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Air pollution and suicide in rural and urban America: Evidence from wildfire smoke

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Air pollution poses well-established risks to physical health, but little is known about its effects on mental health. We study the relationship between wildfire smoke exposure and suicide risk in the United States in 2007 to 2019 using data on all deaths by suicide and satellite-based measures of wildfire smoke and ambient fine particulate matter ($PM_{2.5}$) concentrations. We identify the causal effects of wildfire smoke pollution on suicide by relating year-over-year fluctuations in county-level monthly smoke exposure to fluctuations in suicide rates and compare the effects across local areas and demographic groups that differ considerably in their baseline suicide risk. In rural counties, an additional day of smoke increases monthly mean $PM_{2.5}$ by 0.41 $\mu g/m^3$ and suicide deaths by 0.11 per million residents, such that a 1-$\mu g/m^3$ (13%) increase in monthly wildfire-derived fine particulate matter leads to 0.27 additional suicide deaths per million residents (a 2.0% increase). These effects are concentrated among demographic groups with both high baseline suicide risk and high exposure to outdoor air: men, working-age adults, non-Hispanic Whites, and adults with no college education. By contrast, we find no evidence that smoke pollution increases suicide risk among any urban demographic group. This study provides large-scale evidence that air pollution elevates the risk of suicide, disproportionately so among rural populations.

air pollution | mental health | suicide | wildfire smoke | environmental economics

Significance

Most of the global population is regularly exposed to unhealthy levels of air pollution. Emerging evidence suggests that this exposure is not only detrimental to physical health but to mental health as well. Using drifting smoke from wildfires as the basis of a natural experiment in the United States, we provide nationally representative causal evidence that air pollution exposure increases suicide rates, with effects being strongest among rural populations who have a higher baseline risk of suicide and higher exposure to outdoor air. These results provide important insight for identifying and protecting vulnerable groups and for accurately quantifying the full costs of air pollution and wildfires.

Air pollution poses a major threat to human health and well-being (1, 2). Long recognized for its impacts on physical health, air pollution exposure has also been linked to altered emotional states, impaired cognitive functioning, aggressive behavior, and lost productivity (3–8). These findings suggest that air pollution exposure could harm mental health, either directly through brain inflammation and oxidative stress (9, 10) or indirectly through economic or other physical hardship. Indeed, emerging evidence links air pollution to mental health problems, including anxiety, depression, and suicide (11–14). Reflecting a "national mental health crisis" in the United States (15), suicide rates have increased by approximately 30% over the past two decades, positioning suicide as the fourth leading cause of years of potential life lost before age 65 in 2020 (16–18). Suicide rates are both highly unequal across demographic groups and systematically higher in rural counties than in urban ones (Fig. 1*A*), and the urban–rural gap has been widening (Fig. 1*C*) (19). Understanding the overall and disparate impacts of air pollution on mental health is crucial for developing effective strategies to protect vulnerable groups and increase population resilience to poor air quality.

This paper studies how air pollution from drifting wildfire smoke affects suicide risk in the United States and how the effects differ between urban and rural areas. Wildfires provide a valuable setting to study the link between pollution and suicide for three main reasons. First, wildfires are a major source of air pollution, accounting for about 20 percent of US emissions of fine particulate matter [particles with a diameter less than 2.5 μm ($PM_{2.5}$)], the component of wildfire smoke of greatest concern to public health (20–22). Wildfires are also expected to become more frequent and severe in the coming decades due to a combination of climate change and ongoing human development in previously wild areas (23, 24), highlighting the growing importance of understanding the impacts of wildfire smoke pollution specifically.

Second, drifting wildfire smoke creates a "natural experiment" to identify the causal relationship between air pollution and suicide. Previous research has found that ambient concentrations of particulate matter pollution correlate positively with suicide deaths (13, 25). However, such correlations do not necessarily characterize the causal pollution–suicide relationship due to the potential for other "third" factors such as holidays, economic activity, and other human behaviors to be common causes of pollution concentrations and suicide (26). Our empirical strategy addresses this limitation by isolating variation in air pollution and suicide outcomes caused by drifting wildfire

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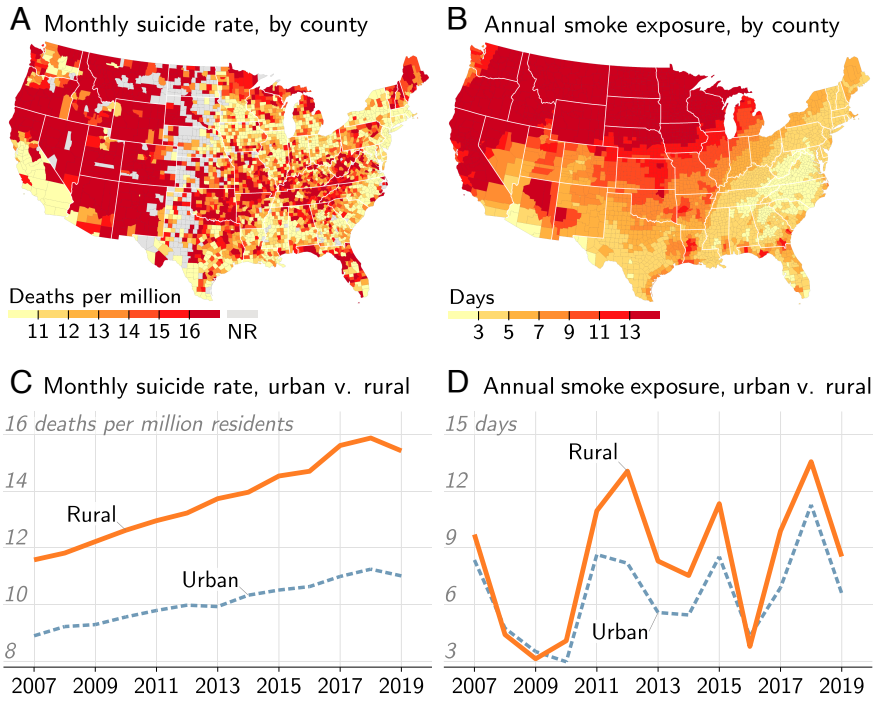

Fig. 1. Trends and spatial variation in suicide rates and smoke days for the contiguous US in 2007 to 2019. (*A*) County-level monthly suicide rates (deaths by suicide per million residents). Values are not reported (NR) for counties with fewer than 10 deaths by suicide over the study period. (*B*) Average number of days per month with medium or thicker smoke coverage, by county. (*C*) Monthly suicide rates for urban and rural counties. Counties are classified using the six-level 2013 National Center for Health Statistics Urban–Rural Classification Scheme. We define urban counties as those that are classified as large central metropolitan, large fringe metropolitan, or medium metropolitan. We define rural counties as those that are classified as small metropolitan, micropolitan, or noncore. (*D*) Average number of days per month with medium or thicker smoke coverage, by urban versus rural counties.

smoke, which is plausibly unrelated to such third factors and therefore provides more reliable evidence of a causal relationship (27).

Third, wildfire smoke plumes are tracked via satellite sensors, which capture plume locations and thickness across all parts of the contiguous United States. We combine the smoke plume measures with data on all suicide deaths in the United States to produce a nationally representative and more precise estimate of the pollution–suicide relationship compared to previous associational studies that have generally relied on much smaller samples based on individual cities or small countries (13, 25). One contemporaneous working paper linking high-polluting wind directions to increases in suicide deaths uses a nationwide sample in the United States in 2003 to 2010 (14). By comparison, our study examines the effects of air pollution from direct measurement of smoke plume transport and explores whether and how the pollution–suicide relationship differs across rural and urban regions by demographic group (sex, age, race, and education). Exposure to drifting smoke plumes affects air quality similarly in both rural and urban areas, which facilitates attributing regional differences in the pollution–suicide relationship to differences in population sensitivity rather than the pollution source. Our approach to estimating the pollution–suicide relationship separately for rural and urban regions by demographic group provides insights into which subgroups are the most vulnerable to air pollution exposure and whether overall urban–rural differences are explained by demographic differences across areas or due to broader regional influences.

Our analysis relies on three primary data sources that allow us to measure monthly wildfire smoke exposure, suicide rates, and ambient $PM_{2.5}$ concentrations for each county in the contiguous United States in 2007 to 2019. The first data source contains satellite-based daily measurements of wildfire smoke plume locations and thickness (light, medium, or heavy) (28, 29). Our focal smoke exposure measure is a count of the number of days in a month each county was covered by medium or thicker smoke, which we hereafter refer to as "smoke days." In supplemental analyses, smoke days are based on heavy smoke or light or thicker smoke. The second data source reports monthly county-level data on all deaths by suicide in the United States derived from the mortality data files maintained by the Centers for Disease Control (30). The third data source provides satellite-based measures of ground-level ambient $PM_{2.5}$ concentrations for each county and month (31). The final analysis sample covers 3,108 counties for a total of 484,848 county-year-month observations.

Our primary empirical strategy relates year-over-year deviations from the mean smoke exposure for a given county and month-of-year (e.g., Orange County, CA in July) to the corresponding deviations in monthly suicide rates. This strategy addresses two primary sources of potential confounding. First, counties where smoke exposure is more common (Fig. 1*B*) may have systematically higher or lower rates of suicide for reasons other than smoke exposure, such as if the area is rural (Fig. 1*D*). By relying only on within-county variation over time, our approach ignores correlations between smoke exposure and suicide outcomes arising from cross-sectional differences. Second, both smoke exposure and suicide rates vary seasonally, which could yield an association due to seasonal influences other than a causal effect of smoke on suicide (32). Removing county-month means excludes correlations arising from local seasonality in smoke exposure and suicide rates from influencing our estimates. Prior studies have used similar empirical strategies to estimate the causal effects of weather (33) and wildfire smoke exposure (34, 35) on social and economic outcomes.

We implement our empirical strategy by estimating linear regression models with the suicide rate as the outcome and the number of smoke days as the focal explanatory variable. Our baseline models include county-month fixed effects, which force the estimates to rely on deviations from county-month means, and also include year-month and county-year fixed effects to control for nationwide time shocks and county-specific annual factors, respectively. We also estimate dynamic versions of the

models that add lags and leads in smoke exposure, allowing for both falsification tests (i.e., effects of current smoke on past suicide rates) and tests of delayed impacts of smoke on suicide rates. In supplemental analyses, we report estimates where the outcome is alternatively chosen to be the count of suicide deaths (in which case the model is estimated by Poisson regression), age-adjusted suicide rates, or a broader measure of mental health-related mortality that in addition to suicide includes deaths from injuries of undetermined intent and certain categories of accidents to account for variation across jurisdictions in the classification of deaths as suicide (36).

We estimate the models using a pooled sample of all counties in the contiguous United States and also separately for subsamples of rural and urban counties. For each county sample, we consider models in which the outcome is the suicide rate among the entire population or specific to a population subgroup, according to an individual's sex, age, race, and educational attainment.

To assess the relationship between smoke exposure and air quality, we also estimate versions of the models in which the outcome is ground-level ambient $PM_{2.5}$. We use the resulting estimated smoke–$PM_{2.5}$ relationship as the "first stage" of an instrumental variables (IV) approach to characterize the relationship between $PM_{2.5}$ and suicide directly.

Results

Fig. 2 shows binned scatter plots relating wildfire smoke exposure to ambient air quality and suicide rates. To create these plots, each variable was first residualized by regressing the variable on the fixed effects in our baseline regression models and obtaining the residuals, which can be interpreted as deviations from the norm for a given county and month. Observations were then grouped into 25-quantiles (subsets of equal sizes) based on residualized smoke days. For example, the 25th bin represents observations above the 96th percentile (about 3.5 more smoke days than normal). Linear trendlines are based on disaggregated observations, and their slopes are identical to the estimates from our baseline linear regression models reported in columns (3) and (6) of *SI Appendix*, Table S1*A*, which also reports estimates based on the pooled sample of all counties.

Fig. 2 *A* and *B* reveal positive and approximately linear relationships between residualized smoke days and ground-level ambient $PM_{2.5}$ concentrations in rural and urban counties, respectively, demonstrating the relevance of measured smoke exposure to air quality. The linear trendlines indicate that one additional smoke day increases mean $PM_{2.5}$ concentrations in the month of exposure by 0.41 $\mu g/m^3$ (P-value < 0.001) in rural counties and by 0.33 $\mu g/m^3$ (P-value < 0.001) in urban counties.

The bottom row of Fig. 2 shows the relationship between smoke days and suicide rates. In rural counties (Fig. 2*C*), we estimate that an additional smoke day increases suicides by 0.11 deaths per million (P-value < 0.001). The binned scatter plot shows that the largest positive deviation in smoke days (3.5 or more) corresponds to the largest positive deviation in suicide rates (0.61 deaths per million). Similarly, the largest negative deviation in smoke days (−2.9 or fewer) corresponds to the largest negative deviation in suicide rates (−0.31 deaths per million). Visually, the largest deviations in smoke exposure appear to power the overall smoke–suicide relationship; smaller deviations are consistent with the overall relationship, but less precise. In contrast to rural counties, where we observe a positive relationship between smoke days and suicide rates, we estimate a near-zero effect of −0.002 suicides per million (P-value = 0.89) in urban counties (Fig. 2*D*). Even with large deviations in smoke, there is no corresponding change in suicide rates in urban areas. The difference in estimated effects between rural and urban counties is statistically significant (P-value = 0.001). This highlights how estimates based on the pooled sample of all counties (0.031 deaths per million, P-value = 0.051, *SI Appendix*, Table S1*A*) can mask important heterogeneity in response across areas.

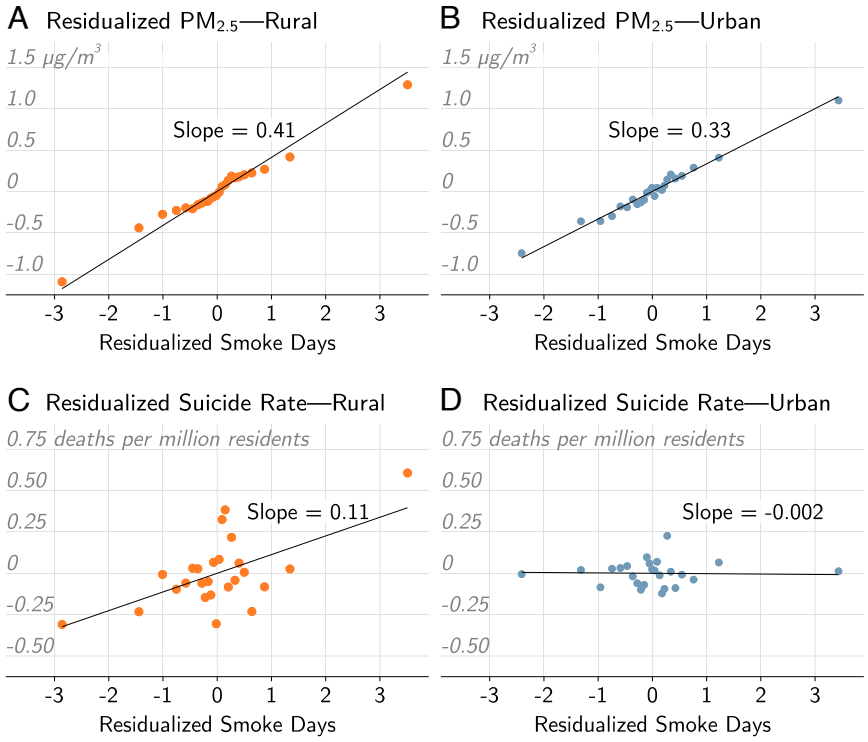

Fig. 2. Relationship between $PM_{2.5}$ concentrations/suicide rates and smoke days in urban and rural counties. The horizontal axis represents deviations in smoke days from county-month means in all four plots. Each circle represents the mean deviation of the outcome for one 25-quantile in the distribution of smoke day deviations (e.g., the farthest right point on each plot represents the mean deviation of the outcome for county-months above the 96th percentile in smoke day deviations). Linear trendlines are based on disaggregated observations, and their slopes are identical to the estimates from our baseline linear regression models reported in *SI Appendix*, Table S1*A*. (*A*) Rural counties, $PM_{2.5}$ deviations on the vertical axis. (*B*) Urban counties, $PM_{2.5}$ deviations on the vertical axis. (*C*) Rural counties, suicide rate deviations on the vertical axis. (*D*) Urban counties, suicide rate deviations on the vertical axis.

The estimated effects of smoke on suicide rates in both rural and urban counties are similar across a wide range of specifications (*SI Appendix*, Table S2), including ones with a more parsimonious set of fixed effects (e.g., county and year-month), more saturated controls (e.g., allowing the year-month fixed effects to differ across census divisions), and models that add flexible controls for local weather conditions. Estimates from Poisson models where the outcome is the count of deaths by suicide imply that an additional smoke day increases the suicide rate in rural counties by 0.10 deaths per million (P-value < 0.001) but has a near-zero effect in urban counties, aligning closely with results from the baseline linear regression models. The results are similar when using age-adjusted suicide rates, suggesting that the controls in the model sufficiently account for changes in age composition (37). When the outcome is defined using a broader measure of mental health-related mortality, of which suicides account for about half, the estimated effects of a smoke day remain similar, suggesting that suicide is the primary driver of the response in this broader category. Finally, we estimate the baseline model separately for each of the six National Center for Health Statistics (NCHS) urban–rural categories and estimate near-zero effects for each of the three urban categories and broadly similar positive effects for the three rural categories, though the estimates are imprecise for the most rural counties that have very small populations (*SI Appendix*, Table S3).

Under an assumption that the effect of smoke days on suicide operates exclusively through increased $PM_{2.5}$ concentrations, IV analysis using smoke as an instrument for $PM_{2.5}$ provides an estimate of the direct effect of $PM_{2.5}$ on suicide deaths. The IV estimate can be computed as the ratio of the "reduced form" effect of smoke on suicide to its "first-stage" effect on $PM_{2.5}$, or it can be computed using two-stage least squares regression. The first stage is well-powered, with an F statistic of at least 235 (*SI Appendix*, Table S1*A*) (38). Among rural counties, the IV estimate implies that an increase of 1 $\mu g/m^3$ in wildfire-derived $PM_{2.5}$ for a month increases the monthly suicide rate by 0.27 deaths per million (P-value < 0.001, *SI Appendix*, Table S1*A*). With a baseline $PM_{2.5}$ concentration of 7.8 $\mu g/m^3$ in rural counties, this implies that a 13% increase in $PM_{2.5}$ concentrations for a month causes monthly suicide rates in rural counties to increase by 2.0%, on average. While our main analysis uses satellite-based measures of ground-level $PM_{2.5}$ concentrations for the entire contiguous US, we find very similar estimates when using direct measurements from ground-based monitors that are available for a smaller set of counties (*SI Appendix*, Table S1*B*).

In supplemental analyses reported in *SI Appendix*, Table S1*A*, we show results in which smoke days are counts of light or thicker smoke, medium or thicker smoke (baseline), or heavy smoke. The estimated effects of smoke days on both $PM_{2.5}$ concentrations and suicide rates increase with smoke thickness, such that IV estimates of the effect of $PM_{2.5}$ on suicide rates in rural counties are broadly similar across the three definitions of smoke days. Because the vast majority of light smoke occurs far from the fires themselves, often by hundreds of kilometers, the similarly sized IV estimates based on light and heavy smoke support the hypothesis that the impact of wildfire smoke on suicide operates primarily through changes in ambient air quality rather than direct exposure to local damage caused by fires.

Fig. 3 reports the estimated effects of smoke on suicide rates for population subgroups defined by an individual's sex, age, race, and educational attainment. In urban counties, we find no statistically significant evidence that smoke increases suicide rates among any subgroup, even those with relatively high baseline suicide rates (reported in *SI Appendix*, Table S4). In rural counties, the point estimates for each subgroup are higher than their urban counterparts, and the implied impacts of smoke on suicide rates vary substantially across subgroups. The rural impacts are concentrated among men, working-age adults,

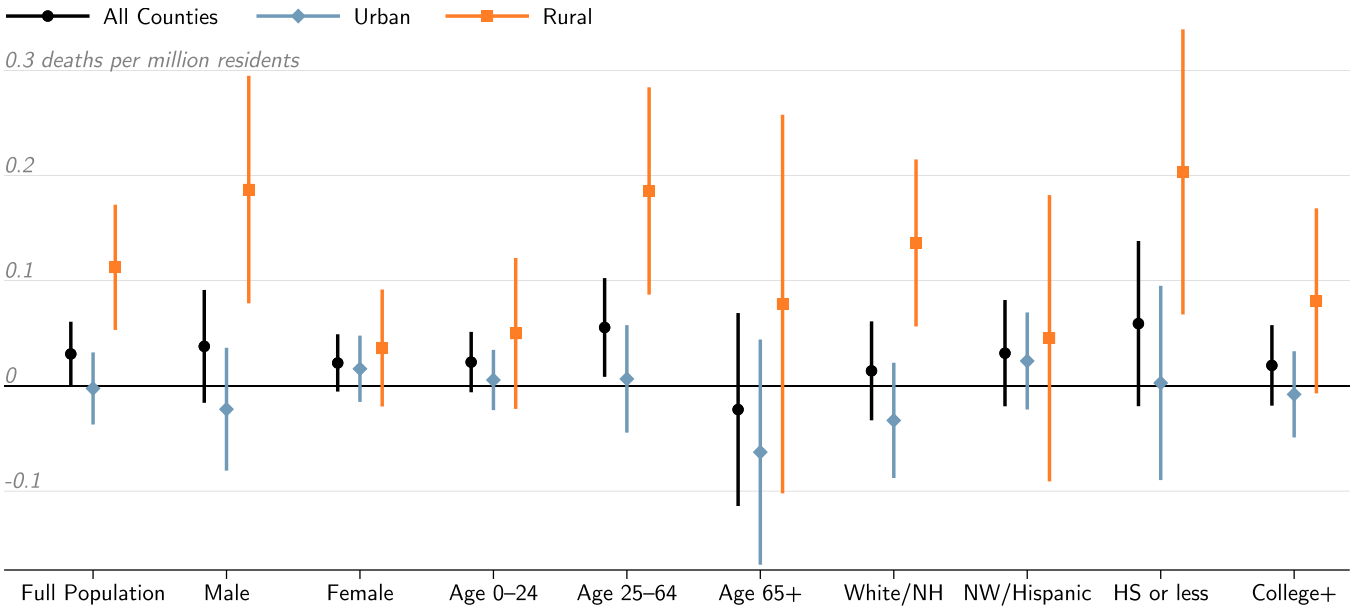

Fig. 3. Effect of one additional day of smoke on monthly suicide rates, by subgroup. Markers and lines indicate the point and 95% CI estimates of the indicated relationships, respectively. Coefficients for each population subgroup and county type are taken from separate regressions based on population subgroup-specific suicide rates in the indicated sample of counties. "White/NH" refers to individuals who are White and non-Hispanic, and "NW/Hispanic" is the converse (non-White or Hispanic). "HS or less" refers to adults 25 y and older with a high school diploma or less, and "College+" refers to adults 25 y and older with some college credits or more. Regression estimates and mean suicide rates for each subgroup are reported in *SI Appendix*, Table S4.

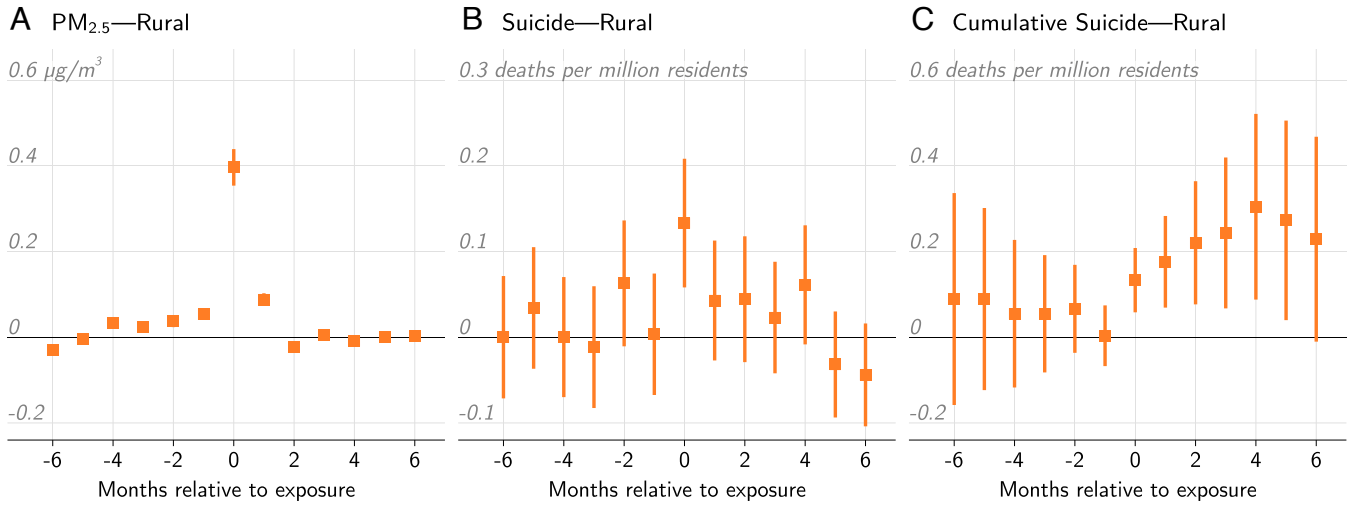

Fig. 4. Dynamic effects of smoke days on ground-level $PM_{2.5}$ concentrations and suicide rates. Dynamic regression specifications include 13 treatment variables representing smoke days in the index month, each of the 6 mo preceding the index month (lags), and each of the six months following the index month (leads). Coefficients on the leads represent the marginal effect of one additional smoke day on the outcome in preceding months (i.e., falsification tests). Coefficients on month-of and lagged smoke days represent the impact of smoke on the outcome in the current and future months (i.e., possible treatment effects). Lines represent 95% CIs. (*A*) Dynamic effects of smoke days on $PM_{2.5}$ concentrations. (*B*) Dynamic effects of smoke days on suicide rates. (*C*) Dynamic cumulative effects of smoke days on suicide rates. Dynamic cumulative effects are sums of coefficients from panel *B*. For the postexposure period, the sums begin at $t = 0$ and work forward (e.g., the cumulative estimate for $t = 2$ in panel *C* equals the sum of coefficients $t = 0$, $t = 1$, and $t = 2$ from panel *B*). For the pre-exposure period, the sums begin at $t = -1$ and work backward (e.g., the cumulative estimate for $t = -2$ in panel *C* equals the sum of coefficients $t = -1$ and $t = -2$ from panel *B*).

non-Hispanic Whites, and adults with no college education. These demographic groups face particularly high baseline suicide risks (*SI Appendix*, Table S4) and are the same groups that have been the focus of recent work on "deaths of despair" in the United States (16).

A dynamic version of the model relating rural smoke exposure to $PM_{2.5}$ concentrations (Fig. 4*A*) and suicide rates (Fig. 4*B*) shows that the effects are concentrated in the month of smoke exposure. There is no statistically significant evidence of changes in suicide rates in the 6 mo before smoke exposure, suggesting that our model appropriately accounts for pre-exposure trends. We also find no evidence of forward temporal displacement ("harvesting"), meaning the short-run increases in deaths appear to be permanent rather than being offset by later declines. This is emphasized in Fig. 4*C*, which shows the effects of smoke exposure on cumulative suicide deaths up to six months after exposure (and before exposure, as a falsification test). While the largest increases in suicides occur during the month of exposure, cumulative estimates suggest that the total impact on suicides doubles over five months before leveling off, although the five-month cumulative effect is not statistically different from the month-of-exposure effect. Taken together, these results suggest that most impacts from smoke exposure occur during or shortly after exposure, validating our focus on the single-month models for our main results. Like the static models, the dynamic models demonstrate that the effects of smoke exposure on both $PM_{2.5}$ concentrations and suicide rates increase with smoke thickness (*SI Appendix*, Fig. S1).

Discussion

Our results demonstrate that air pollution from wildfire smoke leads to excess suicide deaths in rural areas, particularly among demographic groups with high baseline suicide risk, whereas we find no evidence that smoke pollution increases suicides among any urban population. These findings highlight the value of using satellite-based measures of wildfire smoke and ambient $PM_{2.5}$ concentrations with national coverage over traditional ground-based monitor data with sparse coverage of rural areas. Quantifying the overall and disparate causal impacts of air pollution on suicide is critical to accurately characterize the full costs of air pollution and wildfires and to effectively target crisis services and mental health resources where they are needed most. Furthermore, understanding the mechanisms by which air pollution leads to increased suicide is valuable to identify and develop strategies that can improve population resilience to air pollution events. We organize this section by first discussing the magnitude of our estimates and then describing the mechanisms that best explain these effects.

To benchmark the magnitudes of the estimated effects of air pollution on suicide, we perform two comparative exercises. We first consider the following question: How much would eliminating $PM_{2.5}$ exposure close the urban–rural gap in suicide rates? If the effects of smoke pollution apply equally to all units of particulate matter, eliminating $PM_{2.5}$ from rural counties would reduce the monthly suicide rate by 2.1 deaths per million (0.27 deaths per million $\times$ 7.8 $\mu g/m^3$ baseline concentration), equivalent to 59% of the urban–rural gap in monthly suicide rates. As a second benchmark exercise, consider our central IV estimate using the pooled sample of all counties: An increase of 1 $\mu g/m^3$ in $PM_{2.5}$ for a month increases the monthly suicide rate by 0.086 deaths per million (P-value = 0.049, *SI Appendix*, Table S1*A*). This is similar in magnitude to the effect of increasing mean monthly temperature by 1 °C, which a previous study found to increase US suicide deaths by 0.066 deaths per million (39).

The IV estimates characterize the causal pollution–suicide relationship, subject to two caveats. First, the IV specification attributes all effects of wildfire smoke to $PM_{2.5}$, yet wildfire smoke is known to raise levels of multiple air pollutants (35). Second, the composition of particulate matter from wildfire smoke differs from that of other sources, with potentially important health implications (40). These caveats mean that one should interpret

our IV estimates as wildfire-specific and that $PM_{2.5}$ in our context represents the bundle of pollutants typical to wildfire smoke.

Our estimates may understate the overall burden of air pollution on mental health for two reasons. First, we focus on suicide outcomes for this study, but air pollution may worsen the severity of other mental health conditions like anxiety and depression. Populations that appear to be resilient to air pollution in terms of suicide risk (e.g., urban populations) may still face tolls on their mental health in other dimensions. Second, our study considers the short-term effects of exposure to transient air pollution events; any longer-term effects would add to the burden we measure. Considering that our findings may present a lower bound on the overall mental health toll caused by air pollution, our finding that air pollution imposes a substantial suicide burden that is concentrated among rural working-age individuals—a population that is often not considered a sensitive group—adds to a growing understanding of the wide-ranging impacts of poor air quality (41).

To understand why air pollution increases suicide and why this effect is found in rural but not urban areas, we consider which mechanisms are most consistent with the differences in pollution, suicide, and their relationship that we measure across regions and population subgroups. We first consider the role of baseline differences in the risk of suicide. Many of the subgroups with above-average suicide rates were also those whose suicide rates increased the most when exposed to smoke pollution. One possible explanation for this pattern is that air pollution exacerbates underlying suicide risk factors. If smoke pollution intensified all suicide risks equally, we would expect its effects on suicide to be proportional to baseline suicide rates. However, the urban–rural patterns we observe reject proportionality: Suicide rates were about 36% higher in rural versus urban counties during our sample period, and not only do we fail to detect an effect of smoke on urban suicides, but we also reject the null hypothesis that the effect on rural suicides is only 36% higher than the urban effect (P-value $= 0.003$). In fact, we can reject (P-value < 0.05) that the rural effect is any value less than 300% of the urban effect. Poisson estimates—which directly capture proportional changes rather than changes in levels—reveal similar findings for urban and rural areas (*SI Appendix*, Table S2), further supporting a conclusion that the urban–rural difference in marginal effects is not simply attributable to differences in underlying suicide rates.

The disproportionate effects of air pollution on suicide between urban and rural areas could be explained by demographic differences between these areas. For example, the share of adults 25 years and older with an education of high school or less is about 10 percentage points higher in rural counties than in urban counties, and the estimated effects of smoke exposure on suicide among this population subgroup are about three times larger than for adults with a college education (Fig. 3). However, our finding that, across a range of demographic subgroups defined by sex, age, race, and educational attainment, the estimated impact of smoke on suicide is higher in rural versus urban areas indicates that overall urban–rural differences are not fully explained by demographic differences between areas but rather reflect broader regional influences.

One such regional influence is that individual exposure to air pollution may be systematically higher in rural versus urban populations, even for the same level of ambient air pollution. We measure ambient levels of smoke and $PM_{2.5}$, but personal exposures under the same ambient conditions have been found to vary by a factor of 20 between individuals depending on housing, investments, behavior, and so on (42). For example, individuals in rural counties spend significantly more time outdoors and are more likely to be employed in outdoor occupations (43). Outdoor labor is particularly relevant in this setting, as the impacts of smoke on suicide are largest among groups who are most likely to be employed in outdoor industries and occupations: men, working-age adults, and individuals with no college education. The outdoor labor hypothesis is also supported by the fact that we found no statistically significant effect of smoke on suicide among the 65+ age group in rural counties, despite their relatively high baseline risk of suicide. Variation in exposure could also result from differences in protective behaviors or investments against smoke exposure. While prior research has not directly examined urban–rural differences in protective behavior, rural populations have lower average incomes compared to their urban counterparts, and previous research has found that people in areas with lower incomes are less likely to search online for the term "air filter" or stay home during a day of heavy smoke (42). Political affiliation in rural counties may also lead to fewer protective behaviors or investments in protective technology, as has been shown for other climate-related risks (44).

If rural populations are more exposed to smoke through the channels described above, these populations would likely also be more exposed to other environmental risks such as extreme temperatures. Previous studies have shown that higher temperatures increase suicide rates and other measures of mental illness in the United States (39, 45) but have not explicitly analyzed urban–rural differences. Our data allow us to replicate these previous findings separately for rural and urban counties. As with the effects of wildfire smoke, we find that the impact of increased temperature on suicide in rural counties is much larger than differences in baseline risk would predict. Specifically, the effect of temperature is more than twice as large in rural counties compared to urban counties (*SI Appendix*, Fig. S2). This finding suggests that the mental health of rural populations is especially sensitive to a broad range of environmental shocks, rather than wildfire smoke in particular. Furthermore, it is consistent with the hypothesis that the larger impacts among rural populations are at least partially attributable to higher levels of exposure to environmental risk among the at-risk populations. Aside from speaking to urban–rural differences, this analysis of temperature provides another benchmark for the magnitude of our estimated impacts of wildfire smoke: In rural counties, we find that the effect on suicide deaths from a 1-$\mu g/m^3$ increase in monthly $PM_{2.5}$ concentrations (0.27 deaths per million, *SI Appendix*, Table S1*A*) is more than twice as large as the impact from a 1 °C monthly temperature increase (0.11 deaths per million, *SI Appendix*, Fig. S2).

Materials and Methods

Mortality Data. Data on monthly deaths by suicide at the county level are derived from the restricted-use Detailed Mortality–All County data, which is part of the Vital Statistics Data maintained by the NCHS and available by request from the Centers for Disease Control (30). The raw data include all deaths reported in the United States during the study period and the primary cause of each. We use the cause of death to identify suicides and to categorize deaths as mental health-related if they were caused by suicide, injuries of undetermined intent, or an accidental death from poisoning, drowning, firearms, or trains (36). For each county and month, we count the number of suicides and mental health-related deaths, both overall and by population subgroups according to sex (male, female), age (0–24, 25–64, 65 and older), race (White and non-Hispanic, non-White or Hispanic), and educational attainment (high school diploma or less, some college credits or more). Because young people may not have had the opportunity to gain a college education, we limit our analysis to adults 25 y

and older when counting deaths by educational attainment. Death rates are calculated as the number of deaths per million population; group-specific death rates are calculated using group-specific population counts (described below). Observations for counties that experience fewer than ten suicide deaths during the study period are suppressed from Fig. 1*A*, but are otherwise used in the analysis.

Smoke Data. Daily measurements of wildfire smoke plume locations and thickness (light, medium, and heavy) were originally developed in ref. 29 using wildfire smoke analysis produced by the National Oceanic and Atmospheric Administration's Hazard Mapping System for the period 2007 to 2019 (28). Smoke analysts process imagery from nine satellites in both the visual and infrared spectra to outline smoke plume contours, which they categorize as light, medium, or heavy density with average particulate concentrations of roughly 5, 16, and 27 $\mu g/m^3$, respectively (*SI Appendix*, Fig. S3 shows the raw smoke polygons for 31 July 2016). The daily-level smoke polygons are converted into daily county-level observations indicating whether the county was covered (20% or more) by smoke and the thickest category of smoke (light, medium, or heavy) to which it was exposed. To match our unit of analysis, we aggregate the daily data to the monthly level by summing the number of days each county was covered by a light, medium, or heavy smoke plume. In our main analysis, we define "smoke days" as the number of days in which a county was covered by a medium or thicker smoke plume.

$PM_{2.5}$ Data. We use satellite-derived estimates of average monthly ground-level $PM_{2.5}$ concentrations that are constructed by combining satellite measurements of aerosol optical depth readings with a chemical transport model and calibrated to ground-based pollution monitors (31). The raw data are monthly level files gridded at the $0.01° \times 0.01°$ resolution (approximately 1.1 km × 1.1 km at the equator). We aggregate these gridded files to the county level to match our unit of analysis.

For supplemental analyses, we also use monitor-based ambient $PM_{2.5}$ data from the EPA's Air Quality System. We calculate a county's air pollution as the inverse-distance-weighted average of all valid readings from monitors within a 20-mile radius of the county. The pollution measure is missing for counties in which the nearest monitor with a valid reading is outside the 20-mile radius.

Other Data. County-level population data are used to calculate suicide rates and for regression weighting. County-year data on population by sex, age, and race are derived from the Surveillance, Epidemiology, and End Results Program (46). We supplement this with county-level educational attainment data from the Economic Research Service branch of the US Department of Agriculture (47) that report the share of adults 25 y and older in each educational attainment group (high school or less, some college or more) for 2000 and a 5-y average for 2016 to 2020. Within each county, we linearly interpolate between 2000 and 2020 to construct annual estimates of county-level educational attainment. We then calculate the number of adults 25 y and older in each group as the product of the educational attainment shares and the total population 25 y and older.

Temperature and precipitation data are obtained from the PRISM Climate Group (48), which reports daily weather values for points on a 4 km × 4 km grid for the United States. We aggregate the data to the county level by taking a weighted average of daily temperature and precipitation for all grid points within a county, where the values for each grid point are weighted by the inverse of the squared distance from the grid point to the county's population centroid.

Counties are classified as "urban" or "rural" based on the 2013 NCHS Urban–Rural Classification Scheme (49). The NCHS classifies counties into six categories. We classify counties as urban if they are in the three most urban categories (large central metropolitan, large fringe metropolitan, and medium metropolitan), and rural if they are in the three least urban categories (small metropolitan, micropolitan, and noncore).

Empirical Approach. We use a panel fixed effects regression to estimate the effect of smoke exposure on suicide rates:

$$Y_{cym} = \beta \text{Smoke}_{cym} + \delta_{cm} + \delta_{cy} + \delta_{ym} + \epsilon_{cym}, \quad [1]$$

where Y_{cym} is the suicide rate (deaths by suicide per million population) in county c, year y, and month m. Smoke_{cym} is the number of days covered by medium or thicker smoke in the same county-year-month. δ_{cm} are county-by-month of year fixed effects; the inclusion of these fixed effects subtracts county-month means from both suicide rates and smoke days. As such, we compare deviations in suicide rates between county-months (e.g., Boise County in July) in relatively smoke-free years (e.g., in July 2015, Boise County experienced 1 smoke day) with the same county-month in relatively smokey years (e.g., in July 2016, Boise County experienced 13 smoke days). Our main specification also includes county-by-year fixed effects, which control for all factors specific to a county that do not vary across months of the year (e.g., declining local employment opportunities) and obviate the need for county-level annually varying covariates. Finally, Eq. **1** includes year-month fixed effects which control for all factors specific to a year-month that are common to all counties (e.g., common impacts of the Great Recession). Regressions are weighted by 2007 county populations and standard errors are clustered at the county level to account for arbitrary serial correlation in the error term within counties.

Alternative specifications include varying sets of fixed effects and flexible controls for local weather conditions (*SI Appendix*, Table S2). The main specification excludes weather controls because smoke can have a causal effect on temperature and precipitation (50, 51).

To calculate the implied direct impact of $PM_{2.5}$ on suicide rates, we employ an IV approach via two-stage least squares. The first-stage estimates the relationship between smoke days and $PM_{2.5}$ via Eq. **1**; the second stage is a regression of suicide rates on the predicted values of $PM_{2.5}$ from the first-stage, including all the same controls as Eq. **1**. Interpreting the results of this approach as the causal effect of $PM_{2.5}$ on suicide rates requires the assumption that smoke exposure affects suicide solely through its impact on $PM_{2.5}$ concentrations.

We estimate a dynamic version of Eq. **1** by including six lags and leads in Smoke_{cym}. Specifically, we replace Smoke_{cym} with $\sum_{t=-6}^{6} \text{Smoke}_{c,ym+t}$. The coefficients on lagged smoke test for delayed impacts of smoke exposure on suicide rates, and the coefficients on the leads represent falsification tests (i.e., the effect of future smoke on past suicide rates).

Data, Materials, and Software Availability. Public-use data and code data have been deposited in ICPSR (52). The analysis relies on Restricted-Use Vital Statistics Data from the Centers for Disease Control and Prevention (CDC). Researchers can request access to these files by following the instructions at https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm (53).

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