

The mortality burden of airborne fine mineral dust in the United States

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Abstract

Regulation under the Clean Air Act has reduced combustion-derived fine particulate matter (PM_{2.5}) in the United States, leaving windblown mineral dust—driven by drought, shrinking water bodies, and fallowed farmland—as a growing and largely unregulated share of what remains. Existing mortality burden estimates have had to borrow concentration–response functions from total PM_{2.5} and disagree fivefold. We link satellite-derived, source-specified dust PM_{2.5} to a county-month panel of all-cause mortality across 3,038 counties from 2000–2019, identifying effects from within-county, within-season dust anomalies. Monthly mortality increases by 0.50% (95% CI 0.25–0.75%) per 1 $\mu\text{g}/\text{m}^3$ dust PM_{2.5}, controlling for co-occurring non-dust PM_{2.5}, robust to the meteorology that generates dust, and is where background air is cleanest. We attribute roughly 8,000 deaths per year to ambient dust, rising to 11,000 when the response is accumulated over three months: \$80–110 billion in annual mortality damages, one-third to one-half of recent estimates for wildfire smoke PM_{2.5} and one-tenth of total PM_{2.5} mortality in the US. Exposure falls disproportionately on Hispanic Americans and projected warming, drying, and current land and water management policies, are likely to increase dust’s impacts on health.

Introduction

Fine particulate matter, defined as particulate matter with an aerodynamic diameter $\leq 2.5 \mu\text{m}$ ($\text{PM}_{2.5}$), is associated with morbidity and mortality even at low concentrations (1, 2). In the United States, decades of regulation under the Clean Air Act have substantially reduced combustion-derived $\text{PM}_{2.5}$ (3–5), shifting the composition of pollution toward sources that lie largely outside regulatory control—principally wildfire smoke and windblown mineral dust. Smoke has drawn sustained scientific and policy attention: recent estimates attribute tens of thousands of deaths per year in the US to smoke $\text{PM}_{2.5}$ (6–8), and smoke mortality now rivals temperature-related mortality as a leading component of the domestic social cost of carbon (9). Mineral dust—derived from wind erosion of soils, lakebeds, and agricultural lands—has received far less attention, even as the forces that generate it (i.e., drought, shrinking terminal lakes, and expanding fallowed farmland) are expected to intensify under climate change and current policy (10, 11). Its mortality burden in the United States has not yet been estimated directly, constraining its inclusion in the benefit–cost analyses that guide air-quality, climate, and land- and water-management policy.

Dust is compositionally distinct from combustion-derived $\text{PM}_{2.5}$. It consists primarily of silicates, carbonates, and iron oxides and can carry adsorbed metals, pesticides, and biological agents, including the *Coccidioides* fungi responsible for coccidioidomycosis (Valley fever), a dust-borne infection largely unique to the Americas (12). Although dust is often associated with coarser particles, and in some cases is defined by its coarse particle mass (12), its fine fraction deposits in the airways and enters the systemic circulation, where redox-active iron promotes oxidative stress and crystalline silica sustains inflammation—the same pathways implicated in the cardiovascular and respiratory mortality associated with total $\text{PM}_{2.5}$ (13).

Direct evidence for the mortality effects of airborne dust in the US is scarce. Dust epidemiology has historically been concentrated in the “global dust belt” spanning North Africa, the Middle East, and East Asia (12); systematic reviews of this evidence base link short-term exposure to elevated cardiovascular and respiratory mortality (14, 15). But a scoping review of more than two hundred dust–health studies found only about 5% conducted in the Americas (16), and the US is particularly understudied (12). US dust concentrations are low by global standards, well below those common in the Sahel, the Middle East, or Central Asia; whether exposures of this magnitude carry a measurable mortality burden is uncertain. As a result, US assessments that place a societal cost on dust—most recently Feng et al.’s finding that wind erosion imposes \$154 billion in annual damages, of which mortality is the single largest component—have had to borrow concentration–response functions estimated for total $\text{PM}_{2.5}$ and apply them to modeled dust (17). The resulting estimates span 2,500 to 13,000 deaths per year (18, 19) depending on how much total $\text{PM}_{2.5}$ is apportioned to dust and how deaths are associated in nonlinear total $\text{PM}_{2.5}$ functions.

Here we provide a national-scale estimate of the dust–mortality relationship in the United States and its implied attributable burden. We link satellite estimates that separate mineral dust from other $\text{PM}_{2.5}$ components at ~ 1 km resolution over 2000–2019 (20) to a county-month panel of all-cause mortality spanning 3,038 counties and 709,133 county-months (48 million deaths; Supplementary

Table S1). We first characterize the distribution of dust exposure across the country and across racial-ethnic and income groups. We then adopt a panel fixed-effects design that identifies the mortality effect from month-to-month dust anomalies within a county and season—variation likely to be driven by episodic atmospheric transport and wind erosion rather than by local economic or demographic change—and we subject it to permutation-based falsification and to direct controls for the meteorology that generates dust. Finally, to estimate the annual burden of airborne mineral dust, we combine this concentration–response with observed ambient concentrations under a counterfactual of no dust, and monetize the attributable deaths using standard valuations.

Results

Distribution and growing contributions of dust PM_{2.5} to pollution exposures

Although dust concentrations are low by global standards, exposures are widespread across the US (Fig. 1). Mean dust PM_{2.5} averaged $0.70 \mu\text{g}/\text{m}^3$ nationally over 2000–2022—average total PM_{2.5} was roughly $10 \mu\text{g}/\text{m}^3$ and fell from roughly 13 to $8 \mu\text{g}/\text{m}^3$ over the period. Dust concentrations are elevated across the Southwest and southern Great Plains (Fig. 1a), and nearly one-fifth of the population (about 59 million people) lived where the long-run mean exceeded $1 \mu\text{g}/\text{m}^3$ (Fig. 1d). Two-week periods averaging above $2 \mu\text{g}/\text{m}^3$ recurred across the western and central US (Fig. 1b); half the US population experienced at least one such period every other year, and the most-exposed tenth experienced the equivalent of roughly six weeks of high dust each year (Fig. 1e). Dust also makes up a growing share of total PM_{2.5} as combustion sources have declined (Fig. 1c,f).

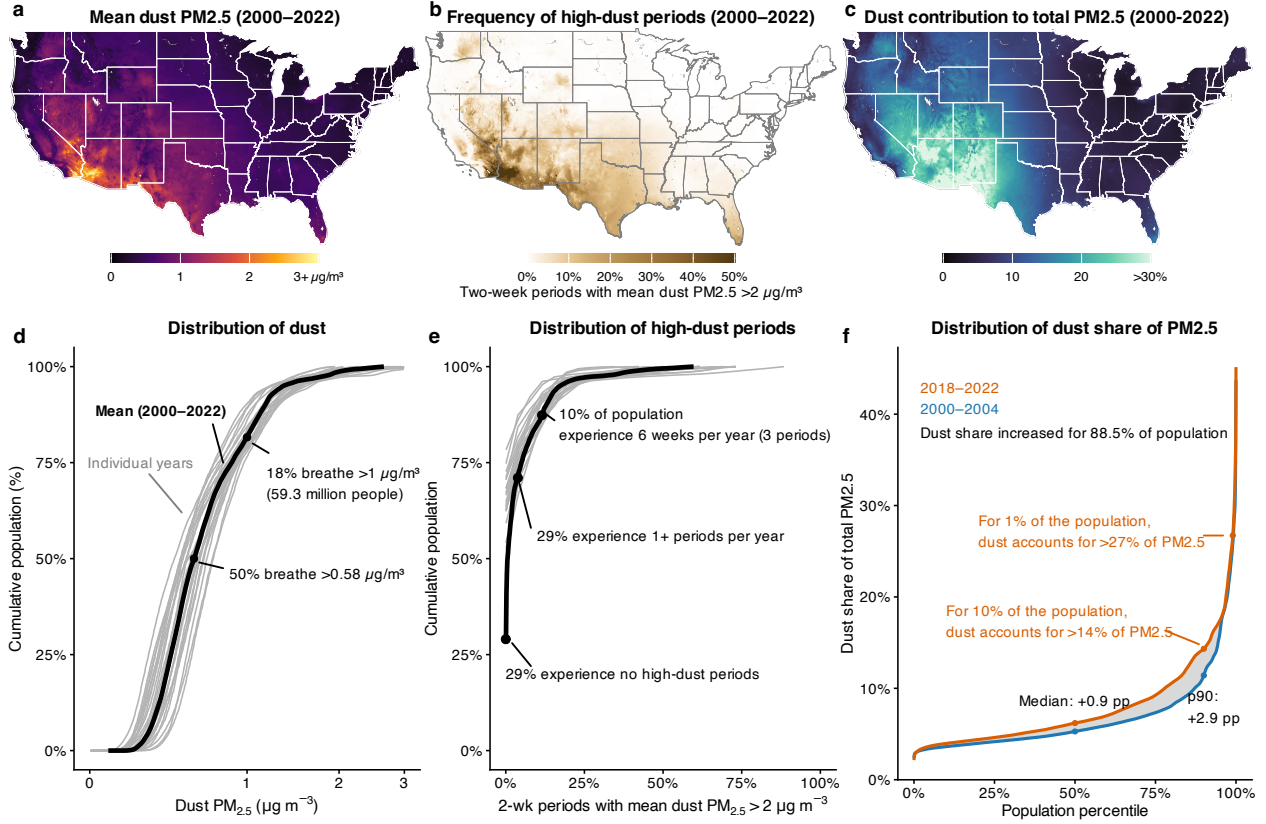


Figure 1: Dust $\text{PM}_{2.5}$ exposure across the contiguous US is widespread, episodic, and a growing share of total $\text{PM}_{2.5}$, 2000–2022. Estimates derive from the van Donkelaar et al. satellite–chemical-transport fusion product, which partitions mineral dust within $\text{PM}_{2.5}$ at $\sim 0.01^\circ$ resolution across North America and is calibrated to ground-based speciation. (a) Mean dust $\text{PM}_{2.5}$. (b) Frequency of high-dust periods (fraction of 14-day windows with mean dust $\text{PM}_{2.5} > 2 \mu\text{g}/\text{m}^3$). (c) Mean dust contribution to total $\text{PM}_{2.5}$. (d) Population-weighted cumulative distribution of long-run mean dust $\text{PM}_{2.5}$; thin grey curves show year-specific distributions, the thick black curve the 2000–2022 mean. (e) Population-weighted cumulative distribution of high-dust frequency. (f) Dust share of total $\text{PM}_{2.5}$ by population percentile, 2000–2004 (blue) versus 2018–2022 (orange).

Racial-ethnic differences in dust PM_{2.5} exposures

Dust PM_{2.5} exposure is unevenly distributed across racial-ethnic groups in the US (Fig 2). Using annual gridded population counts by race-ethnicity and household-income decile from the Census’s Gridded Environmental Impacts Frame tabulated from administrative records (Methods), we find that Hispanic Americans experienced population-weighted dust PM_{2.5} of 0.96 $\mu\text{g}/\text{m}^3$ over 2000–2022, 39% above the national population-weighted mean; American Indian and Alaska Native populations 0.81 $\mu\text{g}/\text{m}^3$ (18% above); and white Americans 0.62 $\mu\text{g}/\text{m}^3$ (10% below). These disparities persist across the entire income distribution: within every household income-decile, Hispanic and American Indian and Alaska Native populations are exposed to more ambient dust PM_{2.5} than white populations. The highest-income decile of Hispanic households is more exposed than the lowest income decile of white households (0.82 vs. 0.64 $\mu\text{g}/\text{m}^3$). Exposure varies less with income itself, with gaps ranging from +6% of the national mean in the third decile to -7% in the top decile, i.e., the Hispanic-white gap is roughly 4x the gap between the most and least exposed income deciles.

Disparities have not narrowed in the past two decades. In absolute terms Hispanic Americans were on average exposed to an additional 0.30 $\mu\text{g}/\text{m}^3$ in 2000 and 0.26 $\mu\text{g}/\text{m}^3$ in 2022; in relative terms, the gap was +38 and +40%, respectively. Hispanic populations are more exposed than their own region’s average in all four Census regions, most markedly in the South (+46%). The gap for American Indian and Alaska Native populations expanded from +7% in 2000 to +28% in 2022 (Fig. 2b), although improving administrative-record coverage may account for part of that increase (Methods); this population is more exposed than average in the West and South. County-level estimates weighted by American Community Survey populations give similar, slightly smaller disparities, and additionally show only modest exposure gradients by poverty status and educational attainment (both within 5% of the national mean) (Table S4).

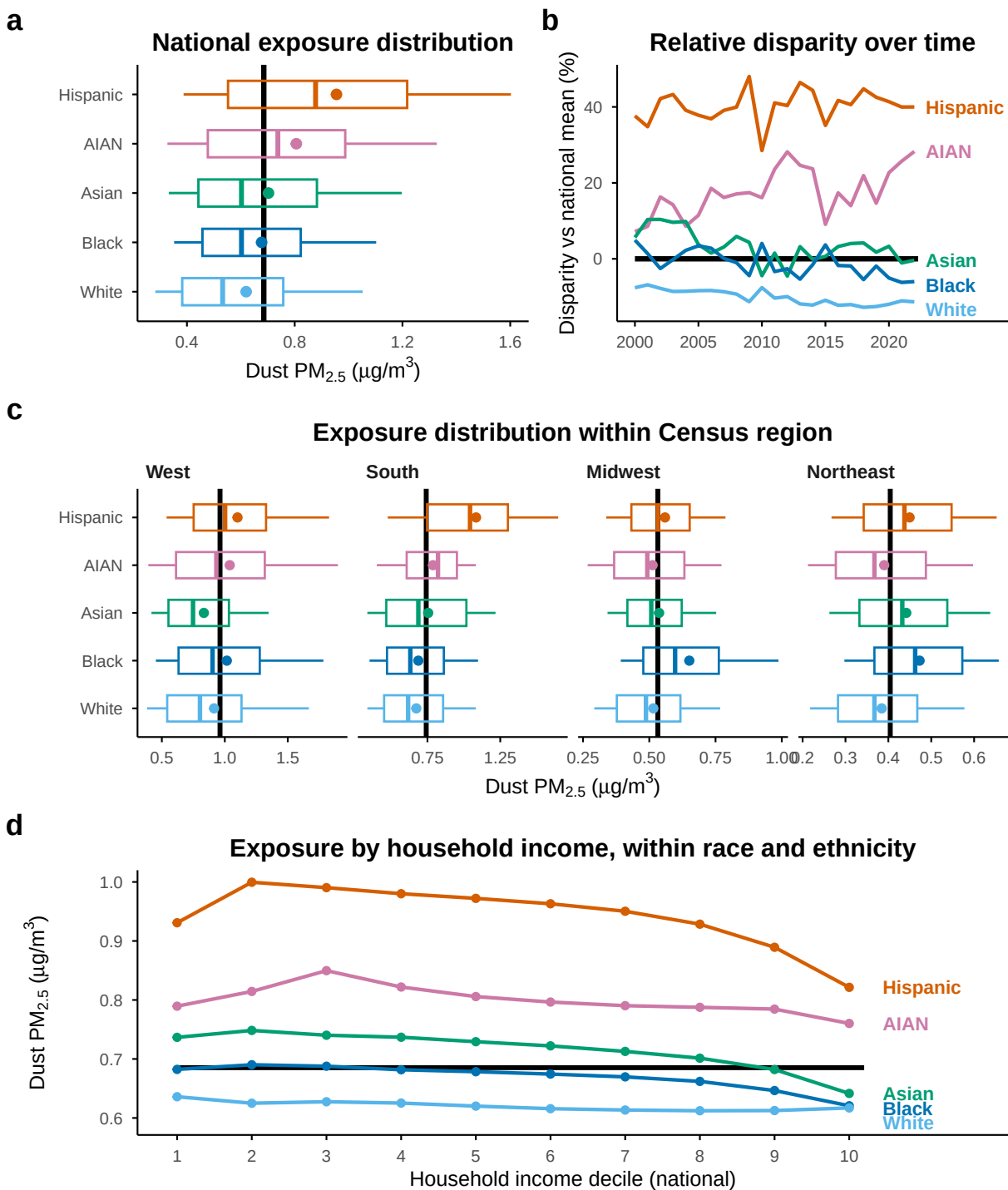


Figure 2: Dust $PM_{2.5}$ exposure by race, ethnicity, and household income, 2000–2022. Dust concentrations are the van Donkelaar et al. product; annual grid-level population counts by race-ethnicity and national household-income decile are from the Census Bureau’s Gridded Environmental Impacts Frame. **a**, National exposure distribution by racial-ethnic group: boxes span the 25th–75th percentiles, whiskers the 10th–90th, the interior line the median, and the point the population-weighted mean; the heavy black line is the mean across all people. **b**, Relative disparity over time: the percent difference between each group’s population-weighted mean and that year’s mean across all people, with population composition updated annually. **c**, The same distributions as in **a** computed within each Census region, each compared against that region’s own population mean. **d**, Population-weighted mean exposure by national household-income decile, estimated separately within each racial-ethnic group; the dashed line is the all-person mean.

Dust raises all-cause mortality across the range of routine exposure

A $1 \mu\text{g}/\text{m}^3$ increase in same-month dust $\text{PM}_{2.5}$ raised all-cause mortality by 0.50% (95% CI: 0.25, 0.75), controlling for co-occurring non-dust $\text{PM}_{2.5}$. Cumulating the response over the current and three preceding months (lags 0–3) gives 0.70% (95% CI: 0.13, 1.27) (Fig. 3b). The concentration-response function appears to be linear (Fig. 3a; Supplementary Fig. S3). A one interquartile range increase in ambient dust $\text{PM}_{2.5}$ (from 0.35 to $0.80 \mu\text{g}/\text{m}^3$) increases mortality rates by 0.22% (95%: 0.11, 0.34).

Future dust (one- and two-month leads), which serve as a negative-control exposure where a spurious dust–mortality association driven by residual within-county time-varying confounding or local trends would generally also appear at these leads, does not impact mortality (Fig. 3b). Our specification passes permutation tests of temporal and spatial confounding (Methods). Permuting each county’s dust time-series breaks the temporal link to mortality while preserving other features of the data. Across 500 replicates, the null distribution centers at zero and none exceeds the observed 0.50% per $\mu\text{g}/\text{m}^3$ effect (Fig. 3c). The complementary spatial permutation, which reassigns dust across counties within year-month cells, also centers at zero and no permutation estimate exceeds 0.50% (Fig. 3c).

Because dust is generated by wind erosion under dry conditions, the meteorology that produces it could raise mortality through other channels. Adding county-month wind speed and vapor-pressure deficit modestly attenuates our estimates (0.45% per $\mu\text{g}/\text{m}^3$; 95% CI: 0.20, 0.70). Our primary model already controls for co-occurring non-dust $\text{PM}_{2.5}$ contemporaneously; removing that control does not affect results (0.50% per $\mu\text{g}/\text{m}^3$; 0.26, 0.75). Alternative fixed effect specifications considered produce effect sizes of equal or larger magnitude. For example, a coarser structure with county, state $\times$ year, and county $\times$ month-of-year fixed effects—replacing the county $\times$ year and state $\times$ year-month terms of our preferred specification—yields 0.71% per $1 \mu\text{g}/\text{m}^3$ (95% CI: 0.46, 0.96). Our estimate is also robust to accounting for spatially correlated shocks (Conley HAC 95% CI: 0.27–0.74 at a 100 km cutoff and 0.28–0.74 at 500 km), to alternative functional forms, and to re-estimating dust at its native biweekly resolution rather than as a monthly mean (Supplementary Figs. S3–S9). We also restrict our analytic sample the sample to the 273 counties hosting an IMPROVE or EPA Chemical Speciation Network monitor, where the satellite retrieval is best calibrated against ground-based composition measurements. (Supplementary Fig S6). We further summarize robustness to unmeasured confounding with an E-value: the minimum association, on the risk-ratio scale, that a confounder would need with both dust and mortality—beyond the covariates already included—to fully explain away the estimate. Our estimated E-value (1.05–1.08) is modest, as expected for a small per-microgram effect; any such confounder would still have to operate within the county $\times$ year, county $\times$ month, and state $\times$ month cells that define our identifying variation and survive the permutation tests.

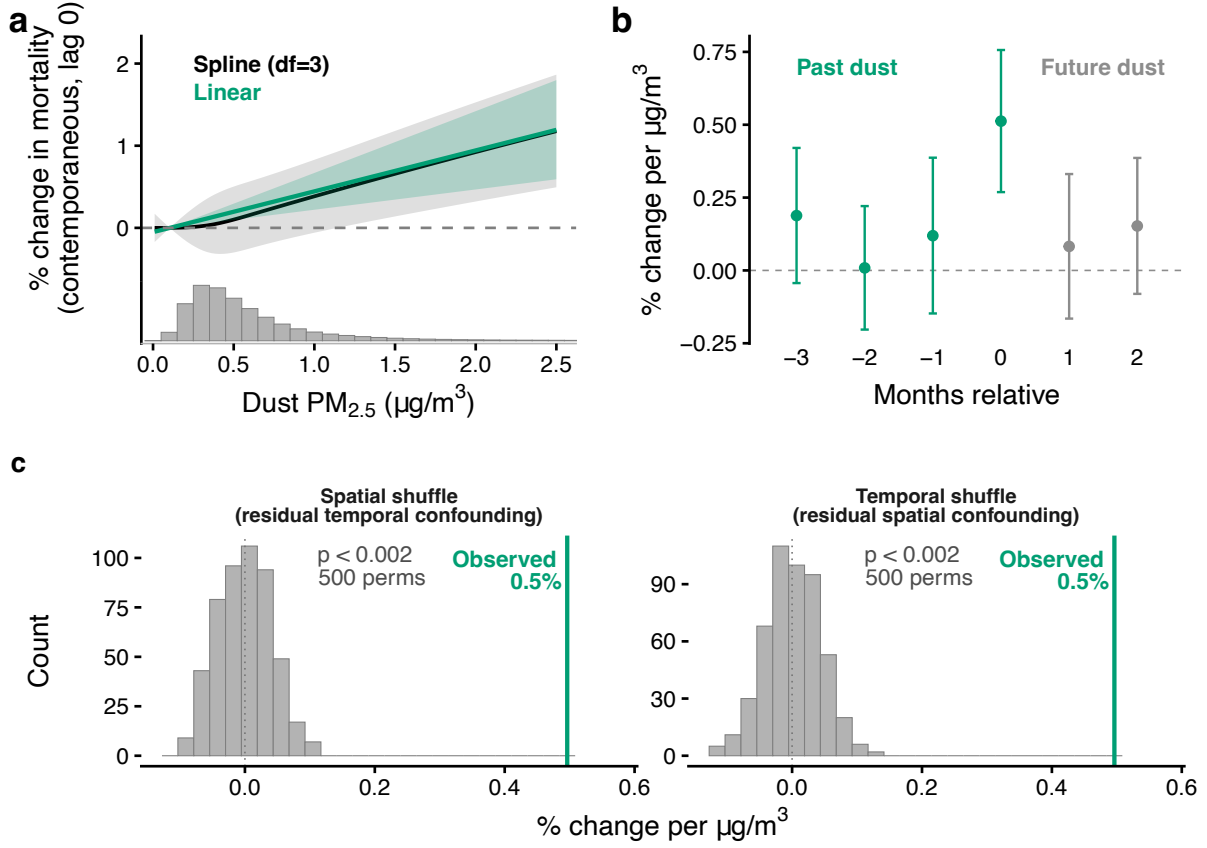


Figure 3: Concentration-response, lag dynamics, and falsification. (a) All-ages contemporaneous (lag-0) concentration-response; natural cubic spline (black) and linear model (green, 95% CI); the histogram shows distribution of county-month ambient dust PM_{2.5} concentrations. (b) Event study: the linear per-μg/m³ effect of dust entered at each monthly horizon from three months before to two months after. Future dust is null, and the effect is concentrated at the contemporaneous month, motivating the lag-0 primary specification. (c) Falsification: null distributions of the contemporaneous linear slope under 500 within-county temporal permutations and 500 cross-county spatial permutations (dust reassigned across counties within year-month); the observed 0.50% per μg/m³ (green) lies far in the tail of both.

Heterogeneity in effects by age, background PM_{2.5}, and region

Effect estimates are similar across age groups, implying larger absolute increases in mortality rates among older adults (ages ≥ 65 : 0.50%, 95% CI: 0.24, 0.76; ages 0–64: 0.47%, 95% CI: 0.00, 0.94; (Fig. 4). Stratifying counties by mean non-dust (“background”) PM_{2.5}, the effect is largest in the lowest tercile ($< 7.5 \mu\text{g}/\text{m}^3$; +1.00% per $\mu\text{g}/\text{m}^3$, 95% CI: 0.54, 1.45), and is roughly half as large in the middle (+0.28%, -0.17 , 0.73) and highest ($> 9.8 \mu\text{g}/\text{m}^3$; +0.41%, 0.01, 0.81) terciles. Interacting these terciles with average dust concentrations shows that the effect concentrates in counties that combine low background pollution with high dust (+1.16% per $\mu\text{g}/\text{m}^3$, 95% CI: 0.71, 1.62; Supplementary Table S9).

We also stratify results by region. In the South, we see a +1.51% (0.91, 2.11) per $\mu\text{g}/\text{m}^3$ effect in low-background counties but no effect in high-background counties (+0.21%, -0.55 , 0.98). Marginal Census-region slopes are individually imprecise, reflecting small within-region samples (South +0.47%, 95% CI: 0.11, 0.83; West +0.60%, 0.16, 1.05; Northeast +1.08%, 0.01, 2.17; Midwest indistinguishable from zero, -0.63 , 0.62).

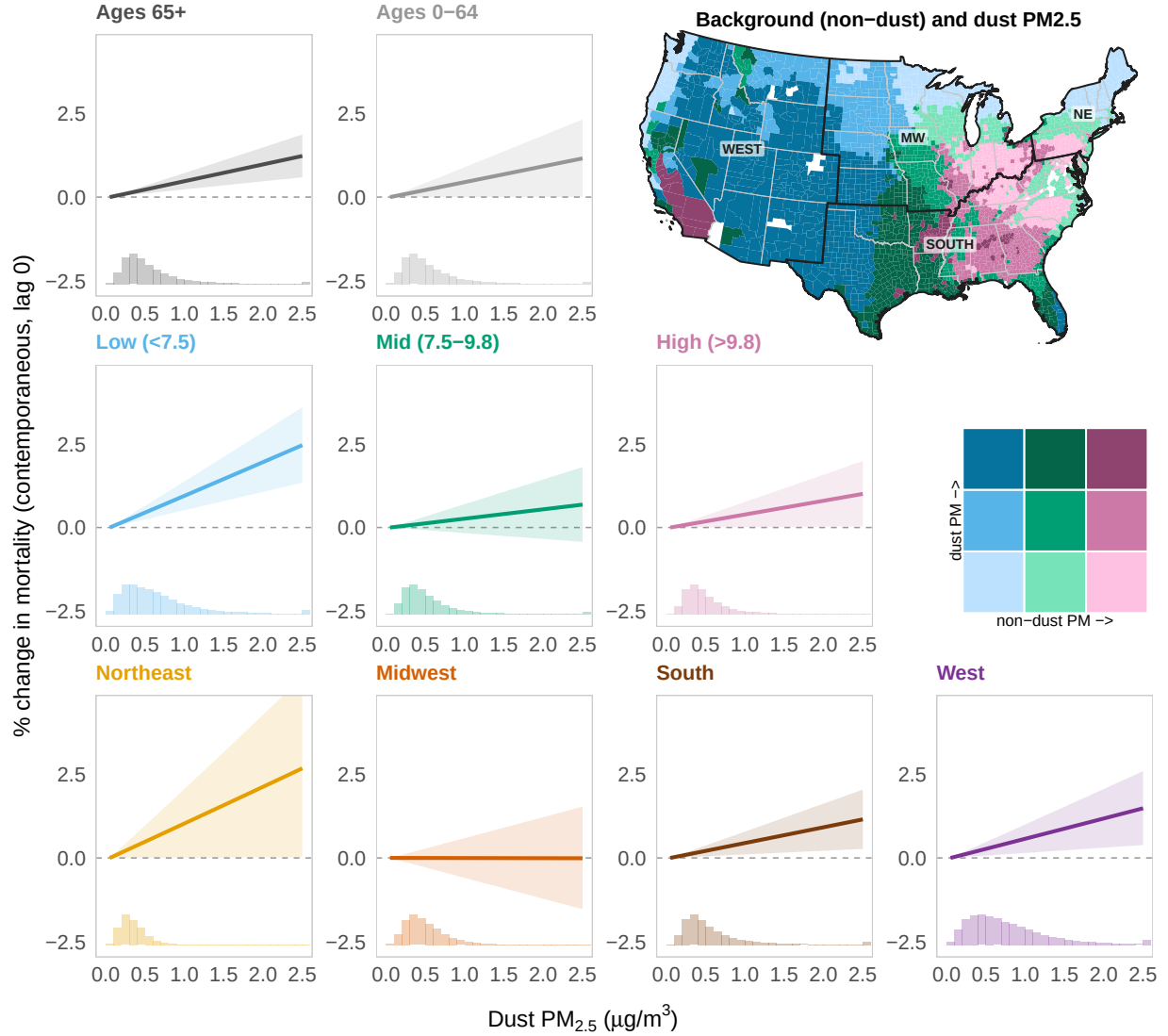


Figure 4: Concentration-response by subgroup. Contemporaneous (lag-0) all-cause mortality response to dust $PM_{2.5}$ estimated separately by age group (0-64, ≥ 65), by tercile of county background (non-dust) $PM_{2.5}$ (Low < 7.5 , Mid $7.5-9.8$, High $> 9.8 \mu g/m^3$), and by Census region. Each panel shows the linear fit with its 95% CI ribbon; embedded histograms show each subgroup's dust $PM_{2.5}$ distribution. The inset map crosses each county's mean background (non-dust) $PM_{2.5}$ with its mean dust $PM_{2.5}$ (both in tertiles; see the bivariate key), with the four Census regions outlined. Region-specific nonlinear concentration-response curves are shown in Supplementary Fig. S7.

Annual mortality attribution

Applying the primary contemporaneous (lag-0) concentration–response to observed exposure, we attribute approximately 7,760 dust-attributable deaths per year over 2000–2019 (95% CI 3,765–11,480), or 0.32% of all-cause mortality (Fig. 5). Using the cumulative three month effect estimate yields 10,870 deaths per year (1,770–19,340). Attributable mortality estimates are consistent across functional forms (4,780–7,760 deaths per year) and across models that let the concentration–response vary by age, region, or county-level income (7,420–7,910 deaths per year, Supplementary Table S8). Monetized at the EPA-default value of a statistical life (\$10.05 million, 2020 US\$), these correspond to roughly \$80 billion (contemporaneous) to \$110 billion (three month cumulative) per year in mortality damages.

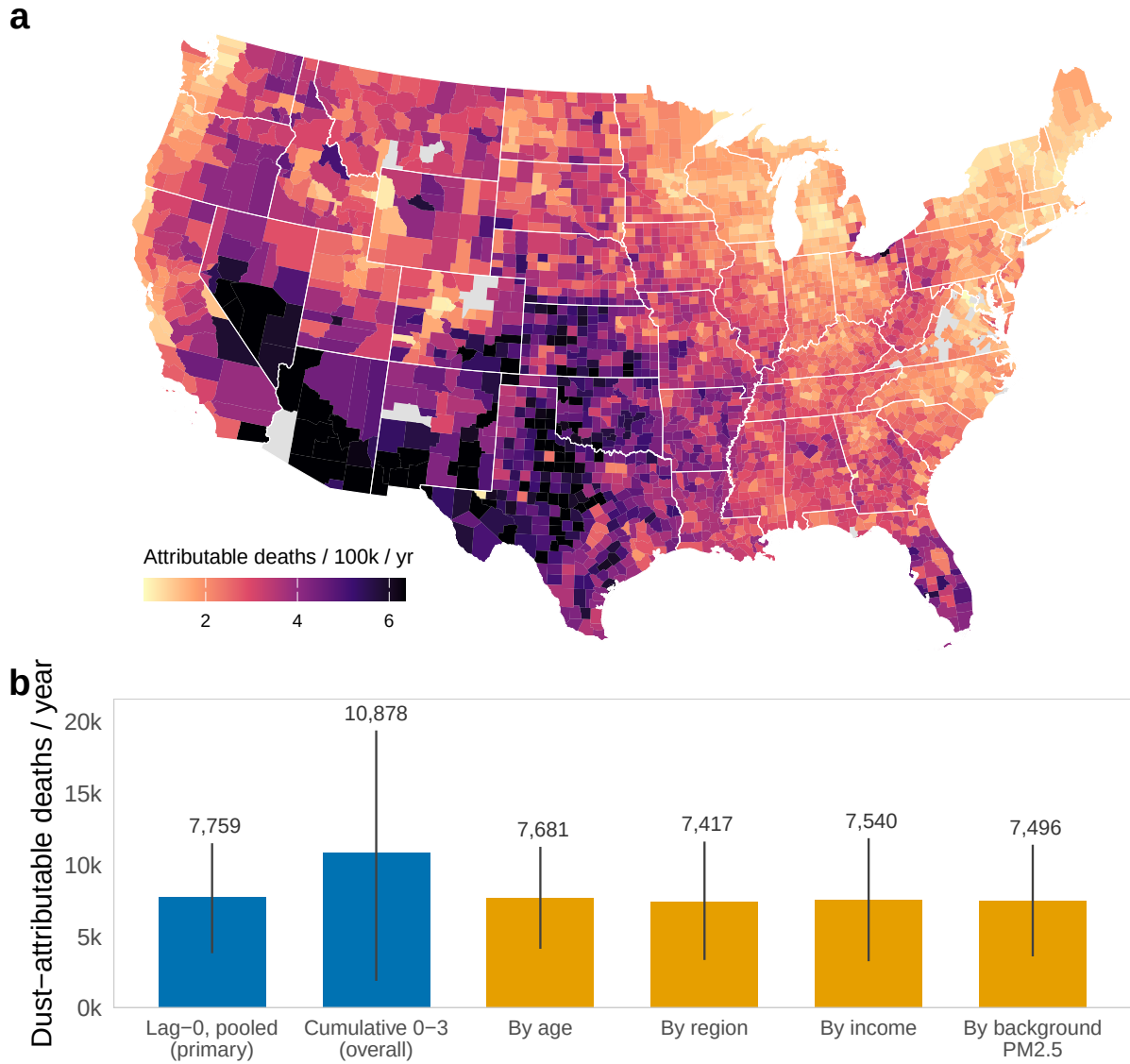


Figure 5: Dust-attributable mortality burden. (a) County-level dust-attributable deaths per 100,000 per year under the primary contemporaneous (lag-0) specification and a zero-dust counterfactual. (b) Attributable deaths per year: the primary lag-0 estimate (blue, with 95% interval), the overall cumulative (lags 0–3) estimate, and a robustness set (cumulative spline and seven-bin functional forms, and reconstructions that let the concentration–response vary by age, region, or background PM_{2.5}).

Discussion

Airborne dust harms health in the United States through several pathways. Dust storms reduce visibility and can cause fatal vehicle accidents (21), including a 2023 pileup on Illinois’s Interstate 55 that killed eight people (22). Evidence suggests that windblown soil spreads the *Coccidioides* spores, and is linked to dramatic increases in Valley Fever case rates across California, Arizona, and the broader Southwest (12, 23). Drying and water diversion have shrunk the the Salton Sea and Great Salt Lake, raising dust concentrations and health risks in the surrounding regions (24–26). Here we document rising background and episodic fine dust concentrations across much of the country, and estimate that it accounts for 8,000–11,000 deaths per year — roughly one-third to one-half of recent estimates of wildfire smoke PM_{2.5} mortality (6, 7) and one-tenth of total PM_{2.5} mortality (27).

Previous US dust mortality burden estimates take concentration–response functions estimated for total PM_{2.5} and apply them to a modeled dust concentration. The Global Burden of Disease–MAPS apportionment attributed roughly 5% of all US PM_{2.5} to dust and, allocated a proportional share of the total PM_{2.5} burden, yielding an estimate of 2,500 deaths due to ambient dust PM_{2.5} in 2017 (18). Yu et al. (19) use MERRA-2 aerosol reanalysis data to estimate fine dust concentrations across the US, and then apply those estimates to an integrated exposure–response function built from total PM_{2.5} studies and attribute 13,000 deaths in 2019 to fine ambient dust. The two differ roughly fivefold, disagreeing on how much dust the population actually breathes and how mortality burdens are allocated on nonlinear total PM_{2.5} curves. The recent WHO-commissioned systematic review and meta-analysis of short-term dust mortality studies rests almost entirely on binary dust-day comparisons and did not support a continuous concentrations–response function (15). We advance on this past work by estimating a dust-specific continuous concentration-response function in a low-dust setting.

The approximately linear response we recover is consistent with the concentration–response established for total PM_{2.5}. Those functions are supralinear overall (2, 28), but large low-exposure cohorts find the steepest slope at the lowest concentrations and no evident threshold below current standards (1, 29, 30), a range over which the curve is approximately linear. Our observed effects similarly indicate that the largest impacts of dust on mortality are where background PM_{2.5} is lowest and largest where other sources of pollution are low and dust is high. Per-microgram effects grow as the exposure-averaging window lengthens, as longer windows capture cumulative and chronic pathways rather than only acute events. Estimates of short-term, all-source PM_{2.5} on mortality range from a 0.07% increase in mortality per 1 $\mu\text{g}/\text{m}^3$ over a two-day period across 652 cities (31) to 0.18% over a three-day period among the US elderly (32). Annual exposures yield 0.71% per $\mu\text{g}/\text{m}^3$ increases in mortality in the US Medicare cohort and 0.91% in a recent global meta-analysis (33). Our monthly estimates fall between these two clusters, and are well aligned with previous estimates of annual dust PM_{2.5} mortality impacts among the US Medicare population (0.53% and 1.30% per 1 $\mu\text{g}/\text{m}^3$ in national and regional samples, respectively (34, 35)).

Our estimate likely understates the mortality consequences of exposure to dust. We do not capture chronic effects or non-fatal outcomes, though dust is associated with cardiovascular and

respiratory morbidity, emergency and hospital visits, and other outcomes we do not quantify here (12, 36). We capture only fine dust, omitting the coarse fraction ($\text{PM}_{2.5-10}$) that makes up most dust mass and has its own established cardiovascular and respiratory effects (12). Cause-specific outcomes would clarify the mechanisms through which ambient dust $\text{PM}_{2.5}$ leads to premature mortality, and daily dust-specific $\text{PM}_{2.5}$, when available, would separate the contributions of sustained elevated concentrations from those of acute shocks.

Unlike most climate-sensitive mortality risks, which depend on mitigation benefits that are realized over decades or on community- and individual-level protections, part of dust’s health burden can be addressed rapidly at the source. Air-quality regulation largely treats dust as background pollution or as an “exceptional event” exempt from compliance (37), which removes incentives for control. Yet much of windblown dust is a downstream consequence of water and land management, and case studies indicate that it can be controlled. Shallow flooding cut emissions at Owens Lake by roughly 99% after upstream diversions desiccated the lakebed (38), which had become the country’s largest single source of particulate pollution following water diversion to Los Angeles. Comparable measures are feasible at the Great Salt Lake (26) and on fallowed farmland of California’s San Joaquin Valley (25, 36, 39). Health burdens are not borne evenly: Hispanic populations in particular are the most exposed at every income level, and the rural and peri-urban populations near dry lakebeds, desert margins, and retired farmland have gained least from the combustion-focused air-quality gains of recent decades (5, 25, 40). Our dust-specific concentration–response function and burden estimates can inform the benefit–cost analyses guiding air-quality, land, and water policy, which until now have had to borrow response functions estimated for total $\text{PM}_{2.5}$. And although national dust concentrations have held roughly steady, dust is a growing share of the $\text{PM}_{2.5}$ Americans breathe, its per-microgram toll is largest where combustion controls have succeeded most, and it has risen across the Southwest and Great Plains (12, 25) and is projected to rise further by late century (10, 41).

Materials and Methods

Data

Mortality. We obtained monthly all-cause death counts by county from the National Center for Health Statistics county-level mortality files for 2000–2019. We analyze total all-cause mortality, and additionally obtained age-stratified counts for adults aged ≥ 65 and 0–64 years. Population denominators are US Census Bureau intercensal estimates, from which we compute the county-month all-cause death rate. The analysis sample comprises 709,133 county-months across 3,038 counties, covering approximately 48 million deaths (Supplementary Table S1).

Satellite dust and total $\text{PM}_{2.5}$. Our primary exposure is dust $\text{PM}_{2.5}$ from the satellite-derived, source-specified dataset of van Donkelaar et al. (20), which combines GEOS-Chem chemical-transport modeling with MODIS, MISR, and SeaWiFS aerosol optical depth to partition total $\text{PM}_{2.5}$

into components (dust, sea salt, sulfate, nitrate, black carbon, and organic carbon) as biweekly gridded fields at $\sim 1 \times 1$ km. For each biweekly grid field we computed the population-weighted mean dust $\text{PM}_{2.5}$ within each county, weighting grid cells by 2020 WorldPop ~ 1 km population counts and using 2020 TIGER/Line county boundaries, and then averaged the biweekly values to calendar months.

We define non-dust $\text{PM}_{2.5}$ as total $\text{PM}_{2.5}$ minus dust from the same product. We use non-dust $\text{PM}_{2.5}$ as a control in our primary regression, and in heterogeneity analyses to define background pollution strata. Because dust and non-dust $\text{PM}_{2.5}$ are obtained by partitioning a single satellite total- $\text{PM}_{2.5}$ field according to the chemical-transport model’s simulated composition, rather than measured independently, the two components share the total- $\text{PM}_{2.5}$ measurement error and their separation depends on the modeled speciation. The non-dust term therefore serves to remove co-varying combustion-related pollution, but we refrain from interpreting its own fitted coefficient as an independent, like-for-like estimate of non-dust toxicity.

Meteorology. Monthly mean temperature and total precipitation are from gridMET, aggregated to the county-month with the same population weights as the dust exposure. We additionally obtained monthly population-weighted wind speed and vapor-pressure deficit (a measure of atmospheric aridity) from gridMET.

Exposure disparities

We measure exposure disparities using the Census Bureau’s public-use Gridded Environmental Impacts Frame (Gridded EIF) (42). The Gridded EIF calculated annual population counts from administrative records, including residential histories, demographics, and incomes from Internal Revenue Service and Social Security records. It produces race-ethnicity and household income deciles data on a 0.01° grid yearly from 1999 to 2024.

We follow recommended practice for assessing and reporting exposure-disparities from Marshall et al. (43), Koolik et al. (44). For each year 2000–2022 we join the EIF counts to that year’s grid-cell mean dust $\text{PM}_{2.5}$ and compute person-year population-weighted mean exposures, $\text{PWM}_k = \sum_{it} P_{ikt} C_{it} / \sum_{it} P_{ikt}$, over grid cells i and years t , as well as population-weighted percentiles of each group’s exposure distribution. We report absolute ($\text{PWM}_k - \text{PWM}_T$) and relative ($(\text{PWM}_k - \text{PWM}_T) / \text{PWM}_T$) disparities against the total population T . Metrics are assessed within each Census region against the region’s own mean, and we further estimate exposure within race $\times$ income decile cells.

We also recompute these metrics using the American Community Survey 2015–2019 county rates: race and ethnicity (B03002, Hispanic of any race; all others non-Hispanic). We also consider below poverty (B17001), below 200% of the poverty line (C17002), and educational attainment (B15003). The two data sources agree (Supplementary Table S4), with the county-level analysis giving slightly smaller racial-ethnic disparities, consistent with attenuation from unobserved within-county variation (45).

Statistical analysis

Primary model. We estimate the dust–mortality relationship with a Poisson two-way fixed-effects regression of the county-month all-cause death rate, weighted by county-month population:

$$\mathbb{E}[Y_{it}] = \exp\left(\theta D_{it} + X'_{it}\delta + \alpha_{i \times y(t)} + \gamma_{i \times m(t)} + \eta_{s(i) \times t}\right), \quad (1)$$

where Y_{it} is the death rate in county i and month t ; D_{it} is dust PM_{2.5} concentration in county i and month t , with coefficient θ ; X_{it} collects the meteorological covariates (temperature and precipitation polynomials) and co-occurring non-dust PM_{2.5} (total PM_{2.5} minus dust), with coefficient vector δ , so the dust effect is estimated net of contemporaneous combustion-related pollution; $\alpha_{i \times y(t)}$ are county $\times$ year fixed effects (absorbing time-invariant county characteristics and county-specific annual shocks); $\gamma_{i \times m(t)}$ are county $\times$ month-of-year fixed effects (absorbing county-specific seasonality); and $\eta_{s(i) \times t}$ are state $\times$ year-month fixed effects, which absorb regional shocks common to a state in a given calendar month (including the broad meteorological anomalies that drive dust), so that identification is on within-state, within-county-season deviations. Standard errors are clustered by county. Supplementary Fig. S1 maps our identifying variation.

Lag structure. Our primary specification uses the contemporaneous dust term (lag 0); for the attributable-burden calculation, and as a robustness check, we also accumulate the effect over lags 0–3, and separately replace the linear dust term with a natural cubic spline to allow a nonlinear concentration–response (Supplementary Figs. S2, S3).

Sensitivity and falsification. We assess robustness of our results to several alternative specifications and subject our preferred specification to falsification tests through permutation. First, confounding. Because the meteorology that generates dust (high wind, low humidity) may affect mortality through other channels, we add population-weighted wind speed and vapor-pressure deficit to the primary model (Supplementary Table S6). Because the satellite product may misattribute wildfire smoke to dust, we exclude western counties during fire season (June–October). We also drop the non-dust PM_{2.5} co-pollutant control, and we compute E-values: the minimum association, on the risk-ratio scale, that an unmeasured confounder would need with both dust and mortality to fully explain away the estimate. Second, we test alternative functional forms. We replace the linear response with natural splines with three to five degrees of freedom and alternative knot placements and to non-parametric five-, seven-, and nine-bin functions (Figure S3). Third, because our monthly exposure estimate averages over a finer resolution, we re-estimate with dust at its native biweekly resolution both as a within-month sum of a nonlinear biweekly basis and as a biweekly distributed lag (Supplementary Fig. S5). Fourth, our dust exposure product is modeled, and is likely to be best constrained in regions with ground-based speciation monitors; to assess whether exposure errors in areas without monitors drive our estimates, we restrict to the 273 counties with an IMPROVE or EPA speciation monitor (Supplementary Fig. S6). Fifth, we estimate four

alternative fixed effects specifications, including simpler and more complicated regional seasonality controls S4, cluster standard errors by state instead of county, and compute Conley spatial HAC standard errors at cutoffs from 100 to 500 km. Finally, we recompute the estimate under 500 within-county temporal permutations and 500 spatial permutations (dust reassigned across counties within state $\times$ year $\times$ month cells). Each breaks either the time series or cross-section link between dust and mortality, while preserving the other, and produces a design-based null distributions against which we compare our observed estimate (46). Results are reported in Supplementary Table S5.

Heterogeneity. We re-estimate the primary model separately for ages ≥ 65 and 0–64; within terciles of county-mean non-dust PM_{2.5} (boundaries 7.5 and 9.8 $\mu\text{g}/\text{m}^3$); within terciles of county-mean dust PM_{2.5} and their cross-classification with non-dust terciles (Supplementary Figs. S8–S9); and within the four Census regions.

Attributable burden.

We attribute deaths under a zero-dust counterfactual as $\text{AD} = \sum_i \text{deaths}_i [1 - \exp(-\hat{\beta}_{\text{cum}} D_i)]$ per year, where $\hat{\beta}$ is the contemporaneous (lag-0) linear coefficient for the primary estimate and the cumulative (lags 0–3) coefficient for the overall estimate that additionally captures delayed mortality. Confidence intervals are Monte Carlo draws from the county-clustered coefficient covariance (1,000 replicates). To test whether heterogeneity in concentration-response functions across observable groups produces different attributable burdens, we refit the primary lag-0 model separately for ages ≥ 65 and 0–64 (using age-specific deaths and rates), terciles of county non-dust PM_{2.5}, Census regions, and terciles of county income (defined as the person-weighted mean national household-income decile of county residents in the Gridded EIF) (coefficients shown in Supplementary Table S8); confidence intervals are quantiles of the sum of the independent strataum level Monte Carlo runs. We monetize with the EPA-default value of a statistical life (\$7.4 million in 2006 US\$, scaled to \$10.05 million in 2020 US\$).

Software. Analyses used R 4.4 with the fixest package (47) for fixed-effects Poisson models, and the splines, arrow, sf, and terra packages.

Article Information

Author contributions. CFG and AKH designed the study. CFG, WL, MVK, and AKH processed data. CFG performed the analysis. All authors interpreted the results, wrote the manuscript, and approved the final version.

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Conflict of interest. The authors declare no conflicts of interest.

Data and code availability. A public replication archive will be made available upon publication. Mortality data are from restricted-use NCHS county-month files (available to qualified researchers under NCHS data-use agreement).

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Supplementary Information

The mortality burden of airborne fine mineral dust in the United States

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This document contains Supplementary Methods and the Supplementary Figures and Tables referenced in the main text.

Supplementary Methods

Gridded EIF

Race-ethnicity uses the EIF’s harmonized, mutually exclusive categories: Hispanic of any race; non-Hispanic White, Black, Asian, and American Indian and Alaska Native; and a residual other-or-unknown group that includes multiracial individuals and those without race information. Counts are noise-infused for disclosure avoidance; we use the post-processed non-negative counts. Individuals without a household-income match are included in race-group totals but excluded from income-decile panels. Administrative coverage grows over the study period (from ~243 million matched persons in the contiguous US in 2000 to ~318 million in 2022), so trends for small groups partly reflect improving coverage.

Selection of the fixed-effects specification

Our identifying assumption is that, conditional on the fixed effects and weather controls, month-to-month deviations of dust within a county and calendar month are unrelated to other determinants of mortality. We tested this assumption, and used it to select the fixed-effects structure, with the permutation-falsification framework of Ma et al. (46).

Two placebo permutations probe the two directions of residual confounding. In the *spatial* permutation, the block of dust values is reassigned across counties within each year-month, breaking the link between a county’s dust and its mortality while preserving the national dust time series; a specification free of residual *temporal* confounding then yields a permuted estimate centered at zero. In the *temporal* permutation, dust is reassigned across months within a county, breaking the time-series link while preserving the cross-sectional pattern; a specification free of residual *spatial* confounding yields a permuted estimate centered at zero. For each specification we redrew the permutation (300–500 replicates) and re-estimated the linear dust slope.

A standard two-way fixed effects design that uses county $\times$ year and county $\times$ month-of-year fixed effects passes the temporal permutation (permuted mean 0.02% per $\mu\text{g}/\text{m}^3$, 95% interval $-0.23, 0.27$) but the spatial permutation the null centers at 0.15% per $\mu\text{g}/\text{m}^3$ (95% interval 0.03, 0.28), with 99.6% of permutations positive. A non-zero spatial null indicates residual temporal confounding. Our state $\times$ year-month fixed effects, which remove shocks shared by a state in a given month, yields a spatial-permutation null centered at zero (permuted mean -0.00% per $\mu\text{g}/\text{m}^3$). We adopt this specification.

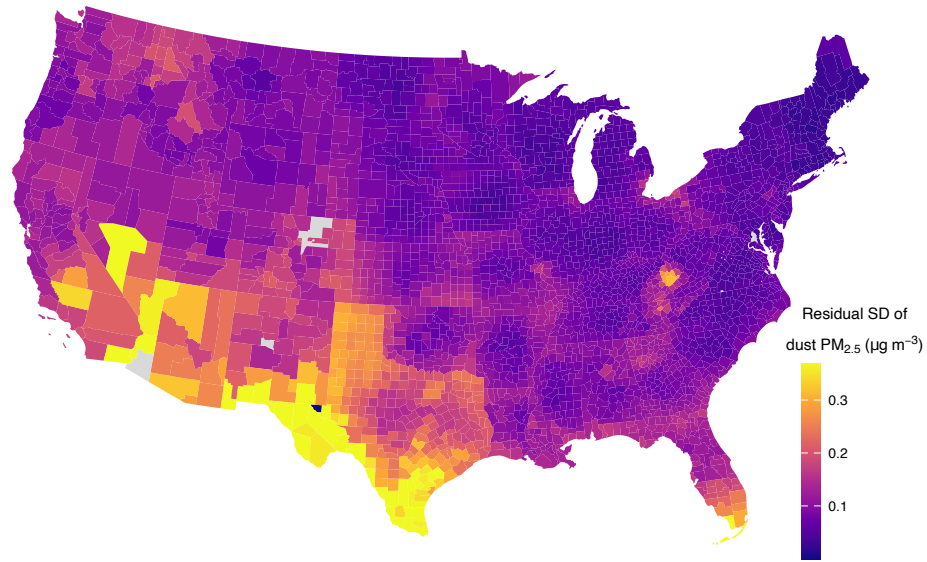


Figure S1: Geography of the identifying variation. The variation the fixed-effects design exploits: each county's within-county, within-season standard deviation of dust $\text{PM}_{2.5}$ after removing county $\times$ year, county $\times$ month-of-year, and state $\times$ year-month means (2000–2019). Residual variation concentrates in the arid Southwest and Southern Plains, mirroring mean dust itself; identification comes from month-to-month deviations around each county's own annual and seasonal levels net of state-wide monthly shocks, so counties with little residual variation (the humid East) contribute little to the estimate. The pooled within-county residual standard deviation after the full fixed-effects structure is $\sim 0.13 \mu\text{g}/\text{m}^3$. The colour scale is capped at the 98th percentile.

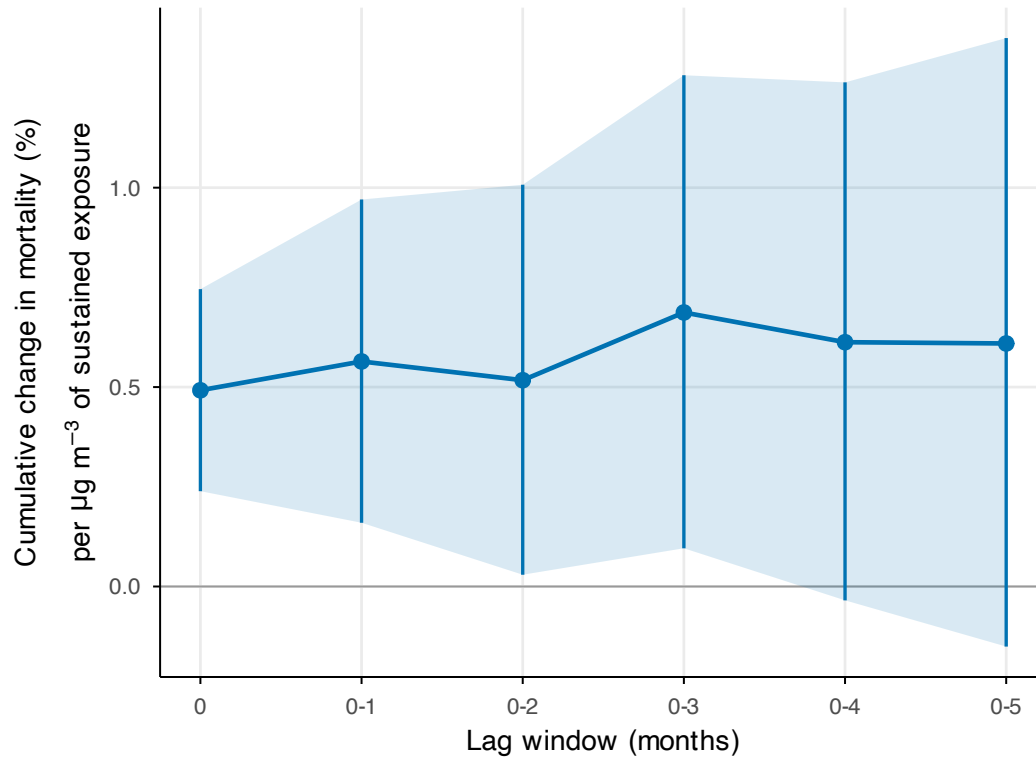


Figure S2: Cumulative dust–mortality effect as the lag window extends from 0 to 5 months. Cumulative effect (percent change per $\mu\text{g}/\text{m}^3$ of sustained exposure) as the distributed-lag window is extended from the contemporaneous month through six months, under the primary specification. Most of the effect is contemporaneous; the cumulative estimate accrues through lag 3 and is broadly stable but progressively less precise as the window extends further, motivating lags 0–3 as the window for the cumulative estimate. That the cumulative effect does not shrink back toward zero at longer windows is also inconsistent with short-term mortality displacement. Points and ribbons are 95% confidence intervals.

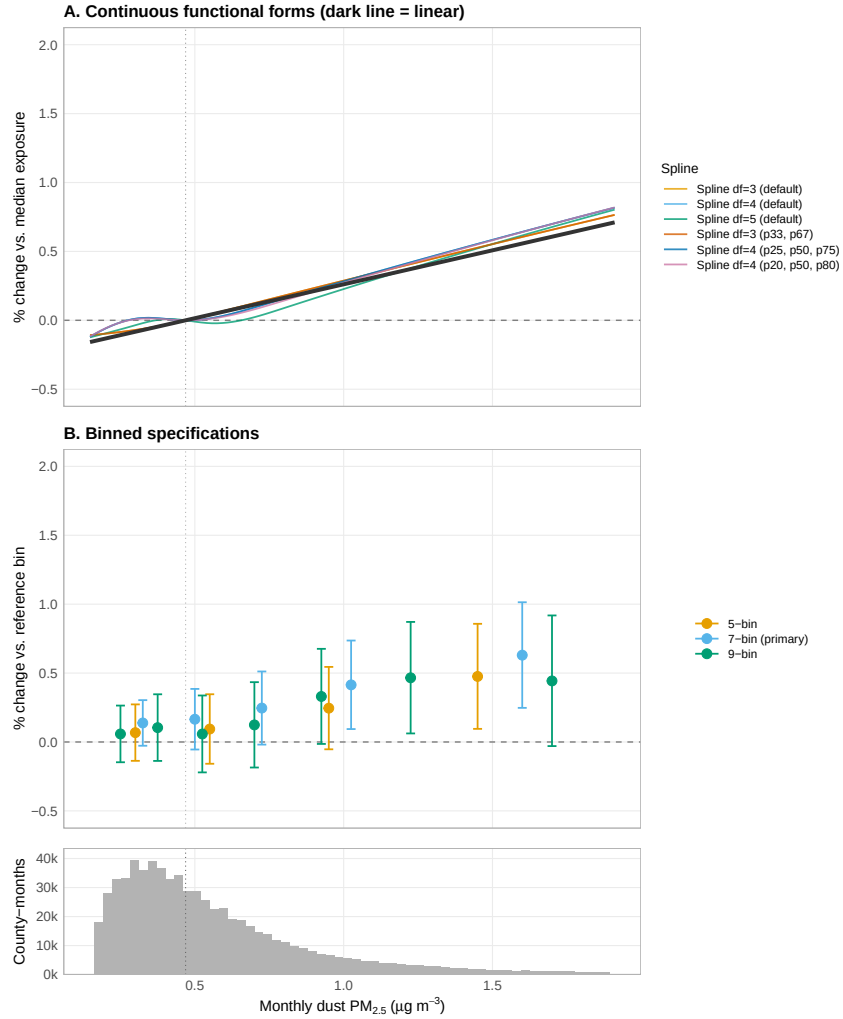


Figure S3: The contemporaneous (lag-0) concentration–response is robust to functional form. (A) Continuous forms: lag-0 percent change in mortality relative to median exposure under natural cubic splines varying the degrees of freedom (3–5) and knot placement, with the linear fit in black. (B) Non parametric binned specifications with 5, 7, and 9 bins (points at bin midpoints, 95% CIs), relative to the lowest-exposure reference bin. The bottom panel shows the county-month dust distribution .

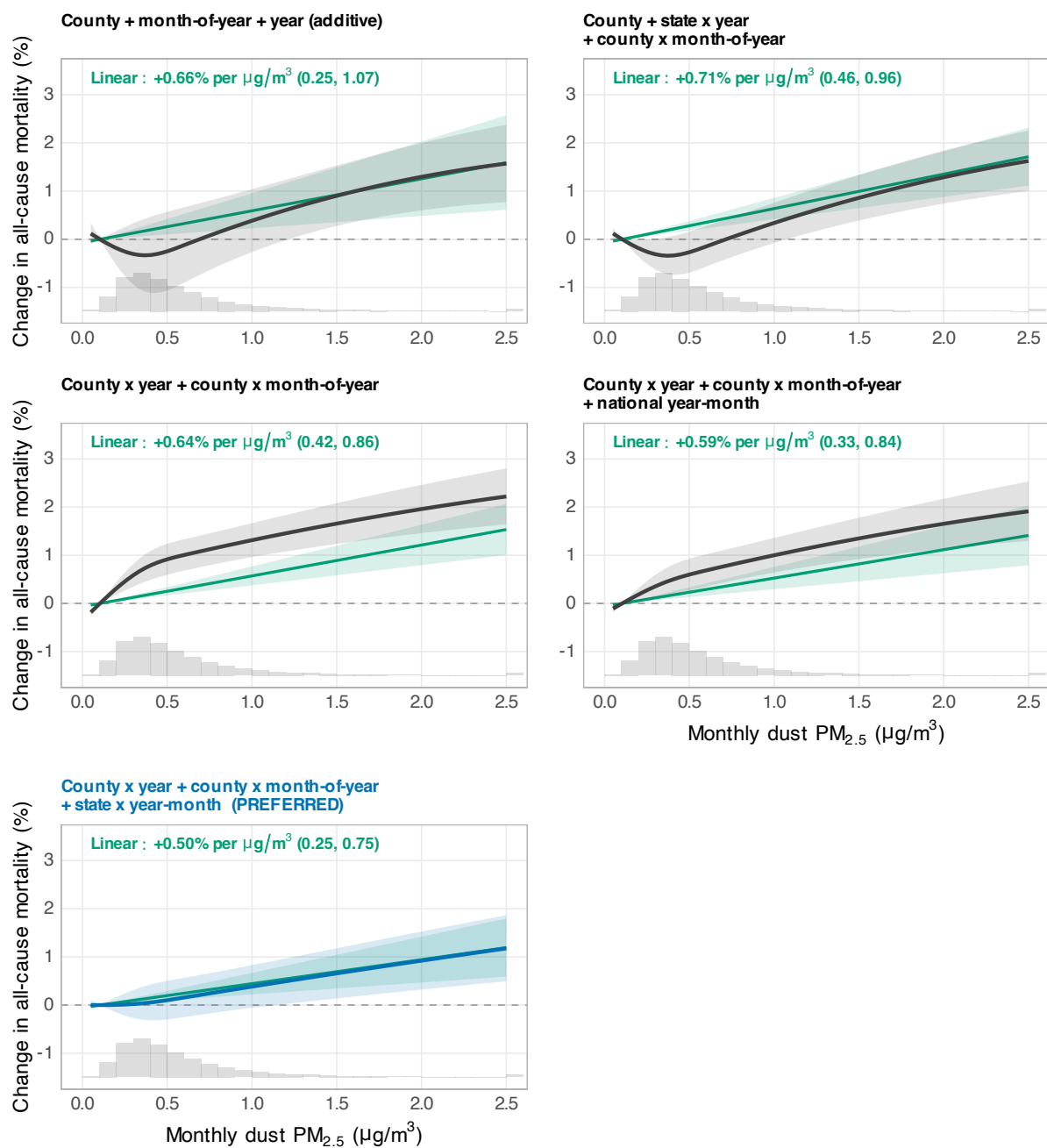


Figure S4: Contemporaneous (lag-0) concentration–response across fixed-effects structures. Lag-0 concentration–response (natural cubic spline, 3 df, delta-method 95% CI) under four fixed-effects structures, labeled by their terms.

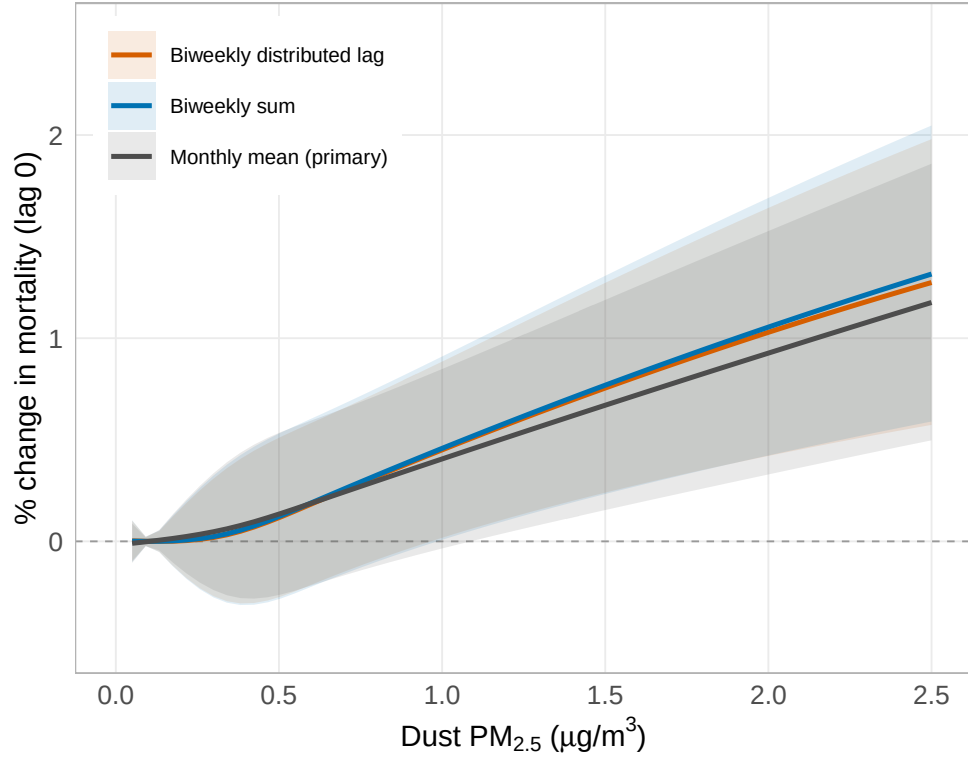


Figure S5: The contemporaneous (lag-0) concentration–response is not an artifact of averaging an episodic exposure to a monthly mean. Lag-0 concentration–response relative to $0.104 \mu\text{g}/\text{m}^3$ under three exposure constructions estimated on the same monthly outcome, fixed effects, weather, and non-dust $\text{PM}_{2.5}$ control, differing only in how the current month’s sub-monthly dust enters: the primary spline of the monthly-mean dust (grey); the within-month *sum* of a nonlinear biweekly basis (blue); and dust entered at its native biweekly resolution as a *distributed lag* over the current month’s ≈ 2 biweekly periods, with no within-month averaging (orange). Ribbons are 95% confidence intervals. All three reproduce the same broadly linear rise across the exposure range; the biweekly-resolved curves are, if anything, marginally higher, indicating the monthly-mean specification is unlikely to be artefactually flattened.

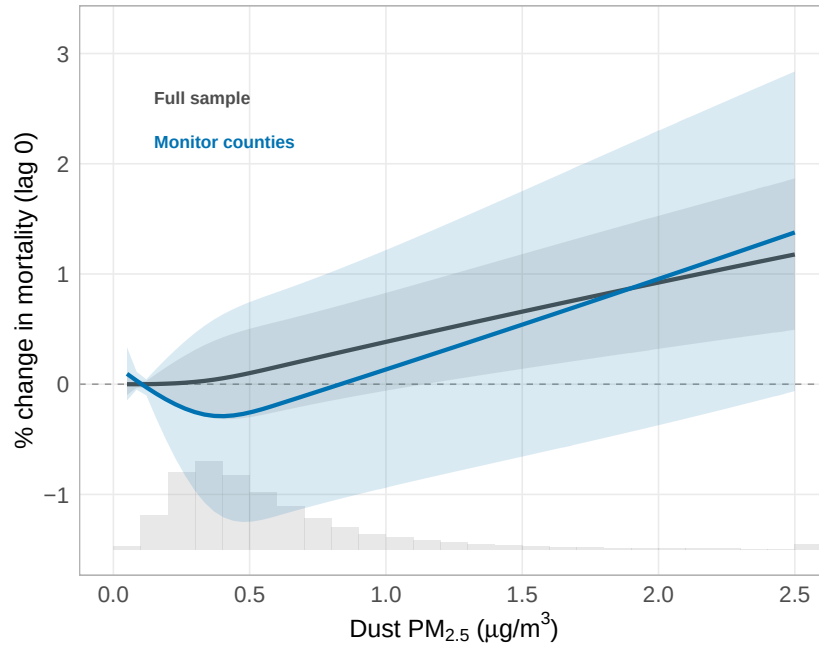


Figure S6: The contemporaneous (lag-0) concentration–response is not an artifact of satellite exposure measurement. Lag-0 concentration–response (relative to $0.104 \mu\text{g}/\text{m}^3$; natural cubic spline, delta-method 95% CI) on the full sample (grey) and re-estimated in the 273 counties hosting an IMPROVE or EPA Chemical Speciation Network monitor (blue), where the satellite-derived dust product can be cross-checked against ground measurements.

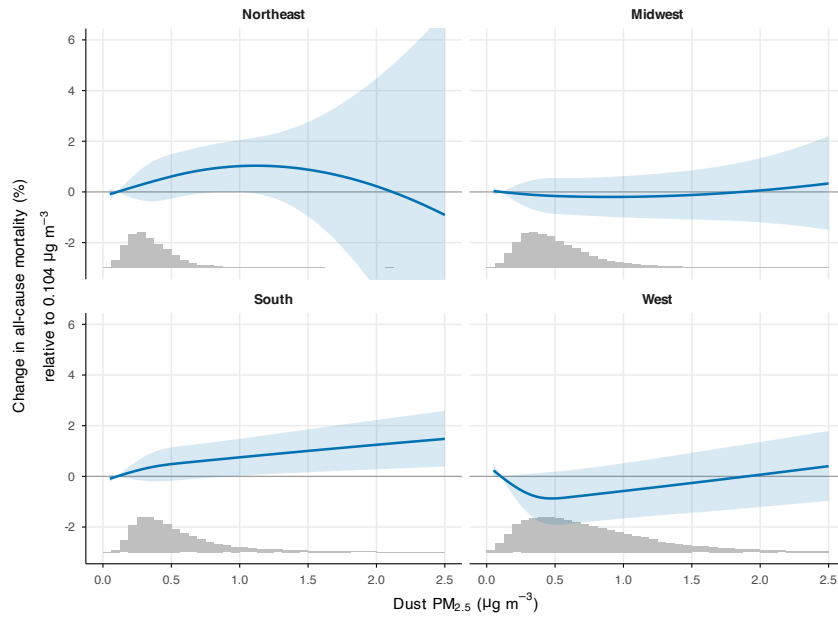


Figure S7: The dust–mortality concentration–response across Census regions. Lag-0 percent change in all-cause mortality (natural cubic spline, 3 df, delta-method 95% CI; relative to $0.104 \mu\text{g}/\text{m}^3$) estimated separately for each of the four Census regions. Histograms show each region’s monthly dust $\text{PM}_{2.5}$ distribution.

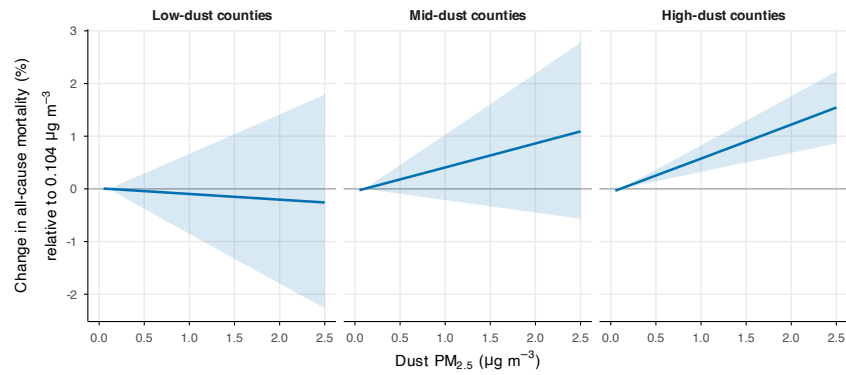


Figure S8: The dust–mortality concentration–response across levels of dust $\text{PM}_{2.5}$. Lag-0 linear dust–mortality slope (% per $\mu\text{g}/\text{m}^3$, 95% CI) estimated separately within terciles of county-mean dust $\text{PM}_{2.5}$ (thresholds at the 33rd and 67th percentiles of the county-mean distribution). Histograms show each tercile’s monthly dust distribution.

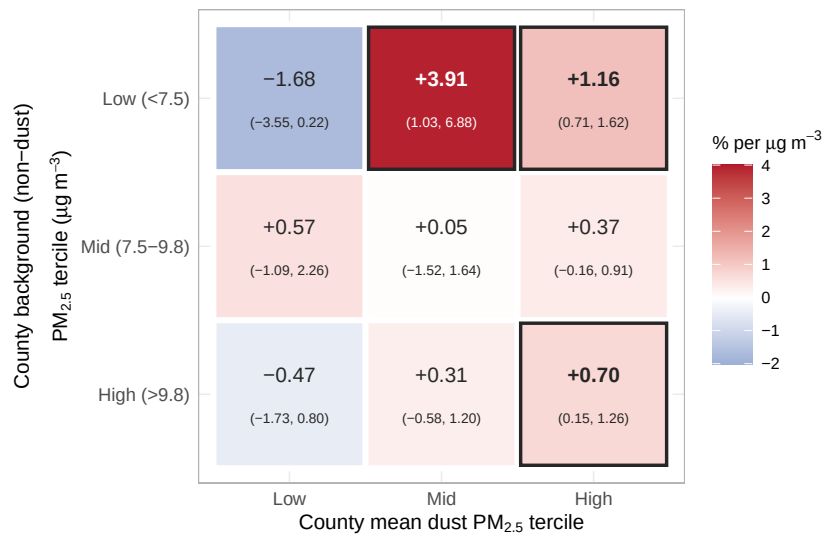


Figure S9: The dust–mortality concentration–response across levels of dust $\text{PM}_{2.5}$ and non-dust $\text{PM}_{2.5}$. Lag-0 linear dust–mortality slope (% per $\mu\text{g}/\text{m}^3$, 95% CI) estimated separately in each cell of a 3×3 cross-classification of county-mean dust $\text{PM}_{2.5}$ tercile (columns) and county-mean non-dust (background) $\text{PM}_{2.5}$ tercile (rows; thresholds in $\mu\text{g}/\text{m}^3$). Bold outlines mark cells where the 95% CI excludes zero.

Table S1: Characteristics of the analytic sample. Unit of observation is the county-month; the sample comprises 709,133 county-months across 3,038 counties and 48.0 million all-cause deaths, 2000–2019.

Characteristic	Mean (SD)	Median (IQR)	Pop.-weighted mean ^a
<i>Exposure</i> ($\mu\text{g}/\text{m}^3$)			
Dust PM _{2.5}	0.59 (0.48)	0.46 (0.31–0.71)	0.68
Non-dust PM _{2.5}	8.30 (3.73)	7.89 (5.72–10.28)	9.39
Total PM _{2.5}	8.89 (3.77)	8.45 (6.28–10.88)	10.06
<i>Mortality</i>			
All-cause deaths per county-month	68 (184)	23 (10–54)	648
All-cause rate, per 100,000 per month	71.2 (25.2)	68.6 (56.5–82.9)	64.8
<i>Meteorology</i>			
Temperature, °C	13.4 (9.9)	14.4 (6.1–21.7)	14.7
Wind speed, m/s	4.1 (0.8)	4.1 (3.5–4.6)	3.9
Vapor-pressure deficit, kPa	0.80 (0.51)	0.74 (0.43–1.03)	0.87
<i>County size</i>			
Resident population	96,708 (313,107)	26,062 (11,631–66,092)	1,110,437

^aCounty-months weighted by resident population (the estimation weight), approximating the value experienced by the average person. On this basis the dust PM_{2.5} percentiles are 0.35 (25th), 0.51 (50th), and 0.80 (75th); the interquartile contrast reported in the main text is anchored on these. Dust, non-dust, and total PM_{2.5} are from the van Donkelaar et al. product; meteorology from gridMET.

Table S2: Dust PM_{2.5} exposure by race-ethnicity and household income, 2000–2022 (Gridded EIF). Person-year population-weighted mean (PWM) exposure, absolute and relative disparity against the mean across all people, and the 10th, 50th, and 90th percentiles of each group’s population-weighted exposure distribution, computed at the 0.01° grid level from the Census Bureau’s Gridded Environmental Impacts Frame (Methods). Population is the mean matched population per year. Hispanic includes people of any race; other racial-ethnic groups are non-Hispanic; the other-or-unknown group includes multiracial individuals and those without race information. Income deciles are national household-income deciles; individuals without an income match are included in the race-ethnicity rows but not the income rows.

Group	Population (millions/yr)	PWM ($\mu\text{g}/\text{m}^3$)	Absolute ($\mu\text{g}/\text{m}^3$)	Relative (%)	10th pctile	50th pctile	90th pctile
All people	282.2	0.685	—	—	0.302	0.578	1.183
<i>Race/ethnicity</i>							
Hispanic	40.7	0.955	+0.270	+39.4	0.388	0.877	1.603
American Indian and Alaska Native	2.3	0.806	+0.121	+17.6	0.328	0.738	1.327
Other or unknown	17.4	0.708	+0.023	+3.3	0.312	0.593	1.222
Asian	11.2	0.703	+0.017	+2.5	0.333	0.603	1.198
Black	34.0	0.677	−0.008	−1.2	0.352	0.603	1.103
White	176.7	0.620	−0.066	−9.6	0.282	0.532	1.052
<i>Household income decile</i>							
Decile 1 (lowest)	28.2	0.697	+0.012	+1.7			
Decile 2	28.4	0.718	+0.033	+4.8			
Decile 3	28.3	0.723	+0.038	+5.6			
Decile 4	28.2	0.711	+0.026	+3.7			
Decile 5	28.2	0.697	+0.012	+1.7			
Decile 6	28.1	0.684	−0.001	−0.2			
Decile 7	28.1	0.673	−0.012	−1.8			
Decile 8	28.1	0.663	−0.022	−3.3			
Decile 9	28.0	0.651	−0.034	−5.0			
Decile 10 (highest)	28.0	0.634	−0.051	−7.4			

Table S3: Relative dust PM_{2.5} exposure disparity within Census region, 2000–2022 (Gridded EIF). Each racial-ethnic group is compared against the person-year population-weighted mean of its own region rather than the national mean, so the table isolates unequal exposure within a region from regional differences in dust. Values are percent differences; the bottom row gives each region’s own mean exposure.

Group	West	South	Midwest	Northeast
Hispanic	+14.4	+46.1	+5.1	+11.2
American Indian and Alaska Native	+8.0	+6.3	−3.7	−3.5
Other or unknown	−5.8	+2.4	−1.5	−0.5
Asian	−13.3	+1.8	+0.9	+9.2
Black	+5.6	−7.3	+22.3	+16.9
White	−4.9	−9.2	−3.2	−4.9
Region mean ($\mu\text{g}/\text{m}^3$)	0.962	0.742	0.532	0.404

Table S4: County-level ACS cross-check of exposure disparities, 2000–2022. Population-weighted mean (PWM) dust $\text{PM}_{2.5}$ computed at the county level with American Community Survey 2015–2019 populations, the design used before grid-level administrative counts were available. For the racial-ethnic groups common to both, the county-ACS estimates closely track the grid-level EIF estimates of Table S2 (e.g., Hispanic +32.9% vs +39.4%; White –9.2% vs –9.6%), with the county analysis slightly attenuated, consistent with unobserved within-county variation. The ACS additionally characterizes dimensions unavailable in the EIF: exposure varies by less than about 5% of the national mean across poverty status and educational attainment. Hispanic includes people of any race; all other groups are non-Hispanic.

Group	Population (M)	PWM ($\mu\text{g}/\text{m}^3$)	Absolute ($\mu\text{g}/\text{m}^3$)	Relative (%)
<i>Race/ethnicity</i>				
Hispanic	58.3	0.915	+0.225	+32.9
American Indian and Alaska Native	2.1	0.849	+0.159	+23.2
Native Hawaiian and Pacific Islander	0.4	0.796	+0.106	+15.6
Asian	17.1	0.703	+0.013	+1.8
Two or more races	7.6	0.678	–0.012	–1.8
Black	39.9	0.661	–0.029	–4.3
Some other race	0.8	0.654	–0.036	–5.4
White	196.3	0.627	–0.063	–9.2
<i>Poverty</i>				
Below the poverty line	42.3	0.723	+0.032	+4.7
At or above the poverty line	272.3	0.686	–0.004	–0.5
<i>Income</i>				
Below 200 percent of the poverty line	97.3	0.722	+0.032	+4.7
At or above 200 percent of the poverty line	217.4	0.677	–0.013	–1.9
<i>Education</i>				
Less than a bachelor's degree	148.7	0.691	+0.001	+0.2
Bachelor's degree or higher	70.5	0.669	–0.021	–3.0

Table S5: Robustness of the dust–mortality estimate to alternative inference, co-pollutant control, and sample restriction. Linear slope (% per $\mu\text{g}/\text{m}^3$, 95% CI) for the contemporaneous (lag-0) primary estimand and the cumulative (lags 0–3) distributed-lag estimand.

Specification / inference	Lag 0	Cumulative 0–3
Primary (county-clustered)	0.50 (0.25, 0.75)	0.70 (0.13, 1.27)
State-clustered	0.50 (0.34, 0.65)	0.70 (−0.15, 1.55)
Conley spatial HAC, 100 km	0.51 (0.27, 0.74)	0.70 (0.01, 1.39)
Conley spatial HAC, 200 km	0.51 (0.31, 0.71)	0.70 (0.03, 1.37)
Conley spatial HAC, 500 km	0.51 (0.28, 0.74)	0.70 (−0.16, 1.56)
Drop non-dust $\text{PM}_{2.5}$ control	0.50 (0.26, 0.75)	0.76 (0.21, 1.31)
Exclude West $\times$ fire season (Jun–Oct)	0.52 (0.25, 0.80)	0.74 (0.14, 1.35)

The primary specification includes the contemporaneous non-dust $\text{PM}_{2.5}$ control. E-values (minimum confounder association, risk-ratio scale, required to fully explain away the estimate): lag-0, 1.08 per $\mu\text{g}/\text{m}^3$ and 1.05 for the interquartile contrast (1.04 at the CI bound); cumulative, 1.09 per $\mu\text{g}/\text{m}^3$ and 1.06 for the interquartile contrast (1.02 at the CI bound). Design-based permutation p -values for the lag-0 estimate are < 0.002 .

Table S6: Controlling for the meteorological drivers of dust. Effect of adding contemporaneous county-month wind speed and vapor-pressure deficit (gridMET, population-weighted) to the primary (lag-0) model. The slope does not materially attenuate, indicating the estimate is not an artifact of the wind and aridity that generate dust.

Estimand	Primary (lag-0) model	+ wind + vapor-pressure deficit
Linear slope (% per $\mu\text{g}/\text{m}^3$)	0.50 (0.25, 0.75)	0.45 (0.20, 0.70)
Interquartile contrast (%)	0.22 (0.11, 0.34)	0.20 (0.09, 0.31)

Table S7: Attributable burden under alternative concentration–response functional forms (lag-0).

Concentration–response form	AD/yr	95% CI
Linear (headline)	7,759	(3,815, 11,501)
Natural cubic spline ^a	4,780	(−8,160, 17,349)
Seven-bin step (vs $<0.25 \mu\text{g}/\text{m}^3$ bin)	5,586	(912, 10,075)
Linear, county cluster-bootstrap CI	7,759	(3,495, 12,263)
Spline, county cluster-bootstrap CI	4,780	(−6,015, 16,997)

^a Uncertainty accumulates over the full exposure range, producing wide confidence intervals for the spline burden estimations.

^b Counterfactual is less the less than $0.25 \mu\text{g}/\text{m}^3$ bin (other counterfactuals shown are $0 \mu\text{g}/\text{m}^3$).

Table S8: Stratum-specific concentration–response coefficients.

Stratification	Stratum	% per $\mu\text{g}/\text{m}^3$ (95% CI)	AD/yr	Total (95% CI)
None (pooled)	—	0.50 (0.25, 0.75)	7,759	7,759 (3,767, 11,482)
Age group	Ages ≥ 65	0.50 (0.24, 0.76)	5,653	7,681 (4,122, 11,252)
	Ages 0–64	0.47 (0.00, 0.94)	2,028	
Background PM _{2.5} tercile	Low ($< 7.5 \mu\text{g}/\text{m}^3$)	1.00 (0.54, 1.45)	3,117	7,496 (3,592, 11,397)
	Mid (7.5–9.8)	0.28 (−0.17, 0.73)	1,565	
	High (> 9.8)	0.41 (0.01, 0.81)	2,813	
Census region	Northeast	1.08 (0.01, 2.17)	1,785	7,417 (3,341, 11,617)
	Midwest	−0.00 (−0.63, 0.62)	−12	
	South	0.47 (0.11, 0.83)	3,042	
	West	0.60 (0.16, 1.05)	2,603	
County income tertile	Low	0.41 (−0.48, 1.30)	720	7,906 (3,588, 12,313)
	Medium	0.55 (0.18, 0.92)	2,288	
	High	0.50 (0.11, 0.90)	4,898	

Table S9: Effect modification by background pollution and dust exposure, jointly. Contemporaneous (lag-0) linear dust–mortality slope (% per $\mu\text{g}/\text{m}^3$, 95% CI) estimated within each cell of a county background (non-dust) PM_{2.5} tercile $\times$ dust-exposure tercile grid.

Background PM _{2.5} tercile	Dust-exposure tercile		
	Low	Mid	High
Low ($< 7.5 \mu\text{g}/\text{m}^3$)	−1.68 (−3.55, 0.22)	3.91 (1.03, 6.88)	1.16 (0.71, 1.62)
Mid (7.5–9.8)	0.57 (−1.09, 2.26)	0.05 (−1.52, 1.64)	0.37 (−0.16, 0.91)
High ($> 9.8 \mu\text{g}/\text{m}^3$)	−0.47 (−1.73, 0.80)	0.31 (−0.58, 1.20)	0.70 (0.15, 1.26)

Table S10: Effect modification by Census region and background pollution, jointly. Contemporaneous (lag-0) linear dust–mortality slope (% per $\mu\text{g}/\text{m}^3$, 95% CI) within cells of Census region $\times$ county background (non-dust) PM_{2.5} tercile. Cells with fewer than 5,000 county-months are omitted.

Region	Background PM _{2.5} tercile	% per $\mu\text{g}/\text{m}^3$ (95% CI)
South	Low	1.51 (0.91, 2.11)
South	Mid	0.26 (−0.26, 0.77)
South	High	0.21 (−0.55, 0.98)
West	Low	0.55 (−0.18, 1.28)
West	Mid	−0.14 (−1.78, 1.53)
Northeast	Low	0.48 (−4.55, 5.77)
Northeast	Mid	0.51 (−1.87, 2.94)
Northeast	High	1.18 (0.06, 2.31)
Midwest	Low	1.04 (−1.44, 3.58)
Midwest	Mid	0.29 (−0.98, 1.58)
Midwest	High	−0.52 (−1.38, 0.35)