Nonlinear and Lagged Effects of Climate and Air Pollution on Childhood Pneumonia in West Java, Indonesia

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Abstrak

Pneumonia balita masih menjadi salah satu penyebab utama kesakitan dan kematian anak di negara berkembang. Di Jawa Barat, tercatat 102.576 kasus pneumonia balita pada tahun 2023, termasuk 5.405 kasus pneumonia berat dan 39 kematian balita akibat pneumonia. Penelitian ini bertujuan menganalisis hubungan nonlinier dan hubungan tertunda faktor iklim serta konsentrasi $PM_{2.5}$ dengan kejadian pneumonia balita di Provinsi Jawa Barat menggunakan Distributed Lag Nonlinear Model (DLNM). Data bulanan kasus pneumonia balita dan populasi balita dari 27 kabupaten/kota selama Januari 2015 hingga Desember 2024 diperoleh dari Profil Kesehatan Jawa Barat, sedangkan data suhu udara rata-rata, maksimum, dan minimum, curah hujan, serta kecepatan angin diperoleh dari NASA POWER dan data konsentrasi $PM_{2.5}$ dari NASA Giovanni. Seluruh variabel lingkungan dianalisis dengan lag hingga 12 bulan. Hasil penelitian menunjukkan bahwa curah hujan dan kecepatan angin memiliki asosiasi kumulatif dan tertunda yang signifikan dengan kejadian pneumonia balita pada tingkat provinsi. Curah hujan menunjukkan asosiasi terbalik, dengan relative risk sebesar 0,892 (95% CI: 0,806–0,987) pada lag 8 hingga 0,828 (95% CI: 0,695–0,987) pada lag 12, sedangkan kecepatan angin menunjukkan asosiasi terbalik, dengan relative risk sebesar 0,922 (95% CI: 0,852–0,997) pada lag 4 hingga 0,880 (95% CI: 0,790–0,980) pada lag 11. Suhu udara dan konsentrasi $PM_{2.5}$ menunjukkan asosiasi kumulatif yang nonlinier tetapi tidak signifikan. Analisis pada tingkat kabupaten/kota menunjukkan adanya variasi spasial dalam estimasi relative risk antarwilayah. Temuan ini menekankan pentingnya mempertimbangkan hubungan nonlinier, asosiasi tertunda, dan variasi spasial dalam mengkaji hubungan antara faktor lingkungan dan pneumonia balita.

Kata kunci: pneumonia; balita; distributed lag; nonlinear model, iklim

Abstract

Childhood pneumonia remains one of the leading causes of morbidity and mortality among children in developing countries. In West Java, 102,576 cases of childhood pneumonia were recorded in 2023, including 5,405 cases of severe pneumonia and 39 pneumonia-related deaths among children under five. This study aimed to analyze the nonlinear and lagged associations of climatic factors and $PM_{2.5}$ concentration with childhood pneumonia in West Java Province using the Distributed Lag Nonlinear Model (DLNM). Monthly data on childhood pneumonia cases and the under five population from 27 districts and municipalities from January 2015 to December 2024 were obtained from the West Java Health Profile, while mean, maximum, and minimum air temperature, rainfall, and wind speed data were obtained from NASA POWER, and $PM_{2.5}$ concentration data were obtained from NASA Giovanni. All environmental variables were analyzed with lag periods of up to 12 months. The results showed that rainfall and wind speed had significant cumulative and lagged associations with childhood pneumonia at the provincial level. Rainfall showed inverse associations, with relative risks ranging from 0.892 (95% CI: 0.806–0.987) at lag 8 to 0.828 (95% CI: 0.695–0.987) at lag 12, while wind speed showed inverse associations, with relative risks ranging from 0.922 (95% CI: 0.852–0.997) at lag 4 to 0.880 (95% CI: 0.790–0.980) at lag 11. Air temperature and $PM_{2.5}$ concentration showed nonlinear but non significant cumulative associations. District and municipality level analyses revealed spatial variation in relative risk estimates across regions. These findings highlight the importance of considering nonlinear relationships, delayed associations, and spatial variation when assessing the associations between environmental factors and childhood pneumonia.

Keywords: *pneumonia; under five children; distributed lag; nonlinear model; climate*

1. Introduction

Childhood pneumonia remains one of the leading causes of morbidity and mortality among children under five, particularly in developing [1], [2]. In Indonesia, pneumonia continues to be a major public health concern, with a prevalence ranging from 1.81% to 6.4% among children under five [3]. In West Java, 102,576 cases of childhood pneumonia were recorded in 2023, including 5,405 cases of severe pneumonia and 39 pneumonia related deaths among children under five. West Java, the most populous province in Indonesia, consists of 27 districts and municipalities with diverse climatic conditions and varying levels of air pollution that may influence childhood pneumonia across regions [4], [5]. In West Java, key climate variables such as temperature, humidity, and rainfall exhibit complex spatio-temporal dependencies and vary significantly across different geographical locations [6]. Environmental factors, including air temperature, rainfall, wind speed, and PM2.5 concentration, may affect childhood pneumonia through nonlinear and delayed mechanisms.

The Distributed Lag Nonlinear Model (DLNM) is therefore appropriate for this study because it can simultaneously capture nonlinear exposure response relationships and delayed effects of environmental exposures on childhood pneumonia. Consequently, conventional regression models that assume linear and immediate associations may not adequately capture these complex relationships, highlighting the need for statistical approaches capable of simultaneously modeling nonlinear exposure response relationships and lagged effects [4], [5], [7]. Considering these characteristics together is important because nonlinear relationships describe how the association changes across exposure levels, whereas lagged relationships describe how the association may persist or emerge over time. Previous studies have investigated the associations between environmental factors and pneumonia using various analytical approaches. Distributed lag models have been applied to climate-related pneumonia hospitalizations in South Africa, while nonlinear associations between PM2.5 and pneumonia have been examined in urban China [8], [9]. Other studies have applied the Distributed Lag Nonlinear Model (DLNM) to respiratory diseases but mainly focused on individual climatic variables or diseases other than childhood pneumonia [5], [10].

These studies demonstrate the relevance of nonlinear and lagged approaches for environmental health outcomes, but the available evidence differs in terms of study population, environmental exposures, disease outcomes, and geographical settings. However, the combined nonlinear and delayed effects of multiple climatic factors and PM2.5 concentration on childhood pneumonia remain insufficiently explored, particularly in tropical regions. In West Java, evidence remains limited regarding childhood pneumonia analyzed across all 27 districts and municipalities using a framework that simultaneously captures nonlinear exposure-response relationships and lagged associations for multiple environmental exposures. Therefore, this study aims to analyze the nonlinear and lagged associations between climatic factors, including air temperature, rainfall, and wind speed, as well as PM2.5 concentration and childhood pneumonia in West Java Province using the Distributed Lag Nonlinear Model (DLNM). The analysis aims to identify exposure response patterns and lag periods associated with each environmental factor and to evaluate the spatial variation in relative risk estimates across districts and municipalities in West Java.

To the best of our knowledge, no previous study has simultaneously evaluated nonlinear exposure response relationships, delayed associations, and spatial variation in relative risk estimates for climatic factors and PM2.5 concentration in childhood pneumonia across all districts and municipalities in West Java. Therefore, this study applied the Distributed Lag Nonlinear Model (DLNM) by integrating provincial and district level analyses within a single analytical framework. Spatial variation in this study refers to differences in the estimated relative risks across districts and municipalities, rather than spatial dependence between neighboring regions, as no formal spatial dependence analysis was performed. By characterizing nonlinear exposure-response patterns, lag periods, and spatial variation in relative risk estimates, this study provides a more comprehensive description of environmental-pneumonia associations across West Java. The findings are expected to provide evidence for climate sensitive disease surveillance and support geographically targeted public health interventions [5], [10].

2. Methodology

a. Data

The study was conducted in West Java Province, Indonesia, comprising 18 regencies and 9 municipalities across 27 administrative regions. Monthly data from January 2015 to December 2024 were collected to investigate the associations between climatic factors, PM2.5 concentration, and childhood pneumonia. The data analyzed included monthly childhood pneumonia cases, under-five population, mean air temperature, maximum air temperature, minimum air temperature, rainfall, wind speed, and $PM_{2.5}$ concentration. Childhood pneumonia cases and under-five population data were obtained from the West Java Provincial Health Profile, while mean, maximum, and minimum air temperature, rainfall, and wind speed data were obtained from NASA POWER, and $PM_{2.5}$ concentration data were obtained from NASA Giovanni. The geographical distribution of the study area is presented in Figure 2.1.

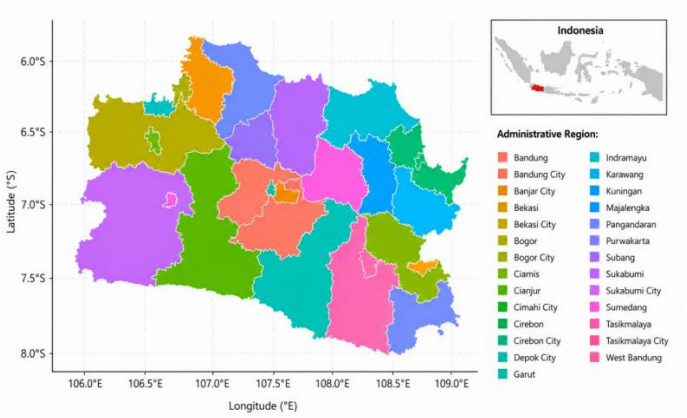


Figure 1 . Administrative map of West Java Province, Indonesia

Figure 2.1 shows the 27 administrative regions included in this study, representing diverse climatic and environmental conditions across West Java Province. The map illustrates the geographical distribution of the 18 regencies and 9 municipalities that constitute the study area and provides the spatial context for the subsequent district level analysis. The monthly childhood pneumonia cases were used as the response variable, with the under five population

included as a log transformed offset. The environmental exposures and their data sources are summarized in Table 1.

Table 1. Research variables and data sources

Variable	Symbol	Unit	Source
Childhood pneumonia cases	$Y_{i,t}$	Cases/month	West Java Provincial Health Profile
Under five population	$Pop_{i,t}$	Persons	West Java Provincial Health Profile
Mean air temperature	X_1	°C	NASA POWER
Maximum air temperature	X_2	°C	NASA POWER
Minimum air temperature	X_3	°C	NASA POWER
Rainfall	X_4	mm/month	NASA POWER
Wind speed	X_5	m/s	NASA POWER
PM2.5 concentration	X_6	µg/m³	NASA Giovanni

Before modeling, all datasets were merged by district/municipality and observation month. Climate and $PM_{2.5}$ exposure data were assigned to each district/municipality for each observation month using the corresponding spatially matched environmental data, ensuring that each environmental exposure was linked to the same district/municipality and month as the childhood pneumonia observations. A monthly time index was created, and the logarithm of childhood pneumonia cases in the previous month was included as a covariate to account for temporal autocorrelation.

b. Distributed Lag Nonlinear Model

Various quantitative time series modeling and forecasting methods, such as Fuzzy Time Series, have been increasingly utilized across different public and administrative sectors in Indonesia to analyze temporal patterns and project future trends [11]. However, the relationship between environmental exposures and childhood pneumonia is often nonlinear and delayed [5], [9]. To simultaneously capture the exposure response and lag response relationships, this study employed the Distributed Lag Nonlinear Model (DLNM) using a cross basis function [9], [10], [12]. For an exposure variable $x_{i,t}$observed in district i at month t, the cross basis function is defined as

$$cb(x_{i,t}) = \sum_{k=1}^{v} \sum_{m=1}^{p} \quad \theta_{km}\, b_k(x_{i,t-\ell})\, c_m(\ell), \qquad \ell = 0,1,\dots,L. \tag{1}$$

where $bk(\cdot)$ and $cm(\cdot)$denote the basis functions for the exposure and lag dimensions, respectively, both specified using natural cubic splines with three degrees of freedom [10]. Three degrees of freedom were selected to provide sufficient flexibility to capture potential nonlinear exposure response and lag response patterns while avoiding excessive model complexity and overfitting. This specification provides a balance between model flexibility and parsimony for the monthly data analyzed in this study. This choice was also applied consistently across all exposure specific models to maintain comparability of the estimated exposure response and lag response relationships. The maximum lag was set to 12 months to represent one annual climatic cycle and capture delayed environmental effects [10]. A 12 month maximum lag was selected

to capture both short term delayed associations and potential seasonal or longer term responses occurring within a complete annual cycle. Six separate DLNMs were fitted, one for each environmental exposure variable, using a quasi Poisson regression model to account for overdispersion in the monthly childhood pneumonia counts [10], [12]:

$$\log(E[Y_{i,t}]) = \alpha + cb(x_{i,t}) + \beta, \log(Y_{i,t-1}) + ns(t_{i,t};,70) + \gamma_i + \log(Pop_{i),t}) \quad (2)$$

where $\mathrm{cb}(\mathrm{x_{i,t}})$represents the cross basis function, $\log(\mathrm{Y_{i,t-1}})$adjusts for temporal autocorrelation, $\mathrm{ns}(\mathrm{t_{i,t}}; 70)$ models long term temporal trends using a natural spline, γ_i enotes district specific fixed effects, and $\log(\mathrm{Pop_{i,t}})$ is included as an offset [10]. After model fitting, residual autocorrelation was assessed using the autocorrelation function (ACF) of the model residuals. The residual diagnostics were used to verify whether substantial remaining temporal dependence persisted after accounting for the previous month pneumonia count and long term temporal trends.

Separate models were estimated for each environmental variable because meteorological variables, particularly temperature indicators, are highly correlated [9], [10]. This approach reduced multicollinearity and improved model stability while allowing the nonlinear and lagged association of each exposure to be evaluated independently [10]. Therefore, the estimated associations should be interpreted as exposure specific rather than mutually adjusted effects [10]. However, because the exposure specific models do not mutually adjust for the other environmental variables, the estimated associations may still reflect confounding from correlated environmental exposures and should not be interpreted as independent causal effects.

c. Model Estimation and Relative Risk

Model parameters were estimated using quasi likelihood to accommodate overdispersion in the monthly childhood pneumonia counts [10], [12]. The estimated cross basis coefficients were then used to calculate the relative risk (RR) at each exposure level and lag period relative to the median exposure value [10]:

$$RR(x, \ell) = exp[f(x, \ell) - f(x_0, \ell)] \quad (3)$$

where $\mathrm{f(x, \ell)}$ represents the estimated exposure lag response surface and $\mathrm{x_0}$ is the median value of each exposure variable. To avoid extrapolation, RR was estimated only within the 10th until 90th percentile range of the observed exposure values.

The cumulative relative risk was obtained by summing the lag-specific effects across the entire lag period [5], [10]:

$$RR_{cum}(x) = exp\left[\sum_{\ell=0}^{L} (f(x, \ell) - f(x_0, \ell))\right] \quad (4)$$

At the provincial level, both cumulative exposure response and lag response associations were estimated with 95% confidence intervals [5], [10]. To evaluate spatial variation in relative risk estimates, the same DLNM framework was applied separately to each district and municipality using its corresponding monthly time series [10]. District level models used a natural spline with 7 degrees of freedom for temporal adjustment, whereas district specific fixed effects were omitted because each model represented a single administrative region [10], [12]. The 7 degrees

of freedom specification was used to provide sufficient flexibility to control for long term temporal trends and seasonal variation without allowing the temporal spline to dominate the exposure response relationships. Model stability was assessed before estimating relative risks. Models producing extreme RR estimates (RR > 30 or RR < 1/30) or convergence warnings were flagged as potentially unstable and excluded from further interpretation and visualization. The RR thresholds were used as a conservative numerical screening criterion to identify estimates that could indicate sparse data, insufficient model stability, or extrapolation at the tails of the exposure response surface rather than as evidence that such RR values are inherently impossible. Excluded models were not used to draw substantive conclusions or to compare relative risks across districts. The analytical workflow is illustrated in Figure 2.2, including data preprocessing, descriptive analysis, cross basis construction, DLNM modeling, relative risk estimation, and result visualization.

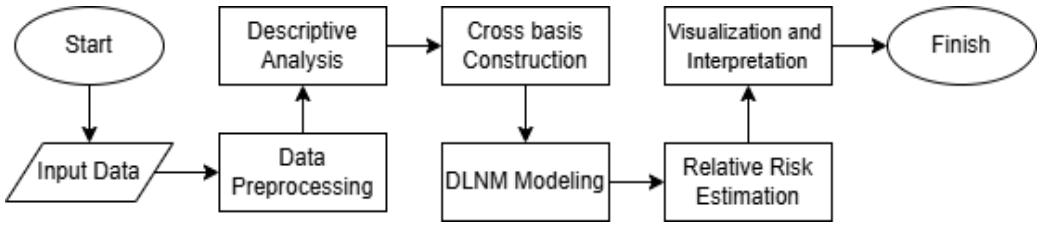


Figure 2. Research workflow of the study

Figure 2 illustrates the analytical workflow adopted in this study, beginning with data preprocessing and descriptive analysis, followed by cross basis construction and DLNM modeling [5], [10]. The fitted models were subsequently used to estimate relative risks, which were then visualized and interpreted to evaluate the associations between environmental factors and childhood pneumonia [5], [10].

3. Results and Discussion

a. Descriptive Statistics

Descriptive statistics were calculated to summarize childhood pneumonia cases and environmental variables across the 27 districts and municipalities in West Java during the study period. The results are presented in Table 2. As shown in Table 2, the monthly number of childhood pneumonia cases averaged 152.90 ± 313.73 cases, indicating substantial variation across districts and municipalities during the study period. Mean, maximum, and minimum air temperatures averaged 24.63 ± 2.03°C, 28.14 ± 1.65°C, and 21.93 ± 2.58°C, respectively, while the mean $PM_{2.5}$ concentration, rainfall, and wind speed were 18.30 ± 5.11 μg/m³, 243.09 ± 163.31 mm, and 1.84 ± 0.70 m/s, respectively. Among the environmental variables, rainfall exhibited the greatest variability, whereas wind speed showed the least, indicating considerable temporal and spatial variation in environmental conditions throughout the study period and supporting the subsequent DLNM analysis.

Table 2. Descriptive statistics

Variable	Mean ± SD	Minimum	Maximum
Childhood pneumonia cases	152.90 ±313.73	0	5633
Mean temperature	24.63 ±2.03	19.88	29.32
Maximum temperature	28.14 ±1.65	24.00	34.12
Minimum temperature	21.93 ±2.58	15.50	28.42
PM2.5	18.30 ±5.11	7.33	43.73
Rainfall	243.09 ±163.31	0.59	966.89
Wind speed	1.84 ±0.70	0.92	5.42

b. Overall Exposure Response Relationship

The Distributed Lag Nonlinear Model (DLNM) was used to estimate the exposure lag response surfaces and cumulative exposure response relationships for six environmental variables over lag periods of 0 - 12 months [5], [9], [10]. The 0-12 month lag period was selected to capture both immediate effects at the time of exposure and delayed effects occurring over a full annual climatic cycle. A maximum lag of 12 months also allows the analysis to account for potential seasonal and longer term delayed responses of childhood pneumonia to environmental exposures. The three dimensional response surfaces illustrate how the estimated relative risk changes simultaneously across exposure levels and lag periods, whereas the cumulative exposure response curves summarize the overall effect of each exposure throughout the entire lag period [5], [9], [10].

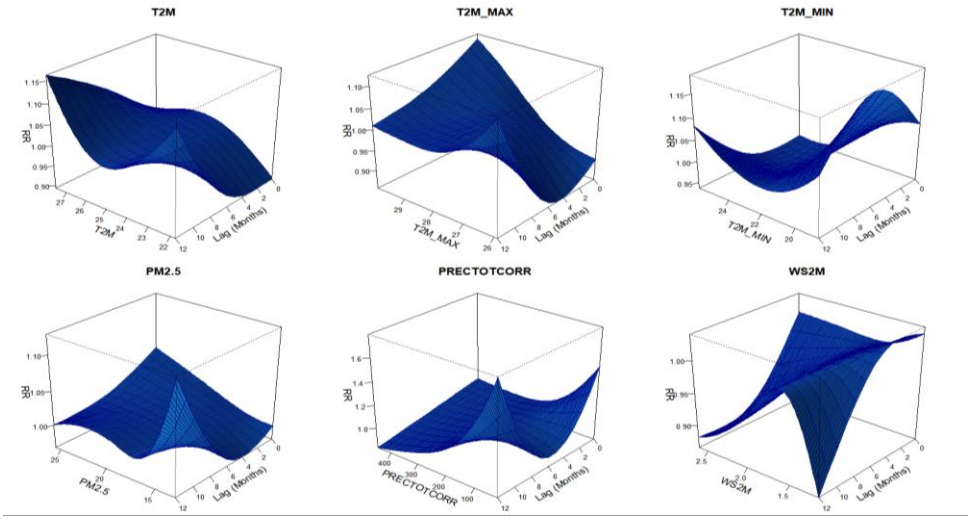


Figure 3. Three dimensional exposure lag response surfaces

Figure 3 shows nonlinear exposure lag response patterns for all environmental variables, although the magnitude and temporal trends differ among exposures [5], [9], [10]. Mean, maximum, and minimum air temperatures together with PM2.5 exhibit relatively modest changes in relative risk across exposure levels and lag periods. In contrast, rainfall and wind

speed showed significant cumulative associations, with higher exposure levels associated with lower cumulative relative risks of childhood pneumonia. These inverse associations indicate that the estimated RR decreased as exposure increased over the evaluated exposure response range, although the observational design does not allow this pattern to be interpreted as a causal protective effect.

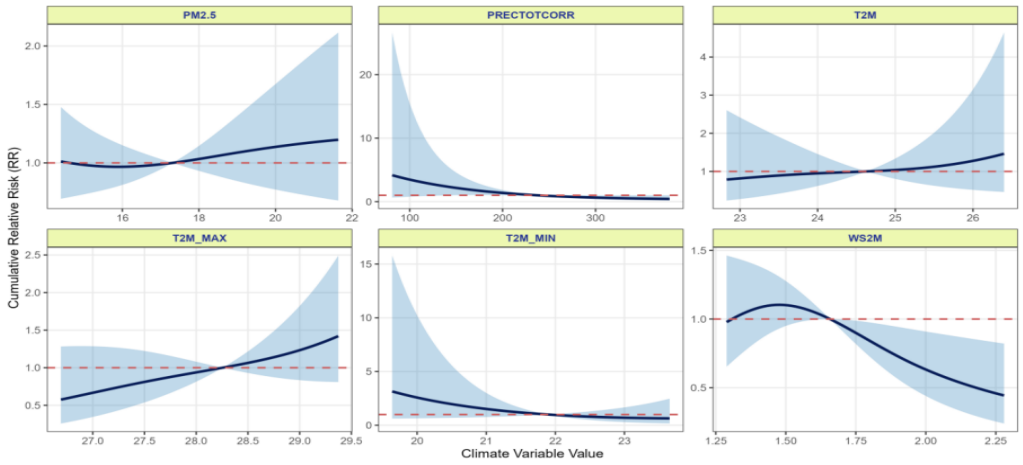


Figure 4. Overall cumulative exposure response curves

The cumulative exposure response curves (Figure 4) indicate that mean, maximum, and minimum air temperatures as well as PM2.5 exhibited nonlinear but non-significant associations with childhood pneumonia, as their 95% confidence intervals consistently included the null value (RR = 1). Similar findings have been reported in previous studies, suggesting that the effects of these environmental factors vary across geographical settings and population characteristics [8], [9], [13]. In contrast, rainfall and wind speed showed significant cumulative associations, with higher exposure levels associated with lower cumulative relative risks of childhood pneumonia, highlighting the importance of applying the Distributed Lag Nonlinear Model (DLNM) to capture complex nonlinear environmental effects that may not be adequately represented by conventional linear models [5], [9], [10].

c. Lag Response Relationship

The lag response analysis was performed to evaluate how the effects of environmental exposures changed over time following exposure [5], [9], [10]. Relative risks were estimated at each lag month by comparing the 90th percentile of each environmental variable with its median value. The results are presented in Figure 5.

Figure 5 shows that rainfall and wind speed were the only environmental variables exhibiting statistically significant delayed associations with childhood pneumonia. Rainfall showed inverse associations from lag 8 to lag 12 months, with relative risks decreasing from 0.892 (95% CI: 0.806–0.987) to 0.828 (95% CI: 0.695–0.987), corresponding to estimated risks approximately 10.8% to 17.2% lower than the reference exposure. Similarly, wind speed showed inverse associations between lag 4 and lag 11 months, with relative risks ranging from 0.922 (95% CI: 0.852–0.997) to 0.880 (95% CI: 0.790–0.980), corresponding to estimated risks approximately 7.8% to 12.0% lower than the reference exposure. In contrast, mean, maximum,

and minimum air temperatures together with PM2.5 exhibited nonlinear but non significant lag response patterns, as their 95% confidence intervals consistently included the null value (RR = 1). These findings indicate that rainfall and wind speed were the environmental exposures with statistically significant delayed associations at the provincial level. However, because the study is observational, these associations should not be interpreted as evidence that changes in rainfall or wind speed directly caused changes in childhood pneumonia incidence [5], [9], [10].

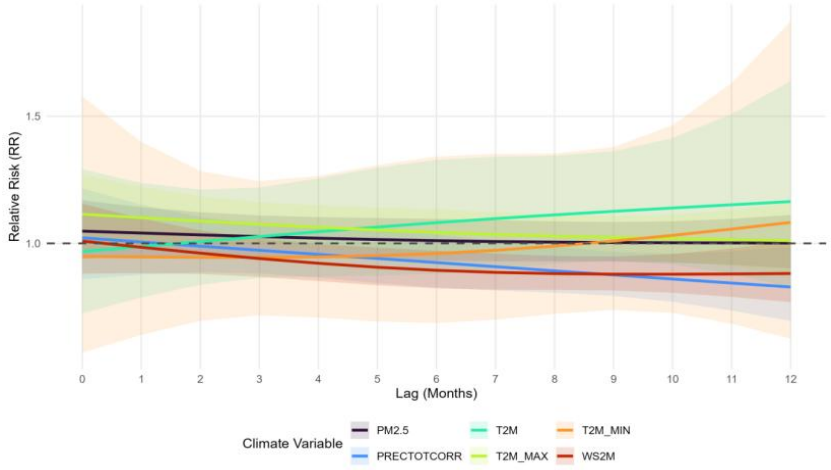


Figure 5. Cumulative lag response relationships between climatic variables and childhood pneumonia

d. Spatial Variation in Relative Risk Estimates

To evaluate spatial heterogeneity, district level DLNMs were fitted for each environmental variable. The spatial distribution of statistically significant relative risks is presented in Figure 6.

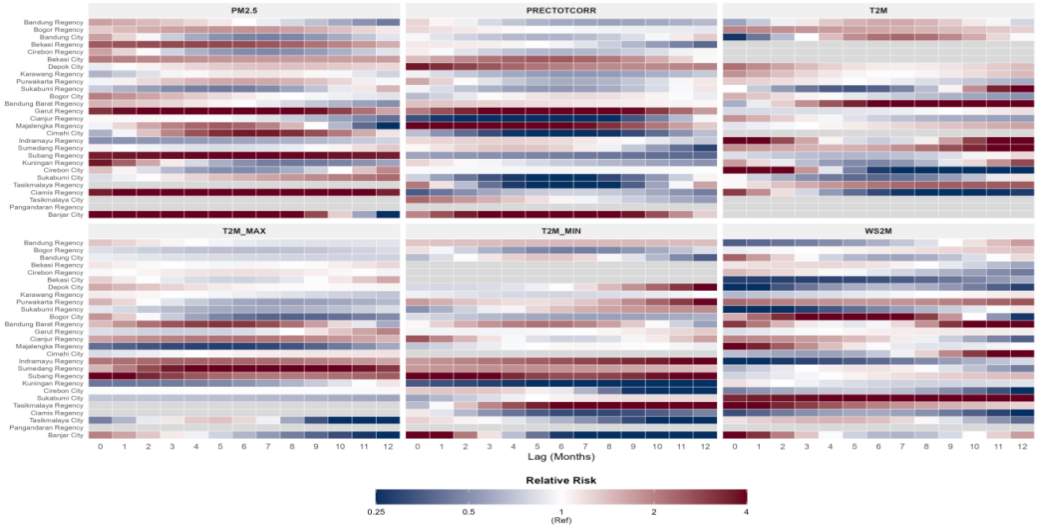


Figure 6. Spatial distribution of relative risks

Figure 6 demonstrates substantial spatial variation in the estimated associations between environmental factors and childhood pneumonia across West Java. Both positive and protective associations were observed depending on the district and environmental variable [10]. Grey cells

indicate districts where statistically reliable estimates could not be obtained because the fitted models did not satisfy the predefined stability criteria. The statistically significant district level relative risks are summarized in Table 3. Statistical significance within an individual district indicates that the estimated association in that district differs from the null value (RR = 1), but it does not by itself demonstrate that the association is statistically different from the corresponding estimate in another district. No formal statistical test of between district differences or spatial interaction was performed in this study; therefore, the district level results are interpreted as evidence of spatial variation in the estimated relative risks rather than formal statistical evidence of heterogeneity between districts.

Table 3. Statistically significant district level relative risks at lag 0

District/Municipality	Climatic Variable	RR (95% CI)
Bandung Regency	Wind speed (WS2M)	0.36 (0.15–0.87)
Bekasi Regency	PM2.5	2.54 (1.21–5.33)
Bogor Regency	Mean temperature (T2M)	1.99 (1.04–3.80)
Cianjur Regency	Rainfall (PRECTOTCORR)	0.32 (0.12–0.82)
Indramayu Regency	PM2.5	0.50 (0.26–0.98)
Sumedang Regency	Minimum temperature (T2M_MIN)	1.77 (1.10–2.85)
Sukabumi Regency	Wind speed (WS2M)	0.16 (0.05–0.59)
Tasikmalaya Regency	Wind speed (WS2M)	4.72 (1.27–17.56)
Bandung City	Mean temperature (T2M)	0.26 (0.08–0.81)
Bandung City	Wind speed (WS2M)	2.40 (1.09–5.32)
Bekasi City	PM2.5	2.09 (1.37–3.20)
Bekasi City	Wind speed (WS2M)	0.28 (0.13–0.62)
Bogor City	Maximum temperature (T2M_MAX)	1.80 (1.05–3.07)
Bogor City	PM2.5	2.05 (1.18–3.55)
Depok City	Mean temperature (T2M)	2.23 (1.06–4.67)
Depok City	Rainfall (PRECTOTCORR)	3.70 (2.22–6.16)
Depok City	Wind speed (WS2M)	0.18 (0.06–0.60)

As shown in Table 3.2, wind speed exhibited a wide range of estimated relative risks, from RR = 0.16 (95% CI: 0.05–0.59) in Sukabumi Regency to RR = 4.72 (95% CI: 1.27–17.56) in Tasikmalaya Regency. Relative to the median exposure, these estimates correspond to approximately 84% lower and 372% higher estimated risks, respectively. $PM_{2.5}$ also demonstrated marked spatial variation, with RR = 2.54 (95% CI: 1.21–5.33) in Bekasi Regency and RR = 2.09 (95% CI: 1.37–3.20) in Bekasi City, corresponding to approximately 154% and 109% higher estimated risks relative to the median exposure, respectively. In contrast, Indramayu Regency showed an inverse association with RR = 0.50 (95% CI: 0.26–0.98), corresponding to an estimated 50% lower risk relative to the reference exposure, whereas temperature variables and rainfall were significant only in a few districts. Overall, these findings demonstrate

substantial spatial heterogeneity in the associations between environmental factors and childhood pneumonia, with several variables becoming significant only at the district level, indicating that province wide estimates may mask important local patterns [4], [7], [8].

Discussion

The present study found that the associations between environmental factors and childhood pneumonia in West Java were nonlinear and delayed, with variation in the estimated relative risks across districts and municipalities [4], [5], [13]. At the provincial level, rainfall and wind speed exhibited significant cumulative and lagged associations, whereas temperature variables and PM2.5 showed nonlinear but non significant cumulative associations. However, district level analyses revealed that several environmental variables became significant in specific locations, indicating that province wide estimates may mask important local environmental patterns [4], [10].

Several mechanisms may plausibly explain the observed associations, although these mechanisms cannot be established causally from the present observational analysis. Rainfall may be associated with respiratory disease through changes in humidity, indoor and outdoor environmental conditions, and pathogen survival or transmission, while prolonged or variable rainfall may also influence human mobility and time spent indoors. Wind speed may affect the dispersion and dilution of airborne particulate matter and other environmental pollutants, potentially altering population exposure to respiratory irritants. These mechanisms may also operate with delays because changes in environmental conditions can influence exposure patterns and respiratory health over subsequent weeks or months. However, the direction and magnitude of these associations may depend on local climatic, demographic, and environmental conditions.

The contrasting findings between the provincial and district analyses highlight the importance of considering spatial variation in environmental epidemiology [4], [10]. The observed differences in district-specific estimates should be interpreted as spatial variation in the estimated associations rather than formal evidence of statistical heterogeneity between districts. Because no formal statistical test comparing associations across districts was performed, differences in the significance or magnitude of district-specific relative risks do not by themselves demonstrate that the associations are statistically different between regions. Specifically, daily rainfall patterns in West Java regions, such as Bandung Regency, have been shown to exhibit high temporal volatility and heteroskedasticity, causing rapid shifts in local environmental conditions [14]. Differences in population density, urbanization, local air quality, healthcare access, and other unmeasured environmental or socioeconomic conditions may also contribute to the observed variation in relative risk estimates across districts. Similar observations have been reported in previous studies, suggesting that geographically aggregated analyses may underestimate localized environmental risks [4], [10].

Several mechanisms may plausibly explain the delayed associations observed for rainfall and wind speed. Rainfall may alter humidity and environmental conditions that influence respiratory pathogen transmission and human exposure patterns. Its association with childhood pneumonia may therefore emerge with a delay rather than during the same month as the exposure. Wind speed may affect the dispersion and dilution of airborne particles and other

environmental pollutants, potentially modifying population exposure to respiratory irritants. Changes in these environmental conditions may also interact with local patterns of indoor and outdoor activity, which could contribute to delayed associations with respiratory outcomes. However, these mechanisms are only plausible explanations and cannot be established as causal pathways from the present observational analysis [4], [10].

Among the evaluated environmental variables, rainfall and wind speed consistently exhibited delayed inverse associations. These findings suggest that the associations between environmental conditions and childhood pneumonia may extend over several months rather than being restricted to the month of exposure (Polrob & La-up, 2025). The delayed associations identified in this study are consistent with previous DLNM based studies reporting that climatic factors may be associated with respiratory diseases through complex temporal pathways rather than immediate responses [10]. The delayed effects identified in this study are consistent with previous DLNM based studies reporting that climatic factors influence respiratory diseases through complex temporal pathways rather than immediate responses [5], [9], [15].

The variation in estimated relative risks across districts may reflect differences in local climatic conditions, population characteristics, urbanization, air quality, healthcare access, and other environmental or socioeconomic factors that were not fully captured in the models. Differences in exposure distributions and population level vulnerability may also contribute to the variation in the estimated associations. These possibilities should be interpreted cautiously because the present analysis does not formally identify which factors account for the observed between district variation.

Several limitations should be considered when interpreting these findings. First, the analyses were based on aggregated district level data and therefore describe population level associations rather than individual risks [10]. Second, several potentially important determinants of childhood pneumonia, including socioeconomic conditions, nutritional status, vaccination coverage, and household environmental factors, were not included in the models (Ciptaningtyas et al., 2026; Kumar & Shally, 2023; Mcallister et al., 2019). This may result in residual confounding, particularly because the exposure specific models did not mutually adjust for other correlated environmental variables. Third, environmental exposures derived from gridded or area level data may not fully represent individual or within district exposure, introducing potential exposure measurement error. Fourth, multiple district level exposure analyses increase the number of statistical comparisons, so some statistically significant findings may have occurred by chance, and the reported confidence intervals should therefore be interpreted cautiously. Finally, several district level models did not satisfy the predefined stability criteria and were excluded from interpretation, limiting the completeness of the spatial analyses and potentially affecting the comparability of estimates across districts.

Overall, the findings suggest that associations between environmental conditions and childhood pneumonia vary across time and geographical areas. Integrating provincial and district-level analyses using the DLNM framework provides a more detailed description of these associations and may support geographically targeted public health surveillance and planning in heterogeneous tropical regions [4], [9]. Integrating provincial and district level analyses using the DLNM framework provides a more comprehensive understanding of these complex

associations and may support geographically targeted public health interventions in heterogeneous tropical regions [5], [10]

4. Conclusion

This study applied the Distributed Lag Nonlinear Model (DLNM) to analyze the nonlinear and lagged associations between climatic factors, $PM_{2.5}$ concentration, and childhood pneumonia in West Java, Indonesia. At the provincial level, rainfall and wind speed showed significant cumulative and lagged associations with childhood pneumonia, whereas mean, maximum, and minimum air temperature and $PM_{2.5}$ concentration showed nonlinear but non-significant cumulative associations. The lag response analysis further indicated that rainfall showed significant associations from lag 8 to lag 12 months, while wind speed showed significant associations from lag 4 to lag 11 months. In contrast, temperature variables and $PM_{2.5}$ concentration did not show statistically significant lagged associations. District- and municipality level analyses revealed variation in estimated relative risks across administrative regions, indicating that the associations between environmental factors and childhood pneumonia differed across regions. Overall, the findings indicate that associations between environmental conditions and childhood pneumonia vary across time and geographical areas. These findings may inform public health surveillance and future geographically targeted intervention planning, while the observational and aggregated study design does not allow causal intervention effects to be established.

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