship between a 10 $\mu g/m^3$ increase in the previous wildfire season's $PM_{2.5}$ and ILI risk (RR = 1.005; 95% CI: 0.920, 1.097; $p = 0.069$). Conversely, WA displayed a significant decrease in ILI risk associated with wildfire season $PM_{2.5}$ exposure (RR = 0.884; 95% CI: 0.842, 0.919; $p < 0.001$).

Short-Term $PM_{2.5}$ Exposure Effects Within the Current Influenza Season (Table 3)

We analyzed the short-term effects of $PM_{2.5}$ exposure across the six states during the current influenza season, focusing on lag times ranging from 0 to 4 weeks before the initial reported case. Overall, the majority of results from the short-term $PM_{2.5}$ exposure analysis (current week's $PM_{2.5}$ = lag 0, previous week's $PM_{2.5}$ = lag 1, etc.) revealed rate ratios with 95% confidence intervals were not significant, suggesting that there was no relationship between short-term $PM_{2.5}$ exposure and influenza or ILI risk after accounting for wildfire PM. There were notable exceptions observed in three of the states that reported laboratory-confirmed influenza cases. For AZ, a 10 $\mu g/m^3$ increase in the $PM_{2.5}$ 4 weeks prior was positively associated with increased influenza risk (RR = 1.067; 95% CI: 1.053, 1.123; $p < 0.001$). In CO, a 10 $\mu g/m^3$ increase in $PM_{2.5}$ 1 week prior was positively associated with laboratory-confirmed influenza risk (RR = 1.047; 95% CI: 1.020, 1.057; $p < 0.001$). And, in OR, a 10 $\mu g/m^3$ increase in $PM_{2.5}$ 3-weeks and 4-weeks prior was positively associated with increased influenza risk (RR = 1.037; 95% CI: 1.016, 1.045; $p < 0.001$ and RR = 1.028; 95% CI: 1.007, 1.027; $p < 0.001$, respectively).

DISCUSSION

We found that in the Western USA, laboratory-confirmed influenza is positively associated with the previous wildfire season's $PM_{2.5}$ exposure. This analysis, which represents large collaborative data sets in terms of both geographic extent and number of samples, provides critical insights into the relationship between $PM_{2.5}$ exposure and laboratory-confirmed influenza vs ILI risk, discussed in more detail below.

Study Timing and Relevance

This study was conducted using data from 2010 to 2019, prior to the onset of the COVID-19 pandemic. This is important to note, as the years following 2019 have seen both increases in wildfire smoke exposure across many of the study states and significant disruptions to influenza patterns and respiratory illness reporting due to pandemic-related public health measures, viral interference, and changes in population behavior.[52] A post-2019 study might yield different results due to these shifts in exposure and disease dynamics.

Wildfire Season $PM_{2.5}$

Our study focused on six states with distinct wildfire seasons, with the longest period estimated for AZ (May−October), then NV and CO (June−September). Three states have an estimated 3-month window period; MT, OR, and WA (July−September), as detailed in Table S1. Although climate shifts have altered the frequency and intensity of wildfires in recent years, we applied a consistent wildfire season definition across the study period, guided by EPA qualifier coding and observed smoke activity as described by Landguth et al. (2020) and others.[35,53] We estimated wildfire-specific $PM_{2.5}$ exposure using a spatial overlay of NOAA's HMS smoke plumes with 1 km gridded daily $PM_{2.5}$ concentrations.[49] While this approach allows for high-resolution estimates, it is subject to limitations. HMS relies on the visual interpretation of satellite imagery and may misclassify light smoke plumes. We opted to include all plume intensities to avoid excluding lower-level but chronic exposures, although this introduces uncertainty. Additionally, daily $PM_{2.5}$ values were aggregated to weekly means to align with influenza data, and 1 km $PM_{2.5}$ data were averaged to the county level. These aggregation steps, while necessary for consistency, can smooth over within-county and short-term variability and may bias effect estimates, particularly in geographically large or heterogeneous counties. Nevertheless, the health implications of $PM_{2.5}$ exposure, especially during wildfire events and during periods of the year, are well-documented. Recent literature reviews have consistently linked

https://doi.org/10.1021/EHP.6c00326
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wildfire smoke exposure to a spectrum of adverse health outcomes, including increased mortality and morbidity associated with hospitalizations and emergency department visits for respiratory and cardiovascular conditions.[54−56]

Wildfire Season $PM_{2.5}$ and Influenza Risk

Our study focused on the relationship between prolonged $PM_{2.5}$ exposure during wildfire seasons and subsequent influenza risk, which is increasingly relevant given the escalating frequency and intensity of wildfires in the Western USA.[57] The toxic nature of wildfire smoke, which contains a unique mix of chemical components compared to other $PM_{2.5}$ sources, underscores the potential for more severe health impacts, as supported by previous studies.[58,59] We observed that a 10 $\mu g/m^3$ cumulative increase in wildfire-season $PM_{2.5}$ was associated with increased influenza risk in AZ, CO, MT, and OR, aligning with past findings in MT by Landguth et al. (2020), who identified a significant long-term effect of wildfire smoke on influenza incidence in MT, while short-term effects were not significant.[35] Importantly, this 10 $\mu g/m^3$ value reflects the total sum across the wildfire season, not a daily or weekly mean. Though this may seem small, cumulative exposure over weeks or months is biologically relevant and likely more reflective of sustained respiratory system stress. Additionally, a study in Seeley Lake, MT, found that long-term exposure to wildfire smoke could lead to lasting decreases in lung function, even two years after the event,[34] further supporting the importance of considering long-term health impacts.

Wildfire Season $PM_{2.5}$ and ILI Risks

Our study's findings regarding WA and NV reveal that wildfire season $PM_{2.5}$ exposure was not positively correlated with an increase in ILI cases, which diverges from the expected outcomes based on previous research. First, we acknowledge that more investigation is needed to warrant a concrete conclusion on these different findings between flu and ILI. However, the discrepancy between the effects of $PM_{2.5}$ on laboratory-confirmed influenza and ILI could also be attributed to a variety of reasons. For example, pollutants may interact differently with respiratory pathogens or conditions. $PM_{2.5}$ is a complex mixture of particles whose specific composition varies by source, such as wildfire smoke, which may influence its impact on different respiratory infections. Influenza, caused by specific viruses, may be more susceptible to the inflammatory responses induced by $PM_{2.5}$, whereas ILI encompasses a broader range of respiratory pathogens and conditions that may not respond uniformly to particulate matter exposure. Another consideration is the difference in surveillance systems used across states. While AZ, CO, MT, and OR reported laboratory-confirmed influenza cases, NV and WA reported ILI cases based on syndromic surveillance using the CDC definition: fever ($\geq$100 °F) and cough and/or sore throat without a known alternative cause.[43] These two types of data differ in specificity, sensitivity, and the infrastructure required for collection. Additionally, public health reporting practices, access to diagnostic testing, and healthcare-seeking behavior vary across states and may influence case counts. For instance, states with more robust surveillance systems or higher levels of public engagement may capture more cases. We did not identify formal changes in case definitions during the study period, but acknowledge that undocumented shifts in practice could have occurred. The observed discrepancies in reported influenza and ILI case counts, particularly the higher totals in AZ and MT compared to more populous states such as CO, OR, and WA may reflect these systemic differences rather than true differences in disease burden. As a result, comparisons across states and especially between states reporting different outcome types should be interpreted cautiously.

Research has shown that the effects of $PM_{2.5}$ on respiratory health can vary depending on the composition of the particulates and the presence of other pollutants, such as SO_2 and NO_2.[14] One study found that copollutants like SO_2 was associated with increases in laboratory-confirmed influenza but displayed no significant relationship with ILI cases.[14] While many studies have shown an association between increased $PM_{2.5}$ and ILI cases,[60−62] there have been a few studies suggesting that $PM_{2.5}$ has a negative association with risk of ILI cases[63,64] while other pollutants had positive correlations.[65] Seasonal and environmental factors, such as temperature, humidity, and precipitation can further complicate this relationship, as it is well-established that the variables play pivotal roles in shaping the timing and prevalence of influenza cases.[66−68] One study found that precipitation can facilitate the deposition of airborne particulates, effectively removing $PM_{2.5}$ from the atmosphere.[69] Another study found that exposure to dry air compromises the immune system's ability to defend against influenza infection.[70] Another key factor that might explain the lack of positive correlation in WA and NV is the effectiveness of public health infrastructure and population behavioral responses during wildfire events. Studies have reported that individuals often alter their behavior, seeking shelter indoors, using air filtration systems, limiting travel, and reducing social contact which can reduce exposure to wildfire-related $PM_{2.5}$, as well as circulating respiratory pathogens.[71,72] These protective behaviors likely diminish the risk of both ILI and laboratory-confirmed influenza given their overlapping transmission pathway. Therefore, the absence of a positive association in these states may reflect not a lack of biological effect but rather the mitigating influence of collective behavioral responses that reduce both pollutant exposure and infection risk.

Short-Term $PM_{2.5}$ Exposure Effects

The analysis of short-term $PM_{2.5}$ exposure during the influenza season across various states reveals nuanced and region-specific effects on health outcomes. Previous studies have consistently suggested that elevated $PM_{2.5}$ levels are associated with an increased number of influenza cases within a short period, typically within a week of exposure.[14,18,21,47,73] After accounting for the impact of wildfire PM in the preceding wildfire season period, the additional impact of short-term PM was not consistently observed at associations at the 0 week lag for any of the states analyzed, with all rate ratios remaining below 1. This suggests that, within the immediate time frame following $PM_{2.5}$ exposure, there may not be a strong or direct link to the increased risk of influenza or other health outcomes in the regions we studied. Our findings align more closely with studies that suggest potential delayed effects of $PM_{2.5}$ exposure. For example, OR displayed significant rate ratios at the 3-week and 4-week lags, supporting the notion that while immediate effects may not be evident, there could still be an elevated risk of adverse health outcomes over a slightly longer period.[74,75] This delayed response might indicate that the impact of $PM_{2.5}$ on health outcomes, such as influenza, could manifest with some lag, possibly due to underlying biological processes or other contextual factors that were not fully captured in our analysis.

https://doi.org/10.1021/EHP.6c00326
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Climate and Regional Variations

Our study covered a vast and diverse geographical area, with six states spanning four different climate regions as defined by the National Oceanic and Atmospheric Administration.[76] These regions exhibit unique environmental conditions that significantly influence health outcomes. For example, the arid climates of AZ and NV may prolong exposure to $PM_{2.5}$ during wildfire seasons due to limited rainfall, which could explain the delayed effects observed in these states. Conversely, states like the OR, which experience higher precipitation levels, showed a subsequent increase in influenza risk at later lags. The pattern could suggest that rainfall may initially clear $PM_{2.5}$ from the air, but as conditions dry out again, the risk may resurface. Notably, one study highlighted that, on average, $PM_{2.5}$ levels decreased by 21% within the first hour after rainfall.[77] Recent research by Lau et al. highlights significant risk factors contributing to increased influenza infections, including high temperatures, both wet and dry conditions, and heavy rainfall.[78] The interplay between climate and air pollution is complex, and our findings underscore the importance of considering these regional differences and interactive effects when developing public health strategies.

We also observed unexpected differences in influenza case burden that do not track with the population size. Despite having larger populations, CO, OR, and WA reported substantially fewer cumulative influenza cases (22,930; 22,484; and 36,701, respectively) than AZ (144,657), MT (76,648), and NV (84,428). These patterns, outlined in Table 1, may reflect differences in surveillance systems, health-seeking behavior, vaccination uptake, or the effectiveness of public health messaging during wildfire seasons.[71,79] Environmental factors such as temperature and precipitation patterns could also modulate both $PM_{2.5}$ exposure and viral transmission potential.[68] Differences across states may also be shaped by population-level exposure patterns. For example, Montana has experienced widespread smoke events for many years, while OR and WA's coastal cities have had limited direct exposure during the study period. Because large metropolitan areas like Portland and Seattle dominate statewide population and health surveillance data, their relative lack of wildfire smoke exposure could dilute associations.

Importantly, because climate, population density, influenza data sources, and wildfire activity differ by state, we modeled each state independently to best account for local variability. Based on model diagnostics, including residual deviance and Quasi-AIC, we selected different seasonality adjustment methods (e.g., Fourier terms vs natural splines) and climate covariate structures by state (Table S2). While this approach improved model fit and interpretability for each jurisdiction, it may limit direct comparison of effect estimates across states. Differences in model structure, especially how seasonality and interactions with climate were handled, could contribute to the variation in observed associations. Therefore, comparisons across states should be interpreted with caution, keeping in mind the contextual and methodological differences inherent in a regionally tailored modeling approach. These findings underscore the complex interplay among wildfire smoke, climate conditions, and infectious disease dynamics. They also highlight the importance of localized data and context-specific modeling to inform public health strategies and exposure risk assessments.

CONCLUSIONS

Our study provides insights into the relationship between $PM_{2.5}$ exposure, climate variables, wildfire seasons, and influenza incidence across six western USA states. Furthermore, this work prompts consideration of the effects of different air pollution exposure sources on different respiratory conditions. Future research should continue to explore the mechanisms driving regional variations in response to wildfire smoke and on different respiratory and immune responses to inform more effective and region-specific health interventions in the face of increasing wildfire activity.

ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/ehp.6c00326.

Tables summarizing influenza data, wildfire season estimates, explanatory variables, and $PM_{2.5}$; figures of weekly temperature and precipitation by state (DOCX)

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https://doi.org/10.1021/EHP.6c00326
Environ. Health Perspect. 2026, 134, 397−407

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Notes

The authors declare no competing financial interest.

Significance Statement. Influenza risk can increase with more exposure from the previous season's wildfire smoke, with additional risk observed in some states with short-term (<4 weeks) $PM_{2.5}$ exposure. However, states that only reported influenza-like-illness cases (Nevada, Washington) did not follow the same pattern as in the states with influenza-only reported cases (Arizona, Colorado, Oregon, Montana). This study was supported through robust modeling of health department influenza and influenza-like-illness data from six Western USA states to identify if longer-term wildfire-specific and shorter-term air pollution exposure affects influenza or influenza-like-illness risk, contributing to our understanding of the environmental determinants of influenza risk.

■ ACKNOWLEDGMENTS

We thank all of the state health departments who responded to our data requests including Arizona State Health Department (Liam Hicks, Xandy Pompa), California (Erin Murray), Colorado (Nisha B. Alden, Elizabeth Austen), Montana Department of Health and Human Services (Stacey Anderson), Nevada Division of Public and Behavioral Health, Office of the State Epidemiologist (Maximilian Wegener), New Mexico (Samuel Scherber), Oregon Health Authority, Office of Health Analytics, Hospital Reporting Program (Benjamin Chan), Washington (Vivian Hawkens and Anna Unutzer). Computational resources from the University of Montana's Computational Ecology Lab, Griz Shared Computing Cluster (NSF 2018112, NSF 1925267), and Data & Modeling Core contributed to this research (NIH P20GM130418).

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https://doi.org/10.1021/EHP.6c00326
Environ. Health Perspect. 2026, 134, 397−407