

Article

A Study on the Impact of Sunlight, Ultraviolet Radiation, and Temperature Variability on COVID-19 Mortality: Spatiotemporal Evidence from Small Countries and U.S. States and Territories

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Abstract

Objectives: While the previous literature has established that meteorological conditions are associated with COVID-19 mortality fluctuations, the relative effect of each of these highly correlated factors remains unclear. This study aims to conduct a comparative analysis to determine which of three main meteorological variables—Ambient Temperature, Ultraviolet (UV) Index, and Sunlight Duration—have the strongest negative association with COVID-19 mortality. The objective is to quantify and rank their impact over a 7-to-21-day biological exposure window. **Methods:** We conducted retrospective spatiotemporal analyses in the form of panel Poisson Distributed Lag Models (PDLMs) regression using daily data from 21 January 2020 to 10 January 2023, spanning 129 distinct geographical regions worldwide. To ensure a direct and fair comparison of effect sizes, all meteorological and environmental variables were Z-score standardized. We estimated three independent PDLMs—each focusing separately on UV Index, Ambient Temperature, and Sunlight Duration—with lags ranging from 7 to 21 days. These models controlled for overarching time trends and utilized a categorical variable to account for Region Fixed Effects modeling time-invariant regional health and socioeconomic determinants (e.g., obesity, age demographics, healthcare capacity). Furthermore, distributed lags of daily PM_{2.5} (air pollution) and relative humidity were explicitly included in each model as dynamic confounders. **Results:** The comparison of PDLM results reveals that the UV Index has the strongest negative association with COVID-19 mortality. A one standard deviation increase in the UV Index corresponds to a massive, highly significant cumulative reduction in deaths observed 1 to 3 weeks later ($p < 0.001$). Sunlight Duration is the second-strongest protective meteorological factor, whereas Ambient Temperature has the weakest effect. The distributed lags of particulate matter (PM_{2.5}) and relative humidity were found to be statistically insignificant when modeled alongside the meteorological variables. **Conclusions:** After standardizing variables and controlling for dynamic environmental confounders like air pollution and humidity, the study findings provide robust empirical evidence that meteorological conditions have a strong significant association with COVID-19 mortality fluctuation with a temporal delay, overcoming the confounding effects of merely dry or clear-air conditions.



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Keywords: COVID-19 mortality; Distributed Lag Model; Poisson regression; UV index; temperature; sunlight; environmental epidemiology

1. Introduction

The COVID-19 pandemic has profoundly impacted global health, with temporal patterns of attributed mortality varying substantially across countries. Understanding the environmental and meteorological determinants of these pandemic mortality results remains a requirement in order to improve pandemic preparedness. While socio-demographic, behavioral, and healthcare-related factors have been extensively investigated, the potential associations of mortality with meteorological variables—specifically Sunlight Duration, Ultraviolet (UV) Index, and Ambient Temperature—have received comparatively limited attention.

The relationship between climate and disease has long been recognized in the history of epidemiology. As early as the 5th century BCE, Hippocrates [1] highlighted the influence of seasonal changes, air, water, and sunlight on human health. His insights on the role of solar exposure and climatic variation in shaping disease outcomes foreshadowed the modern exploration of environmental determinants of health. Building upon this historical foundation, our study revisits these questions in the context of the COVID-19 pandemic.

Recent analyses of long-standing seasonal coronaviruses provide valuable insights leading to the hypothesis of endemic seasonality of SARS-CoV-2. One study investigated the patterns of monthly incidence for four human coronaviruses (HCoV-NL63, HCoV-229E, HCoV-HKU1, and HCoV-OC43) across 19 cities in the temperate regions of North America, Europe, and Asia, projecting that SARS-CoV-2 is likely to adopt a similar seasonal transmission pattern, with higher incidence during late fall and winter months [2], emphasizing that understanding these seasonal transmission dynamics is critical for shaping timely and region-specific public health interventions as the virus transitions to endemicity. Another study [3] highlights that the transmission of COVID-19 must be influenced by seasonal environmental factors, with increased infectivity and mortality in colder climates, highlighting the need to consider seasonality when designing intervention strategies. Seasonal patterns are observed across a variety of viral respiratory diseases (VRDs), including influenza viruses, parainfluenza virus (PIV), respiratory syncytial virus (RSV), rhinoviruses, and human coronaviruses (HCoV) [4,5]. Recent comparative analysis also suggests that COVID-19 dynamics are significantly driven by seasonality and environmental factors, exhibiting behavioral patterns similar to influenza-like illnesses [6].

A recent multidisciplinary review [7] emphasizes that the seasonality of COVID-19 should not be evaluated solely based on weather and human behavior; instead, host immune system seasonality also deserves attention as a potentially significant factor. The authors highlight that predicting the seasonality of infectious diseases has become increasingly complex due to rapid, human-induced changes in the Earth's environmental rhythms. They conclude that a proactive, interdisciplinary approach is essential to effectively understand, mitigate, and prevent the seasonal dynamics of infectious diseases within our rapidly changing human–Earth system.

Some previous studies have investigated the role of seasonality in COVID-19 mortality trends. For instance, Ref. [8] found a significant negative correlation ($p < 0.0001$) between temperature and COVID-19 mortality in European countries and U.S. states with a temperate climate, suggesting that lower temperatures may be associated with higher mortality rates. Furthermore, other authors [9] reported that seasonal variation, influenced by both climate and human behavior, contributed to the temporal patterns of COVID-19 mortality across different geographic regions. SARS-COV-2 virus viability was rapidly lost at higher temperatures and higher relative humidity (e.g., 38 °C and relative humidity of >95%). The apparent better stability of SARS coronavirus at low temperature and low humidity environment may facilitate its transmission in the community [10].

Fisman [11] noted that well known respiratory diseases caused by pathogens show seasonal patterns, and that animal coronaviruses are often seasonal and capable of establishing persistence. Hendley et al. [12] reported that two previously identified human coronaviruses, 229E and OC43, exhibit clear winter seasonality, suggesting that seasonality may influence pathogen evolution by creating alternating phases of intense transmission and population bottlenecks, which can both restrict strain diversity and drive rapid genetic changes. Choi [13] found that the seasonal variation in air humidity and temperature aligns with the seasonality of COVID-19 in temperate regions but not in tropical monsoon climates. Interestingly, O'Reilly et al. [14] emphasizes that “the direct effect of winter environmental conditions on transmission is likely to be small,” because while winter may increase viral persistence outdoors and in unheated indoor environments, the majority of SARS-CoV-2 transmission occurs indoors, where environmental conditions remain relatively constant. Zoran et al. [15] emphasized that heavy air pollution, including PM_{2.5}, PM₁₀, ozone (O₃), and nitrogen dioxide (NO₂), may contribute to higher SARS-CoV-2 transmission rates and increase the severity of COVID-19 in the Madrid Community, while Valero et al. [16] reported that in Spain, COVID-19 cumulative incidence was negatively associated with mean air temperature and rainfall, positively associated with altitude, and that provinces with average temperatures below 10 °C had nearly double the incidence compared to provinces above 16 °C. Quinn et al. [17] observed that the pandemic waves aligned closely with the seasonal patterns of human beta-coronaviruses, specifically HCoV-OC43 and HCoV-HKU1 in Northern Europe. According to Park et al. [18] “in northern and southern hemisphere temperate regions, an annual seasonal pattern of human coronaviruses is predictably limited to a few months during winter with higher positivity rate in children compared with adults.”

Some mechanistic hypotheses have been suggested to account for the seasonality of infectious respiratory diseases, including factors like patterns of human behavior, seasonal fluctuations in immune system function, variations in vitamin D levels, changes in melatonin, and seasonal differences in pathogen infectivity [19]. Besides the postulated seasonality of the human immune system's response [20], influenza seasonality can also be associated with factors that vary with the season, such as crowding, ambient temperature, indoor heating, air travel, bulk aerosol transport, or the El Niño Southern Oscillation (ENSO) [21]. As noted by Fisman et al. [22], there is limited conclusive evidence regarding the causes of seasonality in viral infections, asking for further investigation in future research.

A growing body of the epidemiological literature is adding evidence that meteorological factors significantly modulate COVID-19 dynamics. Specifically, higher ambient temperatures may be associated with reduced viral persistence and lower transmission rates [23,24]. Similarly, solar radiation—as measured by either Sunlight Duration or UV Index—has been widely associated with reduced mortality suggesting a protective effect. Observational studies consistently demonstrate that prolonged sunlight exposure and elevated UV Index exhibit a strong negative association with COVID-19 disease severity and mortality, which may be due both to environmental viral inactivation and enhanced host immunity via Vitamin D synthesis [24–26]. However, while the protective effects of these three meteorological variables have been studied in isolation, their high collinearity and the confounding nature of human seasonal behavior have made it difficult for previous models to isolate which specific factor has the strongest association with pandemic positive outcomes, i.e., the reduction in mortality, thus we focus on the methodological gaps remaining in the existing literature. Firstly, many studies rely on simultaneous associations (e.g., studying the association of today's weather with today's deaths), ignoring the critical biological lag between environmental and pathogen exposure and mortality, which the

literature has identified as spanning from one to three weeks. Secondly, most global studies apply a “one-size-fits-all” approach to seasonality, applying uniform seasonal adjustments across all regions, assuming that the distinct winter–summer oscillations observed in temperate zones are universal. This approach is mathematically flawed for tropical and island nations, where seasonal variations are minimal or driven by different dynamics (e.g., monsoon cycles) rather than thermal winters [13]. Furthermore, standard models often fail to separate the seasonality of meteorological variables from that of the socio-institutional variables—such as school calendars, holiday gatherings, and indoor crowding behaviors—which create periodic waves of pathogen transmission that are independent of the weather [7,19–22,27].

In this study, we address the limitations of the previous literature by the comparison of the results of three independent panel PDLM time series regression spatiotemporal analyses of the associations with COVID-19 mortality of the three most relevant meteorological factors in the literature: Ambient Temperature, UV Index, and Sunlight Duration. Some epidemiological models deal with seasonality by filtering out unobserved seasonal human behaviors like indoor crowding or school terms, which are often determined by meteorological conditions. Attempting to filter out this seasonality using artificial regularization waves (e.g., global sine/cosine functions) offers the inconvenience of severe over-adjustment bias, inadvertently stripping the meteorological variables of their true explanatory power. Instead, the approach in this study isolates the true meteorological signal through the following methodology. First, the models include a Region Fixed Effects categorical control variable that provides an aggregate representation of all static socioeconomic characteristics of the regions included in the study (e.g., diabetes prevalence, obesity, age structure), allowing the natural variance of the weather to speak for itself. Second, we include in the statistical model the biological reality of the disease progression by considering lags in a 7-to-21-day exposure window using a Poisson Regression Distributed Lag Model (PDLM). Third, to ensure that the observed weather effects are not merely proxies for “cleaner air” or “drier days,” we explicitly control for the dynamic lags of daily PM_{2.5} (air pollution) and relative humidity.

2. Data and Methods

2.1. Data

In this study, data were collected from 50 United States (US) states, the District of Columbia (D.C.), and 5 territories, and a selection of 73 small or geographically compact countries (e.g., Malta, Luxembourg), as well as larger nations where the population is highly concentrated in specific areas (e.g., Sweden, Norway). For simplicity, we denote them as Regions in the following. The complete list of the 129 Regions and their descriptive variables is provided as Supplementary Material that is available in the zenodo repository (<https://doi.org/10.5281/zenodo.18847049>) (accessed on 1 January 2026).

2.1.1. Mortality (Dependent Variable)

Epidemiological data encompassing COVID-19 mortality were compiled for the study period spanning 21 January 2020 to 10 January 2023. For the countries other than U.S. states included in the analysis, raw daily new death counts were extracted from the “Our World in Data” (OWID) repository, a well recognized source for global COVID-19 pandemic tracking (<https://ourworldindata.org/coronavirus>) URL (accessed on 1 January 2026). The corresponding baseline population figures for these nations were sourced from the 2019 estimates of the World Bank.

For the U.S. regions, mortality count data were obtained from The New York Times COVID-19 GitHub repository (<https://github.com/nytimes/covid-19-data>) (accessed on

1 January 2026), which aggregates and verifies official reports from state and local health departments. Daily new deaths for the U.S. regions were derived by computing the first-order difference of the reported cumulative death counts over time. State-level baseline population data for the year 2020 (reflecting the official 1 April 2020, decennial census counts) were acquired from the U.S. Department of Agriculture's Economic Research Service (USDA ERS) (<https://data.ers.usda.gov/reports.aspx?ID=4049>) (accessed on 01 January 2026). Institutions reporting deaths try to assign (after the fact) the deaths to the day of occurrence, hence some delay in correct reporting may happen. As we are using the data much later than the reported days we assume that all pertinent corrections have been made.

To mitigate short-term reporting artifacts and artificial weekend fluctuations intrinsic to pandemic data reporting, a 7-day moving average was systematically applied to the daily death time series across all 129 geographic Regions. The smoothed daily death counts serve as the dependent variable in the proposed models, while the regional population figures are incorporated as a logarithmic offset term within the Poisson regression framework. This approach strictly adheres to count-data econometric standards, allowing the model to accurately estimate the true incidence risk.

2.1.2. Daily Temperature, Sunshine and UV Index

Meteorological data were systematically retrieved for the 129 Regions over a 3-year period, spanning from 21 January 2020 to 10 January 2023. The dataset covers three key variables: Ambient Temperature, Ultraviolet (UV) Radiation, and Sunlight Duration.

- Ambient temperature data were obtained from the Open-Meteo Historical Weather API. Specifically, the “2m temperature” variable was extracted for each location to represent the daily average ambient temperature.
- Ultraviolet (UV) radiation data, aka UV Index, were obtained via the NASA POWER API (Prediction of Worldwide Energy Resources). We utilized the “ALL-SKY_SFC_UV_INDEX” parameter, which accounts for cloud cover to reflect actual surface exposure. To capture the peak radiative stress, hourly data were retrieved for every day within the study period to identify the daily maximum UV radiation value.
- Sunlight Duration data were obtained from the Open-Meteo Historical Weather API, utilizing the “sunshine_duration” variable. Daily totals of sunshine seconds were retrieved for the entire observation period.

Meteorological variables were not smoothed; raw daily observations were used. To ensure epidemiological relevance, meteorological variables were extracted based on the geographic coordinates of the most populous urban centers within each Region, serving as proxies for population exposure. This approach ensures that the climatic measurements are population-representative, maintaining comparability across all regions analyzed in the study. Since COVID-19 transmission is driven by human interaction, population-dense centers represent the primary loci of infection. Therefore, weather conditions in these demographic hubs provide a more epidemiologically relevant proxy for population exposure than geographically averaged weather data, which would be skewed by uninhabited rural areas. All specific data sources and city names are detailed in the Supplementary Materials (namely files “sunshine.xlsx”, “UV.xlsx”, and “degrees.xlsx”).

2.1.3. Relative Humidity and PM2.5

Daily relative humidity data were obtained from the Open-Meteo Archive API for the same period (21 January 2020–10 January 2023). For each city, the centroid (geometric center) of the predefined city bounding box was calculated and used as the location coordinate.

Hourly relative humidity at 2 m above ground level (relative_humidity_2m) was retrieved for this central point and then averaged to obtain daily mean values.

Daily PM2.5 (particulate matter $\leq 2.5 \mu\text{m}$) concentrations were obtained from the Copernicus Atmosphere Data Store, using the CAMS Global Reanalysis (EAC4) dataset. Data were retrieved for the period 21 January 2020 to 10 January 2023 at 6 h intervals (00:00, 06:00, 12:00, 18:00 UTC). For each city, a predefined geographic bounding box was used. PM2.5 values were extracted for all grid cells within this area, and an area average was calculated across latitude and longitude. The values were converted from kg/m^3 to $\mu\text{g}/\text{m}^3$ and aggregated to daily means.

A comprehensive descriptive summary of the raw environmental and demographic variables—including overall means, variances, minimum, and maximum values across the study period—is provided in “Supplementary File S2 (Descriptive_Data_Summary.xlsx)”.

2.2. Statistical Analysis

To ensure a direct fair comparison of effect sizes across variables with fundamentally different measurement scales, all environmental and meteorological exposure variables were standardized using Z-scores (mean = 0, standard deviation = 1) prior to model estimation.

The dependent variable was the daily number of deaths for each region. Mortality counts were analyzed using panel Poisson Distributed Lag Models (PDLMs) solved by the generalized linear model (GLM) approach, the standard framework for modeling count outcomes under a log-link specification [28,29]. Panel regression data [30] consist of the concatenation of the time series corresponding to various samples of a population. In this study, the meteorological and environmental time series of all regions are concatenated to compose a lengthy time series. To be more specific, the time series are **not** aggregated by averaging across regions. However, region specific effects could also be computed from the model estimates if desired by restricting the data to the region, i.e., not constructing a panel regression. Population size was incorporated into all models as a logarithmic offset term to estimate mortality rates rather than raw counts, a conventional approach when modeling incidence or rate data [30]. The panel regression model is formalized as follows:

$$Y_{it} \sim \text{Poisson}(\mu_{it}), \quad (1)$$

$$\log(\mu_{it}) = \log(\text{Pop}_i) + \alpha_i + \beta_1 \text{Time}_t + \sum_{k=7}^{21} \gamma_k \text{Climate}_{i,t-k} + \sum_{k=7}^{21} \delta_k \text{Hum}_{i,t-k} + \sum_{k=7}^{21} \theta_k \text{PM2.5}_{i,t-k}. \quad (2)$$

where Y_{it} is the daily death count at time t in Region i , Pop_i is the population offsets in Region i , α_i is the region i fixed effects modeled by a categorical variable, $\text{Climate}_{i,t-k}$ is one of the meteorological variables (either Ambient Temperature, UV Index, or Sunlight Duration, depending on model) at lag k relative to time t , and $\text{Hum}_{i,t-k}$ and $\text{PM2.5}_{i,t-k}$ are the dynamic confounders, i.e., relative humidity and PM2.5 air pollution at lag k relative to time t , in Region i . A linear time trend $\beta_1 \text{Time}_t$ is also added to capture global temporal mortality dynamics not explained by other predictors.

To isolate the specific impacts of the three primary meteorological exposures—Ambient Temperature, UV Index, and Sunlight Duration—three independent PDLMs were estimated based on Equation (1). For each meteorological variable, a distributed lag structure was implemented covering lags 7–21 days prior to observation. This lag window was selected to capture delayed meteorological effects on the disease progression consistent with incubation, disease progression, and reporting delays. The multi-lag specification follows established environmental time series methodology in which all lag terms are simultaneously estimated to avoid mutual confounding across lag days [31,32]. To rule out the

possibility that solar or thermal variables merely acted as proxies for clear sky or dry weather conditions, daily relative humidity and PM_{2.5} were explicitly included in all models as dynamic control variables with identical 7–21-day lag structures.

The modeling strategy of the study deliberately excluded both parametric seasonal waves [31,32] (e.g., harmonic sine/cosine functions) and non-parametric seasonal controls (such as cyclic week-of-year fixed effects) from the primary specification. This decision was made to avoid over-adjustment bias. Human seasonal behaviors—such as indoor crowding and altered social dynamics—are inherently driven by the meteorological conditions themselves; therefore, it can be argued that filtering out the “season” through mathematical waves or strict cyclic indicators artificially suppresses and absorbs the true explanatory power of the meteorological variables.

Regional fixed effects were included through a region identifier categorical variable to control for time-invariant heterogeneity between healthcare systems, reporting practices, or cultural factors. The inclusion of fixed effects in panel regression models is a standard strategy to eliminate bias from time-invariant omitted variables [30,33]. In accordance with standard panel data econometric theory [30,34], time-invariant demographic and socioeconomic covariates (e.g., baseline diabetes prevalence, age distribution, and others) are aggregately represented by the regional intercepts and naturally excluded from the equations to avoid perfect multicollinearity (rank deficiency).

Standard errors were adjusted using a quasi-likelihood dispersion correction to account for over-dispersion, a standard approach for count models when variance exceeds the mean [29]. Model adequacy was evaluated through Variance Inflation Factors (VIFs), residual diagnostics, autocorrelation function (ACF) analysis, and correlation structure assessment.

3. Results

To evaluate the isolated effects of meteorological conditions on COVID-19 mortality, three independent Poisson Distributed Lag Models (PDLMs) were estimated. By utilizing Z-score standardized variables, the coefficients represent the change in log-mortality associated with a one-standard-deviation increase in each respective environmental factor. Figure 1 plots the COVID-19 daily deaths and the environmental and meteorological variables for the state of New York.

Figure 2 illustrates the cumulative impact of environmental variables on COVID-19 mortality over the biologically significant lag window of 7 to 21 days. Each curve represents the results of an independent PDLM analysis over a model given by Equations (1) and (2) where the *Climatic* variable corresponds to one of the meteorological variables. All three meteorological variables exhibited a statistically significant negative association with mortality for (almost) all lags, which may be interpreted as a protective effect. The “Supplementary File S1 (Standardized_DLM_Results).xlsx” provides comprehensive outputs, including daily lag coefficients, robust standard errors, dispersion parameters, and Pseudo-R² metrics, for the independent UV Index, Ambient Temperature, and Sunlight Duration models. For the reader interested in the local regional results, i.e., the 7–21-day lags PDLM restricted to each region, we provide in the file “Regional Effects.xlsx” in the Supplementary Materials the cumulative effects found for each meteorological variable.

The results of the separate panel PDLM analysis reveal that all three meteorological variables—UV Index, Ambient Temperature, and Sunlight Duration—have a statistically significant ($p < 0.001$) negative association with COVID-19 daily mortality, which can be interpreted as a protective effect. However, the magnitude of this effect varies considerably. The UV Index has the strongest association. Controlling for regional fixed effects, global time trends, and dynamic confounders (PM_{2.5} and relative humidity), a one-standard-

deviation increase in the UV Index resulted in the largest cumulative reduction in mortality across the 7-to-21-day exposure window. Sunlight Duration is the second-strongest association, while Ambient Temperature exhibited the weakest impact among the three. Importantly, within these strictly controlled specifications, the distributed lags for relative humidity and PM2.5 (air pollution) were largely statistically insignificant, confirming that the observed mortality reductions are primarily driven by solar radiative intensity rather than general clear-sky or dry-weather conditions.

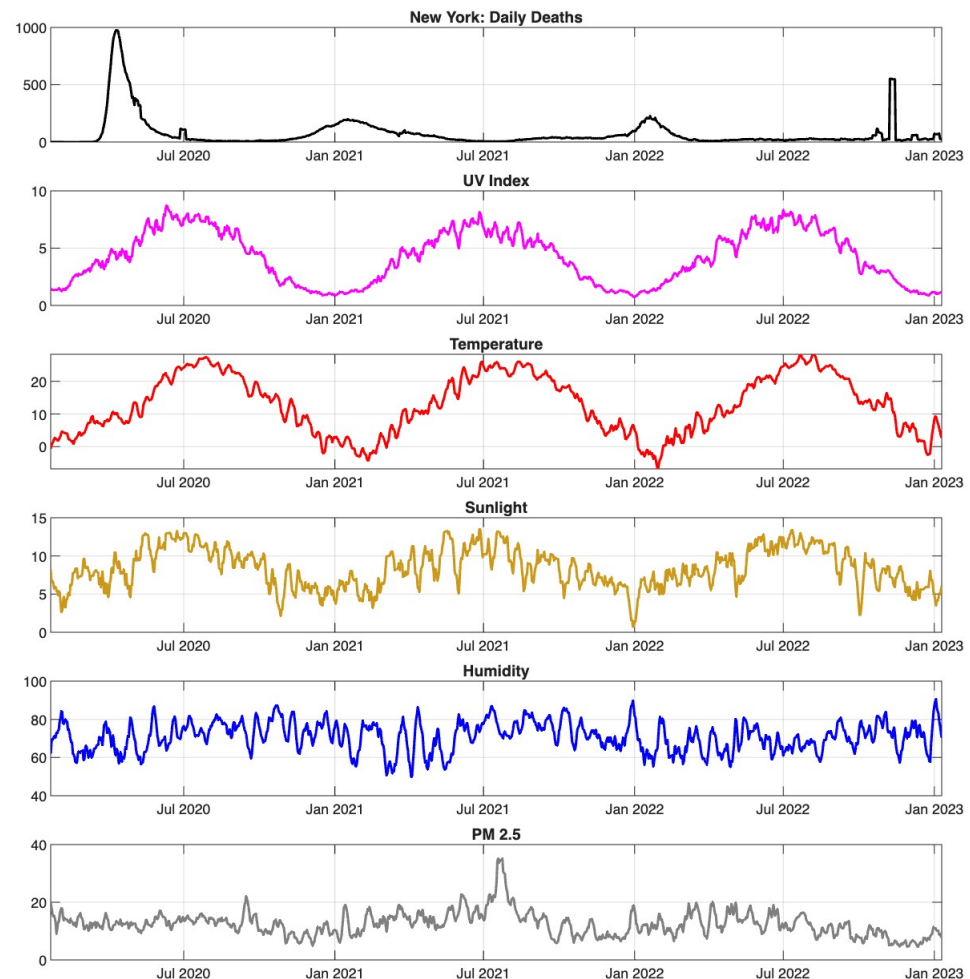


Figure 1. Time series of meteorological and environmental variables and the daily COVID-19 death count for the state of New York. Death count time series is smoothed by 7-day moving average. Meteorological and environmental time series are not smoothed for use in the regression models.

Figure 3 presents how each independent model can be used to reconstruct the aggregated time series analysis across all 129 regions. Each of the models tracks the actual global mortality waves (black dots) with high fidelity, successfully capturing major pandemic peaks and troughs. The close alignment between the predicted and actual mean values confirms that the model generalizes well across diverse geographies without overfitting to local outliers.

At the regional level, the correlation between observed and predicted daily deaths for the UV Index model was evaluated across 20 geographic regions in Figure 4. The UV Index model demonstrated the highest regional fidelity, consistently producing the strongest Pearson correlation coefficients in both temperate and tropical zones. The regional fit analysis confirms that the standardized PDL model effectively captures local mortality dynamics without relying on artificial seasonal control techniques. For comparative purposes, the

corresponding micro-level predictive accuracy plots for the Ambient Temperature and Sunlight Duration models are provided in “Supplementary Figure S1” and “Supplementary Figure S2”, respectively.

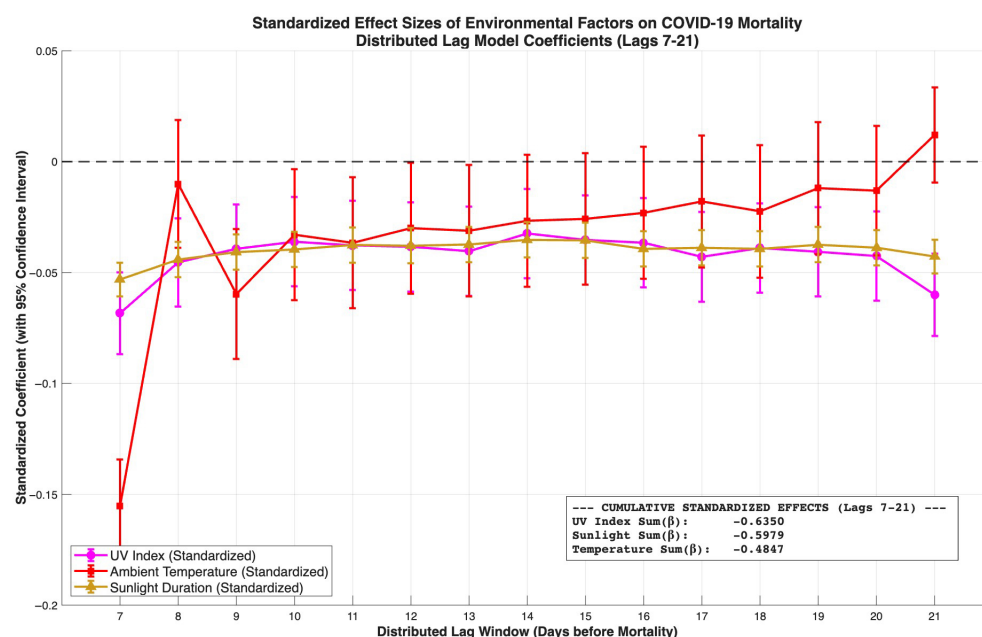


Figure 2. Standardized effect size comparison across lag days. This figure illustrates the estimated coefficients and 95% confidence intervals for the UV Index, Ambient Temperature, and Sunlight Duration PDLMs from lag day 7 to 21. Values below the zero line indicate an association with reduction in mortality (protective effect). All variables are Z-score standardized, allowing for direct comparison of effect magnitudes. The UV Index (magenta) consistently demonstrates the most significant protective impact across the full biological lag window.

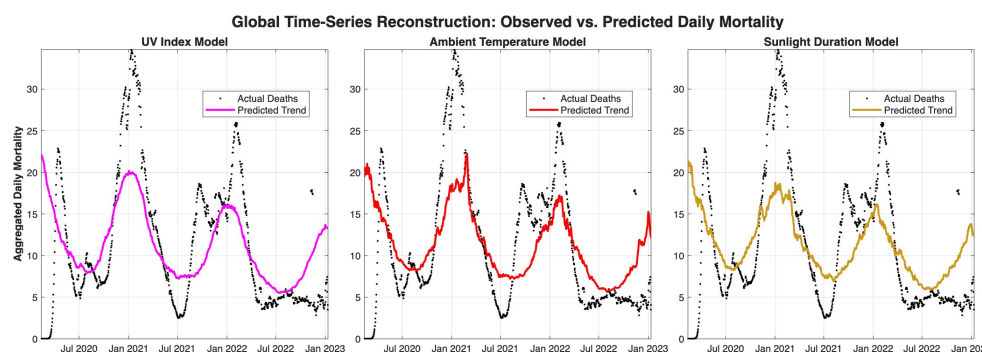


Figure 3. Comparison of actual globally aggregated daily COVID-19 deaths versus model predictions. The PDLMs successfully capture macro-level pandemic fluctuations using only the linear time trend, regional fixed effects, and standardized environmental and meteorological predictors. From left to right, predictions from the UV Index, Ambient Temperature, and Sunlight Duration models.

Model Fit Diagnostics

The study includes comprehensive model diagnostics aiming to ensure the statistical validity of the reported estimates. Figure 5 shows graphically the diagnostics results for the specific UV Index panel regression model, the file “Supplementary Material (Model_Diagnostics).pdf” contains the diagnostic plots for the remaining models. Variance Inflation Factors (VIFs) results indicate borderline collinearity for the UV Index lags (VIF ~12) and no multicollinearity for the Sunlight Duration model (all VIFs < 10). While the Ambient Temperature model exhibited elevated VIFs for consecutive daily lags, this is an ex-

pected characteristic of Distributed Lag Models due to the natural temporal autocorrelation of meteorological variables. Because our methodology explicitly evaluates the cumulative impact across the 7- to 21-day lag window rather than isolated single day coefficients, this expected within variable collinearity does not compromise the stability of our estimations. Multicollinearity typically inflates standard errors, which conventionally masks statistical significance. However, despite this severe variance inflation, the aggregated cumulative effects of the primary meteorological variables in our models yielded exceptionally small p -values ($p < 0.001$). In econometric practice, when variables remain highly statistically significant despite elevated VIFs, the multicollinearity does not invalidate the results; rather, it underscores the overwhelming strength and robustness of the underlying signal [35]. Consequently, while individual daily lag coefficients should be interpreted holistically rather than in isolation, their overall cumulative impact provides a mathematically sound measure of environmental influence.

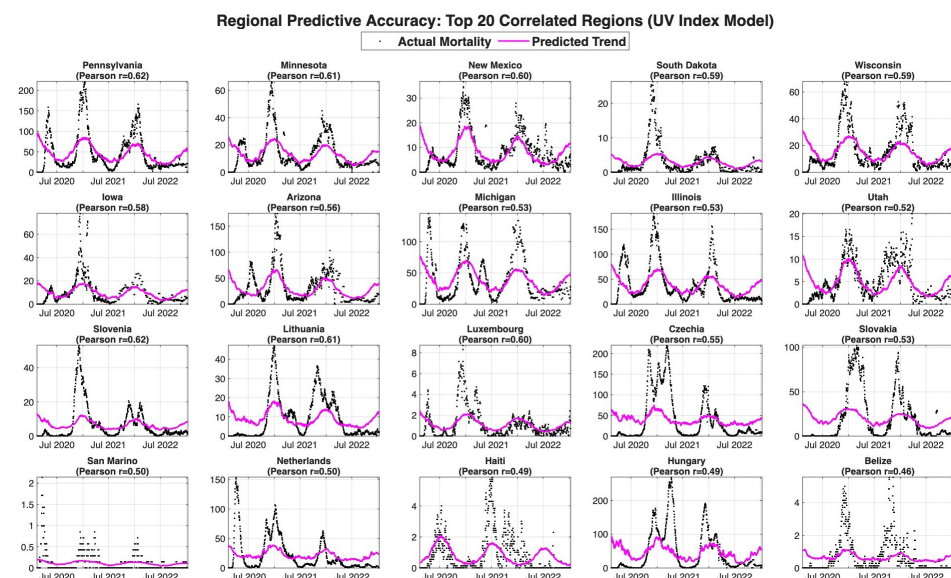


Figure 4. Comparison of the reconstruction of COVID-19 deaths and the predictions of the UV Index model for several selected regions. Each plot includes the Pearson correlation coefficient between actual data and the reconstruction.

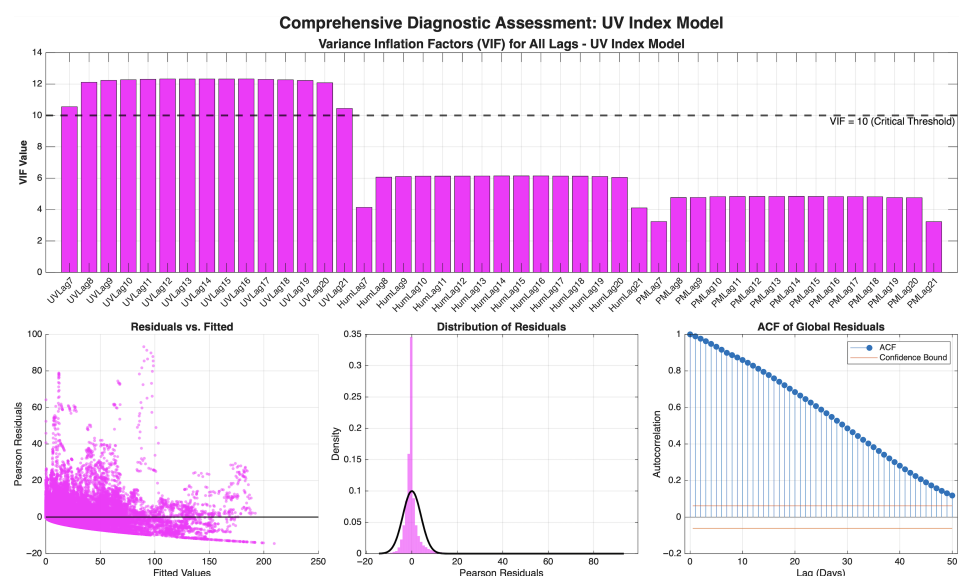


Figure 5. Results of the comprehensive diagnostic assessment of the UV Index model, namely, the Variance Inflation Factors (VIFs) for all the lagged versions of the time series included in the model, the scatter plot and distribution of Pearson residuals, and the autocorrelation of global residuals.

Furthermore, Pearson residuals were consistently centered around zero across all three models, confirming the appropriateness of the selected modeling framework. Finally, Autocorrelation Function (ACF) plots of the global residuals demonstrated a gradual decay, corresponding to the hypothesized transmission dynamics of infectious diseases. To rigorously account for this observed residual autocorrelation and potential over dispersion, robust standard errors were systematically applied in all coefficient estimations.

4. Discussion

This study provides robust empirical evidence that meteorological conditions significantly modulate COVID-19 mortality dynamics, operating with a precise temporal delay. By standardizing the environmental variables and evaluating them within an extended 7-to-21-day distributed lag framework, we overcome the limitations of traditional correlation studies. The analysis unequivocally identified the UV Index as the most potent independent meteorological protective factor associated with decreased COVID-19 mortality. While Ambient Temperature and Sunlight Duration also exhibited protective qualities, their standardized effect sizes were substantially overshadowed by the UV Index.

The suggested protective effect of the UV Index aligns with established photobiological mechanisms. Ultraviolet radiation has demonstrated direct virucidal properties against coronaviruses, including SARS-CoV-2, by damaging viral RNA and protein structures, thereby drastically reducing environmental persistence in aerosols and on surfaces [36,37]. Furthermore, UV exposure is the primary catalyst for human synthesis of Vitamin D, a critical modulator of immune function and respiratory defense mechanisms [38]. Low Vitamin D levels are prevalent during winter months in higher latitudes and have been consistently linked to increased susceptibility to severe COVID-19 outcomes.

This study contributes also to the understanding of the role of commonly considered epidemiological confounders. Previous ecological studies have frequently hypothesized that winter surges in viral transmission are driven by lower absolute humidity (which facilitates aerosol suspension) or increased wintertime air pollution (which impairs respiratory health) [39,40]. However, when dynamic lags of relative humidity and PM_{2.5} were modeled alongside meteorological variables, they were found to be statistically insignificant. This indicates some kind of omitted variable bias in the previous literature: in the absence of UV Index data, models may falsely attribute mortality surges to humidity or pollution, simply because these variables coincide with dark, cloudy, or winter days. The results in this paper confirm that the observed fluctuations in COVID-19 mortality are better explained by the presence of solar radiation itself, than by the secondary conditions such as dry or clear air.

The modeling strategy employed in this study also addresses the issue of the over-adjustment bias caused by artificial seasonal smoothing. Many contemporary models utilize harmonic regressors (sine/cosine waves) or strict cyclic indicators in order to filter out “seasonality” [23], operating under the assumption that human behaviors (such as indoor crowding or school terms) must be controlled for. However, human behavioral shifts are inherently dictated by the weather. Filtering out the “season” through imposing sine/cosine waves inadvertently acts as conditioning on a mediator, absorbing and suppressing the true explanatory power of the environmental and meteorological variables.

By deliberately excluding adjustment for seasonal waves and instead relying on a robust Region Fixed Effects specification, this study allowed the natural effect of the climate on mortality to be accurately measured. The Fixed Effects approach seamlessly absorbed all unobserved, time-invariant regional heterogeneity—such as baseline healthcare capacity, historical reporting practices, and critical demographic structures (e.g., obesity rates, age distributions).

Several limitations must be acknowledged when interpreting these findings. Deliberately excluding explicit seasonal controls (such as harmonic sine/cosine waves or cyclic week fixed effects) from our primary specification, the estimated effects of the environmental variables should be evaluated in conjunction with broader seasonal dynamics. Because weather conditions dictate human socialization patterns—such as increased indoor crowding and reduced ventilation during colder, darker months—the environmental and meteorological variables in our models inevitably capture some of these unobserved behavioral confounders. Therefore, this study does not attempt to establish a causal relation between mortality and meteorological variables.

Furthermore, the statistical significance of the regional intercepts confirm that baseline vulnerability is still heavily modulated by intrinsic regional demographic, socioeconomic, and policy factors. Climate acts as a powerful amplifier or mitigation factor of pandemic risk, but it is not a solitary determinant. A substantial proportion of mortality variation is undoubtedly driven by factors beyond meteorological conditions, such as healthcare system capacity, vaccination rates, and the timing of public health interventions. From a broader perspective, however, the results in this study highlight the necessity of integrating meteorological monitoring into infectious disease forecasting. Regions experiencing prolonged periods of low UV Index—regardless of temperature—should maintain heightened public health vigilance. Recognizing the specific biological deficit caused by low solar intensity can inform targeted, region-specific interventions during winter months, such as improved indoor ventilation, UV sterilization strategies, and proactive Vitamin D supplementation programs.

5. Conclusions

In conclusion, there is mounting evidence that ambient temperature, sunlight, and UV Index influence respiratory disease transmission dynamics. This study was specifically designed to compare their relative impacts under identical, rigorous conditions. By evaluating these three key meteorological variables through a standardized 7-to-21-day Poisson regression distributed lag model adjusted for environmental confounders (i.e., air pollution (PM_{2.5}) and relative humidity) and region heterogeneity, our findings identify the UV Index as the most dominant natural factor associated with decreased COVID-19 mortality, which can be interpreted as a protective effect. Recognizing this specific meteorological signal highlights the importance of targeted public health interventions during low UV Index winter months—such as improving indoor air sterilization and promoting Vitamin D supplementation—to enhance resilience against future respiratory epidemics.

The study has some limitations that point to future research efforts. First, our models do not include interventions both pharmaceutical and non-pharmaceutical because of the difficulty to obtain the data in many regions considered. Secondly, follow-up studies could achieve conclusions over a better spatial resolution over large regions if epidemiological variables can be robustly extracted for smaller population units, given that environmental and meteorological variables can be measured with almost arbitrary resolution. Finally, the integration of cultural and political determinants in the models could provide a new perspective on pandemic preparedness, but they remain elusive to quantification.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/covid6040056/s1>: Degrees.xlsx, Regional Effects.xlsx, sunshine.xlsx, Supplementary Figure S1.jpg, Supplementary Figure S2.jpg, Supplementary File S1 (Standardized DLM Results).xlsx, Supplementary File S2 (Descriptive Data Summary).xlsx, Supplementary Material (Model Diagnostics).pdf, Supplementary Regions.xlsx, UV.xlsx.

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