

Uncovering Respiratory Effects of Short-term PM2.5 Exposure through Global Nocturnal Cough Monitoring

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Abstract

Air pollution remains one of the leading environmental determinants of health, yet the short-term effects of fine particulate matter (PM_{2.5}) on early respiratory symptoms are poorly characterized owing to the limited availability of high-resolution, population-scale data. Enabled by global mobile sensing through a widely used application (Sleep Cycle) and AI algorithms that automatically detect coughs from nocturnal audio recordings, we derived nighttime cough frequency across 34 cities in 12 countries on five continents over 500 consecutive days, conducting the first multi-city assessment linking daily PM_{2.5} exposure to nocturnal respiratory symptoms. Single-city generalized additive models, multi-city panel regressions adjusting for meteorological variables and influenza rates, and quasi-experimental analyses consistently showed that same-day PM_{2.5} exposure was associated with higher cough frequency (relative risk = 1.004, 95% CI 1.001–1.008 per 10 $\mu\text{g}/\text{m}^3$), with amplified effects during influenza peaks. The exposure–response curve was monotonic but nonlinear, with measurable risks evident even below the World Health Organization 24-hour guideline (15 $\mu\text{g}/\text{m}^3$) and sharply increasing risks above 50 $\mu\text{g}/\text{m}^3$. These findings provide real-world, population-level, multi-city evidence of acute respiratory effects from short-term PM_{2.5} exposure, reveal the absence of a safe threshold, and underscore the urgency of stringent air-quality interventions to protect respiratory health. This study also demonstrates how large-scale, AI-based mobile sensing can transform real-time environmental epidemiology by enabling population-level monitoring of early respiratory symptoms worldwide.

Main

Air pollution is a leading global health threat, contributing to an estimated seven million premature deaths each year through cardiovascular and respiratory pathways [1]. Among airborne pollutants, fine particulate matter (PM_{2.5}; aerodynamic diameter $\leq 2.5 \mu\text{m}$) is of particular concern because particles penetrate deep into the lungs and enter the bloodstream [2].

While long-term exposure to PM_{2.5} is a well-established driver of cardiopulmonary morbidity and mortality [3, 4], the short-term effects of PM_{2.5} are less consistently

characterized. Several time-series and case-crossover studies have reported acute increases in respiratory hospital admissions and reductions in lung function following short-term $\text{PM}_{2.5}$ elevations [5–7]. However, these investigations predominantly capture severe clinical events and rely on hospital-based records, thereby overlooking early and transient respiratory responses that occur at a population level. In addition, most prior work has been limited to single-city or regional studies (i.e., often in North America or East Asia), typically using weekly or multi-day averages rather than daily measurements, and rarely considering interactions with concurrent respiratory infections.

Consequently, a major evidence gap remains: *how does short-term $\text{PM}_{2.5}$ exposure influence early, non-clinical respiratory symptoms in real-world settings and across diverse global environments?* Addressing this gap requires high-resolution, population-scale data capable of capturing subtle respiratory fluctuations with both spatial and temporal granularity—data that conventional surveys or administrative health records cannot provide.

Recent advances in mobile health (mHealth) technologies now make such measurements possible. Widespread smartphone and wearable adoption enables continuous, real-world health monitoring across populations [8–10]. Among emerging modalities, nocturnal audio monitoring provides a noninvasive, scalable method to detect coughing—an objective, quantifiable marker of airway irritation and early respiratory dysfunction [11, 12]. Mature AI algorithms can automatically identify cough events from nightly audio recordings with high accuracy, enabling large-scale, near-real-time assessment of respiratory activity [13–16]. Because nocturnal cough reflects both environmental triggers and infection-related inflammation, it offers a unique window into early respiratory responses to air pollution and their potential amplification by concurrent infections.

Leveraging this opportunity, we analyzed 500 consecutive days of nighttime audio data from a widely used mHealth application (Sleep Cycle), spanning 34 cities in 12 countries and $\text{PM}_{2.5}$ levels ranging from < 5 to $> 100\mu\text{g}/\text{m}^3$. A machine learning algorithm identified cough events from raw audio and aggregated them to the city-day level. Linking these data to daily city-level $\text{PM}_{2.5}$ concentrations enabled us to quantify short-term respiratory effects of air pollution with unprecedented temporal and spatial resolution. To our knowledge, this represents the first global, multi-city mHealth analysis of acute respiratory responses to $\text{PM}_{2.5}$ exposure.

Our analytical framework integrates three complementary perspectives (see Figure 1). At the city level, we applied generalized additive models (GAMs) to estimate same-day associations between $\text{PM}_{2.5}$ and nocturnal cough frequency, flexibly adjusting for meteorological and seasonal factors. At the global level, we synthesized city-specific estimates through meta-analysis and multi-city panel regressions incorporating city and day fixed effects to account for heterogeneity and to test effect modification by influenza incidence. Finally, we leveraged wildfire-smoke episodes as quasi-natural experiments, using a historical comparison framework conceptually similar to a difference-in-differences approach to evaluate the short-term causal impact of $\text{PM}_{2.5}$ surges on cough frequency at city level.

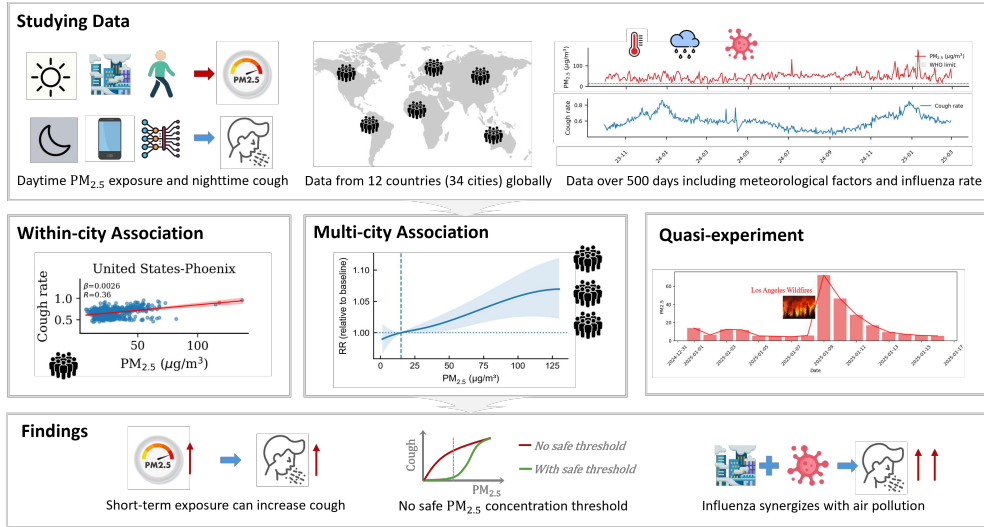


Figure 1 Overview of the study design and multi-city analyses linking $PM_{2.5}$ exposure to nocturnal cough frequency. Daily nocturnal cough frequency was passively recorded via a smartphone-based mHealth platform and linked with city-level $PM_{2.5}$, meteorological, and influenza data across 34 cities worldwide. City-specific GAMs, pooled meta/panel models, and a wildfire-based quasi-experiment collectively quantified short-term respiratory effects of $PM_{2.5}$. Higher $PM_{2.5}$ levels were consistently associated with increased nocturnal cough, with stronger effects during concurrent influenza activity and no evident safe threshold.

Across these analyses, short-term exposure to ambient $PM_{2.5}$ was consistently associated with increased nocturnal cough frequency. City-specific estimates were positive in 24 of 34 cities, and pooled panel regressions yielded a relative risk (RR) of 1.004 (95% CI 1.001–1.008) per 10 $\mu g/m^3$ increase in same-day $PM_{2.5}$. The exposure–response curve was monotonic and nonlinear, showing measurable risks even at concentrations below the WHO 24-hour AQG threshold (15 $\mu g/m^3$), consistent with global burden of disease findings of no safe exposure level [17, 18]. Effects were amplified during periods of heightened influenza activity, underscoring biologically plausible pollution–infection synergy. Wildfire-based quasi-experiments further supported the causal interpretation.

Together, these findings demonstrate that even short-term and low-level $PM_{2.5}$ exposure can measurably disrupt nocturnal respiratory health. By revealing consistent associations across diverse global settings, this study provides globally generalizable evidence that strengthens the scientific basis for informing air-quality guidelines and public health policy. Beyond establishing population-level epidemiologic links, our approach also underscores the transformative potential of mHealth technologies for environmental and respiratory monitoring. Continuous, passive assessment of nocturnal cough offers a scalable pathway toward integrating mHealth data into real-time surveillance systems, enabling earlier detection of pollution–infection interactions and more adaptive public health responses.

Country	City	$\hat{PM}_{2.5}$ ($\mu\text{g}/\text{m}^3$)	$\hat{\text{Hours}}$ (h)	$\hat{\text{Users}}$	cough frequency	$\hat{\text{Temp}}$ ($^{\circ}\text{F}$)	$\hat{\text{Prec}}$ (mm)
Australia	Brisbane	25.24	21801.53	3207.90	0.58	72.22	0.17
Australia	Melbourne	32.55	32035.59	4733.06	0.57	59.51	0.08
Australia	Sydney	21.73	33913.19	5077.72	0.58	65.94	0.10
Brazil	Sao Paulo	51.16	16205.57	2610.39	0.69	70.06	0.14
Chile	Santiago	65.30	3304.35	529.85	0.73	60.23	0.02
China	Beijing	89.29	8676.64	1361.63	0.56	51.28	0.06
China	Chengdu	106.10	2479.89	396.87	0.56	60.42	0.09
China	Hangzhou	91.64	2816.38	448.37	0.53	63.20	0.17
China	Shanghai	89.94	11831.51	1823.13	0.56	60.75	0.11
France	Paris	52.92	23640.09	3594.38	0.64	53.57	0.09
Germany	Berlin	52.15	18642.77	2796.61	0.59	49.54	0.07
Germany	Leipzig	40.06	2110.56	316.64	0.63	49.76	0.07
Italy	Bergamo	13.92	808.74	122.65	0.69	56.04	0.15
Italy	Florence	4.74	1665.54	255.41	0.85	41.26	0.07
Italy	Milano	18.98	11405.31	1778.10	0.69	56.04	0.15
Italy	Monza and Brianza	12.69	960.34	145.09	0.87	56.04	0.15
Italy	Rome	6.39	6396.10	1003.14	0.77	61.16	0.07
Japan	Osaka	50.89	35809.11	6022.17	0.77	61.65	0.15
Japan	Tokyo	34.83	119307.82	19961.92	0.71	60.75	0.17
South Africa	City of Cape Town	48.47	2619.96	388.20	0.68	64.32	0.26
South Africa	City of Johannesburg	11.03	2312.30	351.26	0.73	66.29	0.25
Sweden	Uppsala	19.56	3782.19	542.99	0.75	41.05	0.05
United Kingdom	Birmingham	33.83	4966.65	754.76	0.72	50.41	0.10
United Kingdom	Cambridge	4.98	4289.14	615.95	0.68	56.04	0.11
United Kingdom	London	46.60	91261.88	13624.78	0.61	51.72	0.09
United Kingdom	Oxford	9.97	5090.97	726.90	0.71	49.98	0.09
United States	Chicago	32.80	26136.04	3944.73	0.58	47.25	0.08
United States	Columbus	34.55	15362.96	2353.17	0.67	63.16	0.39
United States	Los Angeles	47.55	52715.50	8002.03	0.60	59.78	0.03
United States	New York	36.54	33945.85	5248.07	0.46	55.34	0.12
United States	Phoenix	29.01	20510.87	3072.00	0.67	72.90	0.01
United States	Pittsburgh	33.99	5883.64	893.38	0.65	50.48	0.12
United States	Saint Louis	34.05	7093.24	1056.83	0.62	57.49	0.13
United States	Salt Lake	28.80	9476.04	1416.31	0.63	53.56	0.04

Table 1 Descriptive statistics of the study population by city. $\hat{PM}_{2.5}$ denotes the average daily concentration of $PM_{2.5}$ over the study period. Hours represents the average daily recorded hours per city, and Users is the average number of users who recorded data. cough frequency indicates the average daily cough frequency. Temp ($^{\circ}\text{F}$) and Prec (mm) represent the average temperature and precipitation, respectively, for each city.

Results

Descriptive statistics of the data

The cough monitoring data analyzed in this study were obtained from a widely used sleep health application, Sleep Cycle¹. Users of this platform primarily monitor their sleep and snoring patterns to assess sleep quality. With millions of active users worldwide, it provides a large-scale dataset that forms the foundation of our aggregated analyses (More details about how the data were collected and processed can be found in the [Method](#) section).

We analyzed data collected over an 18-month period (October 2023 to April 2025) across 34 cities in 12 countries spanning Asia, Europe, North America, South America, Oceania, and Africa, representing regions with the highest user activity. Cities were included if their average user rate (daily active users per population) exceeded 0.5 per 10,000 residents, corresponding approximately to at least one active user per 10,000 inhabitants.

Demographic statistics of the included populations are summarized in [Supplementary Table 1](#). The sample was broadly representative, covering both genders and a wide age range across diverse socio-demographic contexts.

¹<https://sleepcycle.com/>

Table 1 presents city-level descriptive statistics. The study sites span a broad environmental gradient—from moderately polluted cities such as Florence (mean $\text{PM}_{2.5} < \mu\text{g}/\text{m}^3$) to highly polluted megacities such as Hangzhou (mean $\text{PM}_{2.5} \approx 100\mu\text{g}/\text{m}^3$)—and include locations in both hemispheres (e.g., Melbourne, Australia). This geographic and climatic heterogeneity enhances the generalizability of the subsequent analyses.

In addition to air-pollution data, we incorporated local meteorological variables—including daily mean temperature and precipitation—as covariates. Because influenza infection is a major biomedical factor contributing to cough [19], we further obtained influenza incidence data from national surveillance systems and converted it to city-level influenza rates used as model covariates.

A representative time-series visualization for Los Angeles (United States) is shown in Supplementary Figure 1. Visual inspection suggests that cough frequency broadly follows seasonal influenza patterns, consistent with previous studies [20, 21]. However, transient spikes in cough frequency often coincide with short-term $\text{PM}_{2.5}$ peaks, indicating that acute air-pollution episodes may contribute to day-to-day respiratory variability beyond the underlying seasonal trend.

Within-city associations between $\text{PM}_{2.5}$ and nocturnal cough

Table 1 highlights substantial intercity variability in both air quality (mean $\text{PM}_{2.5}$ ranging from < 5 to $> 100 \mu\text{g}/\text{m}^3$) and nocturnal cough frequency (0.53–0.85). These contrasts likely reflect differences in socioeconomic conditions, energy use, baseline respiratory health, and other contextual factors. To account for this heterogeneity, we first conducted city-specific analyses.

For each city, we fitted a generalized additive model (GAM) including a linear term for $\text{PM}_{2.5}$ and spline terms for meteorological, temporal, and influenza covariates to estimate short-term, within-city effects (see Method section). The results are summarized in Figure 2(a), where cities are ordered by their estimated coefficients. Among the 34 cities, Florence (Italy) exhibited the strongest acute effect ($\beta = 0.0783$, 95% CI: [0.0058, 0.1508]). Overall, 24 of the 34 cities showed positive associations, indicating that higher same-day $\text{PM}_{2.5}$ exposure was generally linked to increased nocturnal coughing.

Because influenza epidemics can confound cough-related outcomes—people tend to cough more frequently during flu seasons regardless of air quality—we further restricted the analysis to non-flu periods. The non-flu season was defined as days when the influenza rate remained below 10%. During these periods, influenza activity was consistently low across all cities, allowing a clearer assessment of the $\text{PM}_{2.5}$ –cough association. Positive linear relationships were observed in 25 of the 34 cities, as shown in Figure 2(b).

In contrast, the pooled multi-city correlation appeared negative, diverging from most city-specific results. This apparent discrepancy arises because the pooled model does not control for systematic between-city differences: cities with both higher baseline cough rates and higher $\text{PM}_{2.5}$ levels exert disproportionate influence on the overall estimate, thereby masking the underlying within-city positive associations.

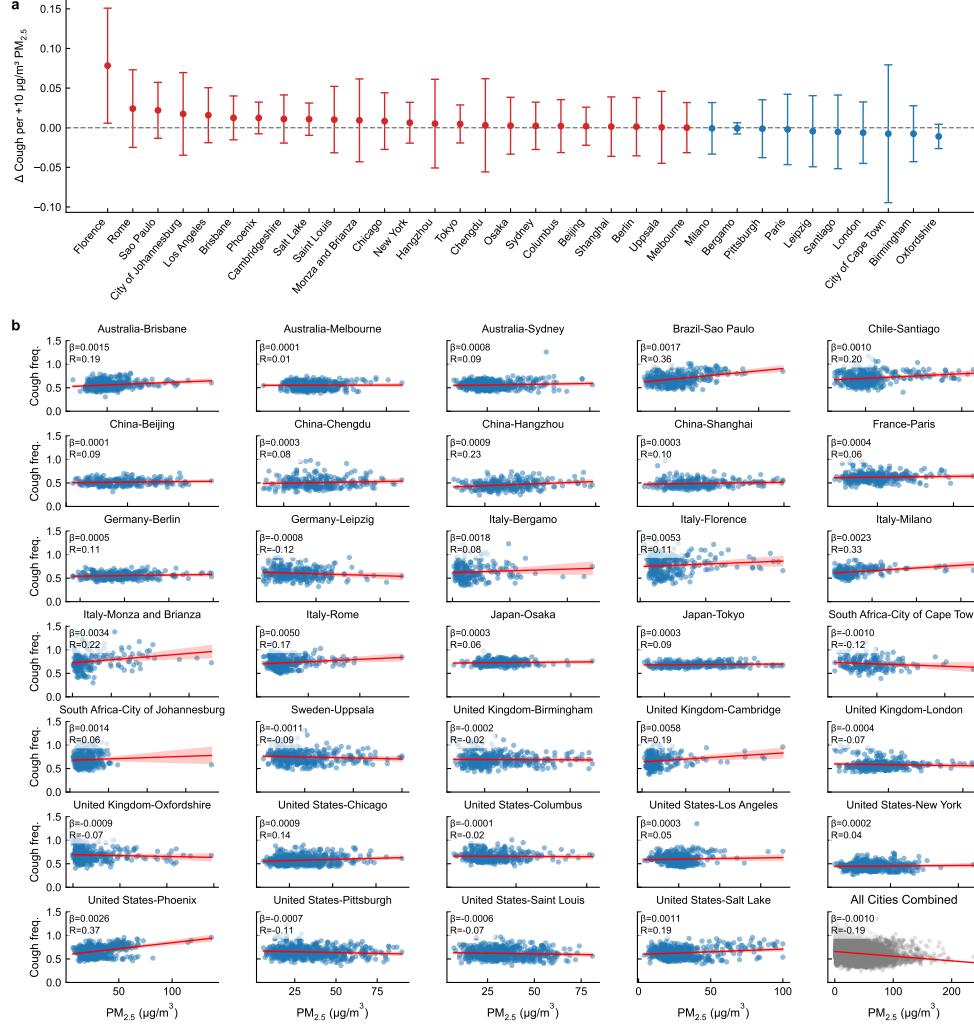


Figure 2 Within-city associations between PM_{2.5} concentrations and nocturnal cough frequency. (a) City-specific PM_{2.5} effects estimated using generalized additive models (GAMs) with a linear PM_{2.5} term. Error bars denote 95 % confidence intervals. (b) Linear associations during non-flu seasons (influenza rate ≤ 10 %). Each panel shows observed data points (blue), the fitted regression line (red), and its 95 % confidence band (shaded area). β denotes the regression slope, and R the Pearson correlation coefficient.

Multi-city associations between PM_{2.5} and nocturnal cough

Building on the city-specific results, we next quantified the overall short-term association between PM_{2.5} and nocturnal cough while explicitly accounting for between-city differences. We first conducted a random-effects meta-analysis using the DerSimonian–Laird method to pool city-level estimates and their standard errors [22]. The

combined effect size was $\beta = 0.0022$ (95% CI: $[-0.0022, 0.0066]$), with negligible heterogeneity ($\tau^2 = 0$, $I^2 = 0\%$) and a 95% prediction interval of $[-0.0022, 0.0066]$.

To further explore temporal lag effects, interaction mechanisms, and potential nonlinear exposure–response relationships, we employed a fixed-effects panel regression model adjusting for meteorological variables and influenza activity (see [Method](#) section). Relative risks (RRs) were estimated per 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ across multiple lag structures. As shown in Figure 3(a), the strongest associations were observed at lag 0 and lag 1, suggesting that same-day and previous-day exposures acutely increase nocturnal cough frequency.

Table 2 summarizes the regression results for lag 0 (Model 1: Main effects). Short-term $\text{PM}_{2.5}$ exposure was significantly associated with higher cough frequency ($\beta_1 = 0.0004$, 95% CI: 0.0001–0.0007, $p < 0.05$), corresponding to a relative risk of 1.004 (95% CI: 1.001–1.008) per 10 $\mu\text{g}/\text{m}^3$ increase. Influenza activity was also strongly associated with increased cough ($\beta_2 = 0.3741$, 95% CI: 0.2148–0.5334, $p < 0.001$). Among meteorological covariates, higher temperature was inversely associated with cough ($\beta_3 = -0.0060$, 95% CI: -0.0075 to -0.0045 , $p < 0.001$), whereas precipitation showed no significant effect. The model explained a large proportion of temporal variation ($R^2 = 0.7336$).

Figure 3(b) illustrates the relationship between $\text{PM}_{2.5}$ and the partial residuals of cough frequency after adjusting for meteorological and influenza effects. The positive slope ($\beta = 0.0002$) was consistent with most city-specific estimates and suggests that the attenuation observed in the pooled multi-city analysis (Figure 2) reflects unaccounted between-city heterogeneity.

Figures 3(c–d) present exposure–response curves without lag for the full year and the non-flu season, respectively. Relative risks were estimated at different $\text{PM}_{2.5}$ concentrations using the WHO 24-hour guideline (15 $\mu\text{g}/\text{m}^3$) as reference. Both curves were monotonic but nonlinear, with wider confidence intervals at higher concentrations. Steeper slopes were observed below the WHO guideline, suggesting measurable risks even at relatively low exposure levels and no apparent safe threshold. In the non-flu season (Figure 3d), the curve steepened above 15 $\mu\text{g}/\text{m}^3$, indicating stronger adverse effects at higher concentrations.

To test for effect modification, we added an interaction term between $\text{PM}_{2.5}$ and influenza prevalence (see [Method](#) section). In Model 2 (Table 2), which included this interaction, the main effect of $\text{PM}_{2.5}$ remained statistically significant ($\beta = 0.0006$, $p = 0.0012$), indicating that air pollution independently contributes to nocturnal cough even under low influenza activity. Influenza prevalence itself was also positively associated with cough frequency ($\beta = 0.4340$, $p < 0.001$), confirming a clear elevation in respiratory symptoms during flu outbreaks. The interaction term between $\text{PM}_{2.5}$ and influenza activity ($\beta = 0.0027$, $p = 0.0647$) was positive and marginally significant, implying that the effect of $\text{PM}_{2.5}$ on cough may be amplified during periods of elevated influenza activity. For example, a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ was associated with approximately a 6% increase in cough rate during low influenza activity, rising to about 8–9% during high-influenza periods. This finding supports a biologically plausible mechanism in which air pollution exacerbates respiratory responses to viral infections.

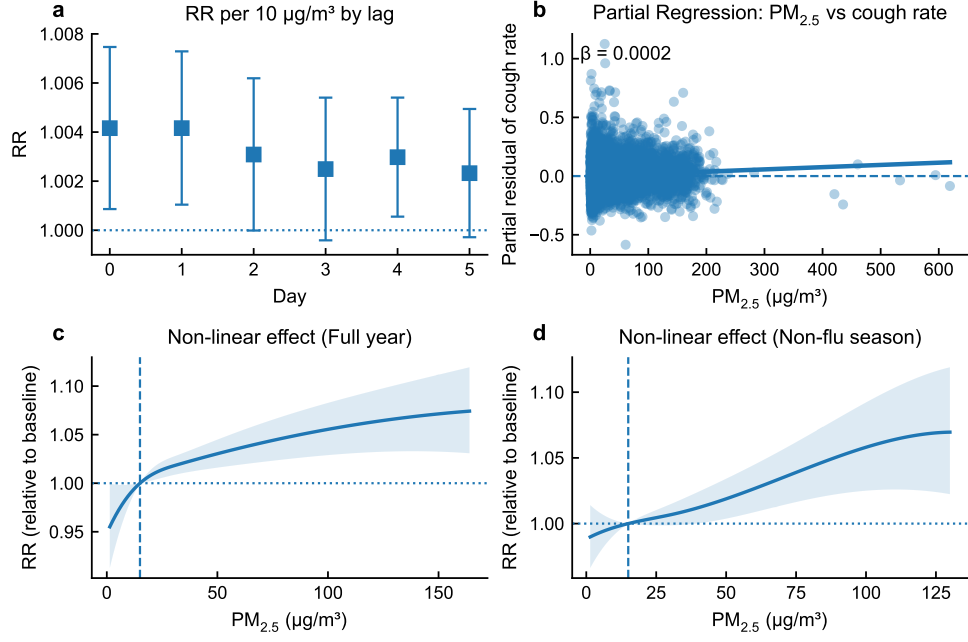


Figure 3 Association between cough frequency and PM_{2.5} exposure. a) Lagged relative risk (RR) per 10 µg/m³ of PM_{2.5} by day; b) Partial regression analysis showing the relationship between PM_{2.5} (lag 0) and cough rate residuals, controlling for other factors; c) Non-linear exposure-response curve for PM_{2.5} (lag 0) and cough frequency across the full year; d) Non-linear exposure-response curve for PM_{2.5} (lag 0) and cough frequency during non-flu season.

Quasi-experiment leveraging wildfire-related PM_{2.5} surges

The time-series analyses above estimated the effects of PM_{2.5} on nocturnal cough after adjusting for multiple confounders. To further strengthen causal inference, we conducted a quasi-experimental analysis exploiting a sudden, exogenous increase in PM_{2.5} concentrations resulting from a major wildfire. Large-scale forest fires serve as natural experiments for evaluating the short-term health impacts of abrupt air-quality deterioration. Within our study period, a severe wildfire occurred in Los Angeles, USA, beginning on January 9, 2025 [23], producing a sharp and sustained rise in PM_{2.5} concentrations.

As shown in Figure 4, PM_{2.5} levels surged during the wildfire episode—by 12.8-fold compared with January 8, 2025, and by 15.8-fold relative to the same calendar day in 2024—and remained elevated through January 12.

Under typical seasonal patterns, nocturnal cough frequency gradually declines in January as influenza activity subsides. However, coinciding with the wildfire-induced PM_{2.5} surge, we observed a distinct rebound in cough incidence beginning on January 9, reversing the downward trend observed in previous years (gray dashed line in Figure 4). Cough frequency on January 9, 2025, was 1.02 times that of January 8, 2025,

Table 2 Fixed-effects regression estimates of the association between PM_{2.5} and nocturnal cough frequency, adjusted for meteorological variables and influenza activity.

Variable	Estimate	Std. Error	95% CI	P-value
Model 1: Main effects				
PM _{2.5}	0.0004	0.0002	[0.0001, 0.0007]	0.0134
Influenza rate	0.3741	0.0812	[0.2149, 0.5334]	< 0.0001
Temperature (°F)	-0.0060	0.0008	[-0.0075, -0.0045]	< 0.0001
Precipitation (mm)	-0.0006	0.0053	[-0.0110, 0.0098]	0.9144
Model 2: Including interaction term (PM_{2.5} × Influenza rate)				
PM _{2.5}	0.0006	0.0002	[0.0002, 0.0010]	0.0012
Influenza rate	0.4340	0.1209	[0.1970, 0.6710]	< 0.0003
PM _{2.5} × Influenza	0.0027	0.0015	[-0.005, 0.055]	0.0647
Temperature (°F)	-0.0069	0.0009	[-0.0088, -0.0051]	< 0.0001
Precipitation (mm)	-0.0036	0.0056	[-0.0147, 0.0074]	0.5200

Notes: Estimates are from two-way fixed effects models (city and time). Standard errors are clustered by both city and date. Model 1 includes main effects only; Model 2 additionally includes an interaction between PM_{2.5} and influenza rate.

and 1.04 times that of January 9, 2024, remaining elevated relative to the historical baseline until January 12.

Taken together, these observations provide quasi-experimental evidence that wildfire-driven air pollution can acutely exacerbate nocturnal respiratory symptoms in urban populations, reinforcing the causal interpretation of the time-series and panel regression findings.

Discussion

This study provides robust, population-scale evidence that short-term exposure to PM_{2.5} acutely increases nocturnal cough frequency, with effects detectable both on the day of exposure and the following day. The association was consistent across multiple cities and remained robust after adjustment for meteorological and infectious confounders. These findings demonstrate that even relatively low concentrations of PM_{2.5}—levels commonly encountered in urban environments—can trigger measurable respiratory symptoms, considering nocturnal cough as an early and sensitive marker of airway irritation. Moreover, the monotonic, nonlinear exposure–response relationship observed here reinforces the absence of a “safe” threshold for fine particulate matter [24–27], challenging the adequacy of current air quality standards and underscoring the pervasive health burden associated with urban pollution.

Our results also reveal that influenza activity modifies the association between PM_{2.5} and nocturnal cough, with stronger effects observed during periods of elevated influenza prevalence. This finding suggests that viral infections may amplify susceptibility to particulate pollution, aligning with prior experimental and epidemiological evidence that air pollutants exacerbate airway inflammation and impair host immune responses [5, 19]. Such synergistic interactions between environmental and

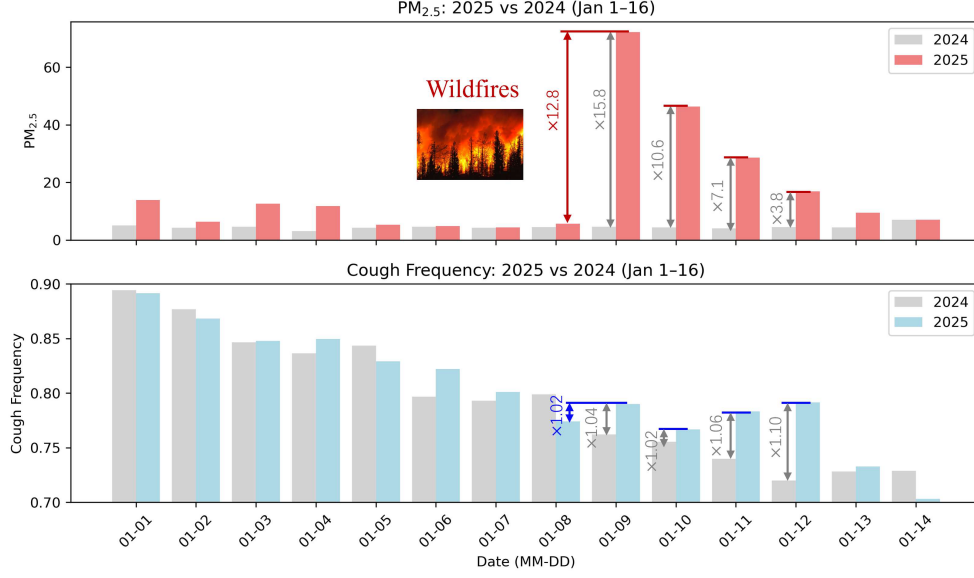


Figure 4 Wildfire-related PM_{2.5} surge and nocturnal cough response in Los Angeles. Daily PM_{2.5} concentrations (top) and nocturnal cough frequency (bottom) during January 1–16, 2025 (colored bars) compared with the same period in 2024 (gray bars). A severe wildfire starting from January 9 2025 caused a sharp and sustained rise in PM_{2.5} levels. Correspondingly, nocturnal cough frequency, which typically declines in January following the influenza peak, showed a distinct rebound during the wildfire episode, diverging from the seasonal downward trend observed in previous years. These quasi-experimental patterns suggest that wildfire-driven air pollution can acutely exacerbate nocturnal respiratory symptoms in urban populations.

infectious stressors may have growing significance as overlapping pollution and epidemic events become more frequent in densely populated regions. Future research should further characterize these interactions across diverse respiratory pathogens and identify populations most vulnerable to their combined effects.

A major innovation of this work lies in the use of mHealth technologies for environmental epidemiology. By capturing nocturnal cough through smartphone-based monitoring and machine learning models, we introduce a scalable, objective, and non-invasive approach for quantifying early respiratory responses in real-world settings. This represents an important complement to conventional endpoints such as hospital admissions and mortality [28–32]. The finding that PM_{2.5} exposure increases nocturnal cough suggests that the true health burden of air pollution may be substantially underestimated when relying solely on severe clinical outcomes. More broadly, our results illustrate the potential of mHealth platforms to enable continuous, high-resolution surveillance of environmental health impacts on a global scale [9, 10].

Beyond its respiratory relevance, nocturnal cough also has systemic implications. Sleep fragmentation caused by nighttime coughing may link air pollution to cardiometabolic dysfunction, impaired cognitive performance, and neurodegenerative

risk [33, 34]. Thus, short-term particulate exposure may have far-reaching consequences that extend beyond acute respiratory irritation, offering a potential pathway through which pollution influences overall neurological and metabolic health. This perspective shifts the focus of air pollution research from overt morbidity and mortality toward earlier manifestations that affect quality of life and long-term well-being.

Marked heterogeneity across cities further indicates that contextual factors—such as socioeconomic status, healthcare access, building environments, and local pollution sources—may shape the magnitude of observed associations. Although $\text{PM}_{2.5}$ concentrations varied widely across locations, the positive direction of effect remained consistent, emphasizing the generalizability of these findings. Future research integrating individual-level data on indoor air quality, pre-existing respiratory disease, and behavioral factors could help disentangle these contextual influences and refine risk estimation.

The wildfire case study provides quasi-experimental evidence that abrupt increases in $\text{PM}_{2.5}$ can acutely aggravate respiratory symptoms beyond expected seasonal baselines. These findings align with recent multi-city studies showing increased hospitalizations during wildfire events [35, 36]. As climate change accelerates the frequency and intensity of wildfires and other extreme weather events [37–39], their contribution to acute respiratory morbidity is likely to grow, rivaling that of chronic anthropogenic pollution sources. Integrating climate resilience and early-warning systems into air quality management will therefore be essential to protect public health.

Despite its strengths, this study has several limitations. Although we adjusted for key meteorological and epidemiological confounders, unmeasured factors—such as personal behaviors, comorbidities, or indoor exposures—may still influence results. The use of mHealth data may also introduce selection bias, as users tend to be younger and healthier than the general population, potentially leading to conservative estimates of the true burden. Furthermore, while cough detection via audio monitoring is clinically validated and scalable, it may not capture subtler respiratory symptoms that could be detected using multimodal sensors. Finally, our observational design precludes inference on long-term sequelae of repeated acute exposures. Longitudinal mHealth studies integrating wearable and environmental sensors could help clarify cumulative impacts over time.

Conclusion. Our findings provide large-scale, real-world evidence that even short-term exposure to $\text{PM}_{2.5}$ can acutely aggravate nocturnal cough, revealing respiratory effects at pollution levels below current guideline limits. By leveraging mHealth data streams across cities worldwide, this study demonstrates a scalable framework for real-time surveillance of air pollution impacts and underscores the urgency of integrating digital health, environmental monitoring, and climate resilience into public health policy.

Method

Data sources

Cough data. Nighttime cough data were obtained from *Sleep Cycle* (Sleep Cycle AB, Sweden), a widely used mobile health application for sleep monitoring. The app

passively records nighttime audio using the smartphone’s built-in microphone, typically between 10:00 PM and 7:00 AM. A validated machine learning–based algorithm automatically detects and timestamps cough events from raw audio recordings. The resulting cough frequency (number of coughs per hour) was aggregated at the city–date level for analysis. All data were collected as anonymized usage statistics and did not include personal identifiers or require additional consent beyond the general terms of app use. Optional demographic information (age and gender) was self-reported and analyzed only in aggregated, anonymized form. Further methodological details are provided in the next section.

Spatial aggregation of individual cough data. Individual-level nocturnal cough records were spatially aggregated to the city level to enable multi-city analyses. Each participant’s geolocation (latitude and longitude) was mapped to its corresponding administrative boundary using the `gadm_410.gpkg` dataset, which provides high-resolution global administrative boundaries from the Global Administrative Areas (GADM) database (version 4.1;²). Administrative polygons corresponding to major urban centers were extracted, and each user was assigned to a city based on their recorded location. We used Level 2 administrative units (corresponding approximately to metropolitan boundaries) to define cities. Daily cough frequencies were then averaged across all users within each city to produce a consistent, city-level time series aligned with local air pollution, meteorological, and influenza data.

Air pollution data. Ambient $\text{PM}_{2.5}$ concentrations were obtained from the World Air Quality Index (WAQI) project³, which aggregates data from government-operated air monitoring stations worldwide. For each city, daily $\text{PM}_{2.5}$ concentrations were retrieved from the central monitoring station, and both same-day and lagged exposures were computed for subsequent analyses.

Weather data. Daily meteorological variables, including average temperature ($^{\circ}\text{F}$) and total precipitation (mm), were obtained from the U.S. National Oceanic and Atmospheric Administration (NOAA) Integrated Surface Dataset (ISD)⁴. For each study city, we selected the nearest meteorological station with complete data coverage during the study period (October 2023 to April 2025).

Influenza rate. Data on influenza activity were obtained from official weekly surveillance reports published by national health agencies in each participating country, including the China National Influenza Center (CNIC), the UK Health Security Agency (UKHSA), and the Italian National Institute of Health (*Istituto Superiore di Sanità*, ISS). These data included weekly influenza-like illness (ILI) rates or virologically confirmed case counts, reported at the regional or national level⁵. Weekly values were linearly interpolated to generate daily estimates, which were then standardized within each country to create a continuous influenza activity index used in regression models.

²https://gadm.org/download_world.html

³<https://waqi.info>

⁴<https://www.ncei.noaa.gov/cdo-web/search>

⁵<https://www.who.int/tools/flunet>

Cough detection from nighttime audio recordings

We collected nighttime audio recordings from a popular mobile health application, Sleep Cycle (Sleep Cycle AB, Sweden), which is widely used across diverse geographic and demographic populations. Participants consented to share de-identified sleep-related data for research purposes. Audio was recorded passively during sleep using built-in smartphone microphones, typically between 10:00 PM and 7:00 AM, with an average duration of 7.6 hours per night.

To identify cough events, we applied a validated, machine learning-based cough detection algorithm optimized for smartphone-grade audio [13, 40]. The model combines convolutional and recurrent layers trained on labeled cough audio datasets, and has demonstrated high precision (>90%) and recall (>85%) in prior evaluations. All audio was segmented into overlapping 10-second windows for inference, with no explicit noise reduction applied; instead, the model was trained on sufficiently diverse data to achieve robustness to background noise. Detected cough events were aggregated at the user-night level and normalized by recording hours to generate a continuous variable representing individual cough frequency (events per hour) for each user. Data were then aggregated at the city-date level by computing a weighted mean of individual cough frequencies, with each user’s weight defined as the user’s recording hours divided by the total recording hours in the city. This is equivalent to normalizing the total number of cough events in a city by the total recording hours in that city.

Generalized additive models (GAMs)

To estimate city-specific short-term effects of ambient PM_{2.5} on nocturnal cough, we fitted a generalized additive model (GAM) for each city i :

$$\text{CoughRate}_{it} = \beta_{0,i} + \beta_{1,i} \cdot \text{PM}_{2.5,it} + f_{1,i}(\text{FluRate}_{it}) + f_{2,i}(\text{Temp}_{it}) + f_{3,i}(\text{Precip}_{it}) + \varepsilon_{it},$$

where CoughRate_{it} denotes the average nocturnal cough frequency in city i on day t , and $\text{PM}_{2.5,it}$ is the same-day ambient PM_{2.5} concentration. Smooth functions $f_{1,i}(\cdot)$ – $f_{3,i}(\cdot)$ represent non-linear effects of influenza activity, temperature, and precipitation, modeled with cubic regression splines. The intercept $\beta_{0,i}$ captures city-specific baseline cough levels, and ε_{it} is the residual error term. For the **lagged analyses**, PM_{2.5, $i,t-l$} (PM_{2.5} concentration l days prior) was substituted for PM_{2.5, it} to assess delayed effects. Standard errors were adjusted for autocorrelation using Newey–West corrections.

Fixed-effects panel regression

To quantify the overall multi-city association while accounting for unobserved heterogeneity, we employed a fixed-effects (within) panel regression model. This approach controls for time-invariant city-level factors such as socioeconomic status, healthcare infrastructure, or baseline cough prevalence. The baseline specification (**Model 1**) was:

$$\log(\text{CoughRate}_{it}) = \beta_1 \cdot \text{PM}_{2.5,it} + \beta_2 \cdot \text{FluRate}_{it} + \beta_3 \cdot \text{Temp}_{it} + \beta_4 \cdot \text{Precip}_{it} + \alpha_i + \varepsilon_{it},$$

where α_i denotes city fixed effects and ε_{it} is an idiosyncratic error term. Cluster-robust standard errors were computed at the city level to account for serial correlation. Lagged exposures $\text{PM}_{2.5,i,t-l}$ were examined in sensitivity analyses. Models were estimated separately for the full year, non-flu season (April–October), and flu season (November–March) to capture seasonal variation.

For interpretability, the estimated coefficient β_1 was exponentiated to yield the relative risk (RR) per 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$:

$$\text{RR} = \exp(\beta_1 \cdot 10), \quad \text{RR}_{95\% \text{ CI}} = \left[\exp((\beta_1 - 1.96 \text{SE}_{\beta_1}) \cdot 10), \exp((\beta_1 + 1.96 \text{SE}_{\beta_1}) \cdot 10) \right].$$

Spline-based exposure–response modeling

To explore potential non-linear associations between $\text{PM}_{2.5}$ exposure and nocturnal cough, we extended the fixed-effects framework by replacing the linear $\text{PM}_{2.5}$ term with natural cubic spline basis functions. Specifically, we modeled:

$$\log(\text{CoughRate}_{it}) = \sum_{k=1}^K \gamma_k B_k(\text{PM}_{2.5,it}) + \beta_2 \cdot \text{FluRate}_{it} + \beta_3 \cdot \text{Temp}_{it} + \beta_4 \cdot \text{Precip}_{it} + \alpha_i + \varepsilon_{it},$$

where $B_k(\cdot)$ denotes the k th natural spline basis function (with $K = 4$ degrees of freedom), and γ_k are the corresponding coefficients. Knots were placed at empirical quantiles (10th, 50th, and 90th percentiles) of the observed $\text{PM}_{2.5}$ distribution. Spline bases were implemented using the `bs()` function from the `patsy` Python package. This model enabled visualization of the exposure–response curve and identification of potential thresholds or non-linear risk patterns.

Interaction Model (Effect modification by influenza)

To test whether influenza activity modified the effect of $\text{PM}_{2.5}$ on cough, we introduced an interaction term between $\text{PM}_{2.5}$ and influenza rate (**Model 2**):

$$\begin{aligned} \log(\text{CoughRate}_{it}) = & \beta_1 \cdot \text{PM}_{2.5,it} + \beta_2 \cdot \text{FluRate}_{it} + \beta_3 \cdot (\text{PM}_{2.5,it} \times \text{FluRate}_{it}) \\ & + \beta_4 \cdot \text{Temp}_{it} + \beta_5 \cdot \text{Precip}_{it} + \alpha_i + \varepsilon_{it}. \end{aligned}$$

A positive and statistically significant β_3 indicates that the association between $\text{PM}_{2.5}$ and cough is stronger when influenza activity is elevated, suggesting biological interaction or synergistic vulnerability. All models were fitted using `statsmodels` v0.14.0 in Python 3.11.

Data availability

Nighttime cough data used in this study were obtained from the Sleep Cycle mobile health platform and are subject to privacy restrictions. For research purposes, de-identified aggregated data may be made available upon reasonable request to the corresponding author, subject to approval by the data provider. All other datasets, including daily PM_{2.5} measurements, meteorological records, and influenza surveillance statistics, are publicly accessible from official repositories as described in the Methods section.

Code availability

The analyses were conducted in Python 3.9 using standard packages including pandas, geopandas, numpy, scipy, matplotlib, seaborn, *statsmodels*, and *linearmodels* for regressions. The code used for data processing, panel regression, and figure generation will be made openly available on GitHub upon acceptance of the manuscript, together with an environment file specifying exact package versions.

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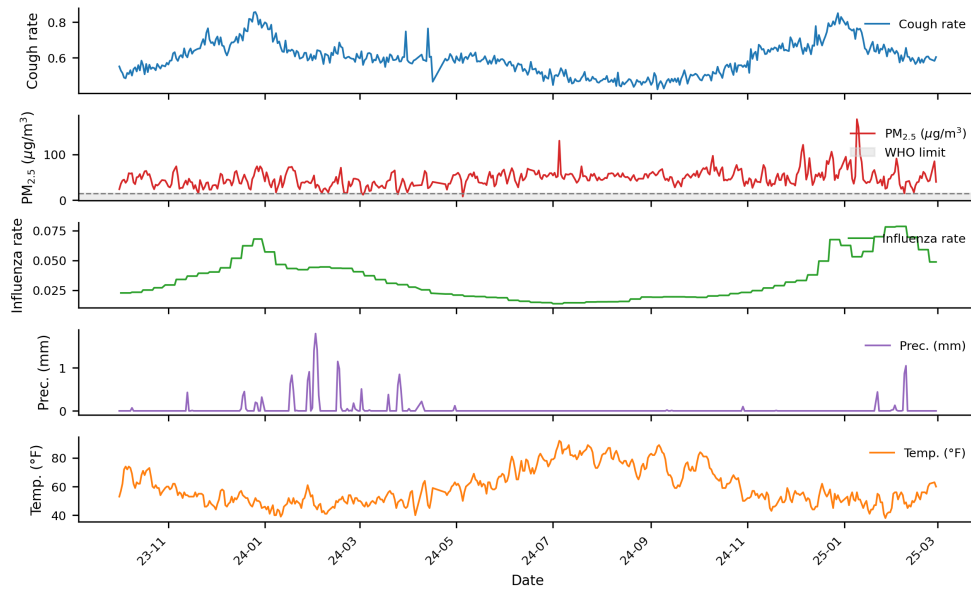
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Supplementary

Supplementary Table 1 Descriptive statistics of the study population by country. Values are reported as male-to-female ratio, mean age with standard deviation, and the 25th (Q1) and 75th (Q3) age percentiles.

Country	Male/Female Ratio	Age (Mean $\pm$ Std)	Age Q1 (25%)	Age Q3 (75%)
Australia	0.93	34.9 $\pm$ 12.07	25	44
Brazil	1.74	36.9 $\pm$ 11.06	29	44
Chile	2.06	37.7 $\pm$ 12.08	28	47
China	1.45	32.6 $\pm$ 9.07	27	37
France	1.45	35.9 $\pm$ 13.02	25	47
Germany	1.87	36.7 $\pm$ 13.04	26	48
Italy	1.93	39.7 $\pm$ 13.05	28	49
Japan	1.83	43.0 $\pm$ 13.07	32	54
South Africa	2.00	41.76 $\pm$ 14.60	31	52
Sweden	1.14	38.31 $\pm$ 14.91	25	52
United Kingdom	1.23	37.0 $\pm$ 13.06	26	46
United States	1.07	39.8 $\pm$ 15.00	27	50

Notes: Demographic information were self-reported and from a subset of the app users that has given us consent to use their data for research. To protect user privacy, we do not report demographic information at the city level; instead, we provide aggregated country-level summaries.



Supplementary Figure 1 Time series of health and environmental variables in Log Angeles over the 500-day study period. The panels display daily trends in cough rate, PM_{2.5} concentration, influenza rate, precipitation, and temperature. The shaded region in the PM_{2.5} panel indicates the WHO recommended exposure limit of 15 µg/m³.