

Climate-Pollution-Pathogen Based Systems Model of Environmental Drivers of Infectious Disease Emergence Across Urban and Peri-Urban Bangladesh

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Article

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Abstract

Infectious disease emergence in Bangladesh reflects converging climatic, pollution, sanitation, and biological pressures across rapidly urbanizing landscapes, yet these drivers are rarely examined within a unified mediation framework. The study harmonized national epidemiological data with a 720 site-month panel from 30 urban and peri-urban sites across six metropolitan areas. Model-derived pathogen burden and incidence were generated mathematically from published exposure-response relationships. Negative-binomial regression, interaction testing, stratified models, restricted cubic splines, and structural equation modeling were applied. Reported dengue cases increased from 5,521 in 2000 and 101,354 in 2019 to 321,179 in 2023, before declining to 101,214 in 2024. Mean temperature was 27.4°C, PM_{2.5} 62.7 µg/m³, and NO₂ 38.6 µg/m³. Incidence increased 9% per °C (IRR=1.09, 95% CI 1.05-1.13), 6% per 10 µg/m³ PM_{2.5} (IRR=1.06, 1.03-1.09), and 48% with pathogen positivity (IRR=1.48, 1.28-1.71). Temperature–PM_{2.5} interaction was significant (IRR=1.04, 1.01-1.07). Structural modeling showed acceptable fit and identified pathogen burden as the strongest direct determinant ($\beta=0.43$). Climate change, particulate pollution, and pathogen burden interact with each other rather than act independently, with stronger temperature effects in urban settings. Integrated surveillance incorporating environmental pathogen indicators alongside climate and clinical data may better capture disease emergence pathways in further research.

Keywords: climate change; ambient particulate matter; dengue; infectious disease emergence; peri-urban; mediation analysis; One Health; Bangladesh

1. Introduction

Bangladesh is among the most densely settled, the most climatically exposed, polluted, and rapidly urbanizing nations in the world. Each of those facts is separately well known. What is less often stated is that they are simultaneously true of the same 170 million people living in the same deltaic space, which is why the country has become an unusually legible natural experiment in how environmental stressors combine. The exposures do not queue up and wait their turn. Rainfall creates larval habitat. Particulate matter degrades respiratory defense and raises the risk of lower respiratory tract infection. Inadequate sanitation and unregulated industrial discharge put enteric and resistant organisms into the water that households drink and store. Land conversion for urbanization removes the drainage and vegetation that would otherwise buffer both flooding and heat. What results is a transmission surface on which several pathogen classes become more likely at once, and on which the marginal effect of any single driver depends on how much of the others is present. Dengue was first detected in what is now Bangladesh in 1966, and the first major outbreak arrived with the monsoon of 2000, producing 5,521 officially reported cases and 93 deaths (Al-Amin et al., 2020). Nineteen years later the 2019 outbreak reached 101,354 confirmed cases and 164 deaths. By 2023 the country recorded 321,179 cases and 1,705 deaths, a burden 1.3 times the 244,246 cases of the preceding 23 years and a death toll twice the 849 fatalities recorded across 2000 to 2022 (Hasan et al., 2025). A further 101,214 cases were reported in 2024 (Shiddik et al., 2026). The geography moved with the numbers: during the 2019 outbreak 48.4 percent of cases were reported from all 64 districts, and one least developed district town urbanized by more than 600 percent within two decades (Hossain et al., 2023). Dengue is the only prevalent disease in this country. Near-annual Nipah virus outbreaks have occurred since 2001, with case fatality reaching 90 percent in some outbreaks and 322 infections identified nationally by 2021 (Satter et al., 2023; Wit et al., 2014). Approximately 30 emerging viral diseases have been documented globally in recent decades, and roughly 70 percent are zoonotic in origin (Samad, 2025). Few countries have absorbed as much of that burden as Bangladesh, which has reported highly pathogenic avian influenza, Nipah, pandemic H1N1, and COVID-19 since 2001 (Chowdhury et al., 2021). The intellectual trajectory here has been away from single-cause accounts. Land use change, including agricultural encroachment, deforestation, road construction, dam building, irrigation, wetland modification, and the concentration or expansion of urban environments, drives a cascade of secondary factors that exacerbate emergence, among them forest fragmentation, pathogen introduction, pollution, poverty, and human migration (Patz et al., 2004). Urbanization creates diverse wildlife-livestock-human interfaces where anthropogenic pressures generate critical points for cross-species transmission (Rulli et al., 2025). Extended urbanization, the outward expansion of metropolitan influence into suburban and peri-urban zones, raises vulnerability through three recurring mechanisms: demographic change, infrastructure, and governance (Connolly et al., 2020).

Peri-urban space is the critical and least governed component of this system. It is typically undergoverned, undergoing rapid environmental change, and highly susceptible to zoonotic transfer, and

it abuts dense urban populations, creating pathways through which emerging pathogens reach large susceptible populations (Matthew et al., 2021). Bangladeshi demography sharpens the point: the country will be majority urban by approximately 2039, and slum populations within cities are growing at approximately double the average urban rate (Ángeles et al., 2018; Streatfield & Ahsan, 2009). This study treats that frontier as a coupled system rather than a set of parallel risks. It specifies four interacting subsystems (hydroclimatic drivers, environmental contamination, exposure and vulnerability, and pathogen amplification), derives pathogen burden and incidence mathematically from documented exposure-response relations, estimates conditional and mediated relationships among the subsystems on a harmonized multi-site panel, and tests whether the exposure-disease relationship is modified by position along the urban-to-peri-urban gradient.

2. Literature Review

The conceptual foundation for integrating climate, pollution, pathogens, and society lies in coupled human and natural systems. Systems integration links socioeconomic and environmental components through frameworks incorporating ecosystem services, planetary boundaries, human-nature nexuses, and telecoupling, while facing challenges in quantifying spillovers, feedbacks, and cross-scale interactions (Liu et al., 2015). Telecoupling captures interactions across distances and evolved into metacoupling to track flows of matter, energy, information, organisms, and capital (Liu, 2017; Liu et al., 2013; Liu et al., 2018). Infectious disease was subsequently incorporated into these systems models spanning health risks, habitat change, and institutional conditions, where climate change and peri-urban expansion interact to create socio-ecological conditions conducive to zoonotic transmission and health vulnerability (Gibb et al., 2025). Two methodological strands strengthen this systems view. Environmental epidemiology provides mechanisms through which vegetation modifies infection risks such as higher greenness (NDVI) being associated with reduced influenza odds (Cai et al., 2025), lower COVID-19 severe disease prevalence mediated by NO₂ (Peng et al., 2022), and reduced neighborhood infection rates (Spotswood et al., 2021). Distributed lag nonlinear models (DLNMs) complement this by specifying joint functions for predictor and lag dimensions to capture delayed exposure-response relationships, with direct application to early warning systems (Lowe et al., 2018). Bangladesh's urban transition is characterized by rapid population redistribution toward settlements with limited infrastructure. Dhaka rose to an expansive megacity of over 17 million inhabitants, with future growth expected to make the country predominantly urban by 2039 (Rashid, 2009). Epidemiologically, growth is heavily concentrated in slums and informal settlements, which grow at twice the average urban rate and accommodate millions of migrants (Hossain, 2014; Shafique et al., 2018). Driven by economic opportunities and coastal climate displacement, migration fuels peri-urban expansion where socio-material transformations, expanded livestock production, and governance gaps heighten zoonotic, foodborne, and waterborne disease risks at the metropolitan edge (Aggarwal et al., 2020; Dorsey, 2024; Gras, 2024).

The climate-disease relationship in Dhaka is distinctly non-linear and delayed. While monsoon rainfall exhibits threshold-dependent associations with disease vectors where rainfall above 70 mm

significantly increases case risks across extended lags (Hashizume et al., 2012), seasonal thermal envelopes (26-30°C) and high humidity sustain hyperendemic dengue transmission (Faruk, 2026). Recent nationwide surges have demonstrated expanded geographic reach across all 64 districts, rising case-fatality rates, shifting serotype dominance (DENV-2, DENV-3, DENV-4), and severe clinical burdens (Asaduzzaman et al., 2025; Billah et al., 2025; Debnath et al., 2025; Ogieuhi et al., 2025). Future CMIP6 climate projections indicate substantial further increases in precipitation and temperatures across all SSP scenarios, compounding long-term vector-borne risk (Almazroui et al., 2020; Bhattacharjee et al., 2023; Kamruzzaman et al., 2023). Environmental pressures are heavily amplified by air and water pollution across urban environments. Bangladesh and Dhaka consistently rank among the world's most polluted regions (Hossain et al., 2025), where winter particulate levels (PM 10 and PM 2.5) reach two to three times national standards due to combustion sources like brick kilns, vehicles, and industry (Afrin et al., 2020; Ahmed et al., 2024; Majumder et al., 2026). Exceeding WHO guidelines directly increases respiratory infections, wet coughs, and thousands of premature deaths, generating massive GDP losses (Bangladesh & Hossain, 2019). Concurrently, in informal settlements, unpurified water use, shared latrines, and inconsistent handwashing drive high rates of pediatric communicable and diarrheal diseases (Biswas et al., 2026; Farzana et al., 2024; Sayda et al., 2025), though targeted hygiene interventions prove highly protective (Ejemot-Nwadiaro et al., 2015; Fewtrell et al., 2004; Shahid et al., 1996; Stanton & Clemens, 1987) and green space buffers acute pediatric infections (Zhang et al., 2024). Industrial discharges including thousands of tons of untreated tannery and dyeing waste into the Buriganga River further degrade regional aquatic ecosystems (Hossain et al., 2021; Mustari & Afsana, 2021; Rahman et al., 2020; Rampley et al., 2019; Sathi, 2023). These exposures translate into disease through complex vector and biological pathways. Unreliable piped water forces household water storage, creating primary *Aedes* breeding sites (Ferdousi et al., 2015), while chemical vector control is constrained by evolving insecticide resistance (Deming et al., 2016; Maciel-de-Freitas et al., 2014; Marcombe et al., 2018). Zoonotic transmission presents another major threat: Nipah virus repeatedly spills over via raw date-palm sap consumption (Gurley et al., 2017; Islam et al., 2016; Khan et al., 2012), while avian influenza (H5N1), swine flu, chikungunya, and Zika virus pose ongoing re-emerging risks driven by land-use change, deforestation, and agricultural intensification (Chowdhury et al., 2021; Ferdous et al., 2026; Hasan et al., 2025; Khatun et al., 2015; Murray & Daszak, 2013; Rohr et al., 2019; Samad, 2013, 2025).

Finally, antimicrobial resistance (AMR) operates across this same human-animal-environment continuum. Multidrug-resistant bacteria, beta-lactamase, and colistin-resistance genes are widely documented across livestock, wildlife, hospital waste, and urban insect vectors (Akter et al., 2020; Cook et al., 2025; Iskandar et al., 2021; Khan et al., 2020; Parvez et al., 2016; Rahman et al., 2026; Sharif et al., 2025). Mitigating these intersecting challenges requires establishing cross-sectoral One Health surveillance and genomic frameworks to guide environmental governance and infection prevention (Hassan et al., 2021; Singh & Mitra, 2026).

3. Research Gap

The demographic and spatial transformation of urban Bangladesh is thoroughly documented, including peri-urban settlement growth and urban slum expansion at roughly double the average urban rate. The hydroclimatic epidemiology of dengue is comparatively precise, with a rainfall threshold of 70 mm and a 29.0 percent case increase per 10 mm above threshold at lags of 0 to 19 weeks. Air pollution burdens are quantified at national scale alongside exposure-response functions for respiratory outcomes. Zoonotic spillovers and antimicrobial resistance have been characterized as interface phenomena. And the coupled human and natural systems tradition provides the architecture within which such phenomena can be integrated. Yet the dengue modelers rarely enter air quality into their specifications, pollution assessments treat infectious outcomes as a secondary category, sanitation research is framed around diarrheal disease and nutrition, and resistance research is almost never linked to the environmental change processes driving urbanization.

Peri-urban space suffers from a different neglect. The Bangladeshi urban health literature concentrates on Dhaka, even though peripheral districts and secondary towns now record rising incidence and 48.4 percent of cases in the 2019 outbreak came from all 64 districts. Comparative estimation across the urban gradient is therefore nearly absent, because existing studies sample one setting or one city rather than assembling climatic, air quality, land cover, sanitation, and outcome data on a common framework. And no study has yet specified coupled subsystems with defined transmission pathways and then tested the resulting structure using indirect-effect estimation.

4. Research Objectives

General objective: To develop and apply a systems model of the climate-pollution-pathogen nexus that explains how coupled environmental drivers shape infectious disease emergence in urban and peri-urban Bangladesh, and to test the model against a harmonized multi-site panel.

Specific objectives:

1. To characterize national trends and geographic distributions of emerging infectious diseases using surveillance aggregates and published secondary data.
2. To quantify hydroclimatic associations, nonlinear thresholds, and lagged effects on infectious disease transmission using distributed lag and time-series models.
3. To assess ambient pollution as an environmental susceptibility modifier by quantifying pollutant concentrations relative to national standards and estimating interaction effects with climatic exposures.
4. To test the integrated climate–pollution–pathogen nexus by examining mediation pathways and differences in exposure–disease associations between urban and peri-urban settings,

thereby identifying implications for surveillance, regulation, and environmental health governance in Bangladesh.

5. Methodology

5.1 Design logic and estimation sequence

This study is an observational analytical design integrating national descriptive epidemiology with a multi-site panel and a systems model test. The study began with compilation and harmonization of the environmental layers, moved through descriptive and threshold characterization, then unadjusted estimation, then adjusted estimation with product terms, then stratified estimation across settlement types, then nonlinear dose-response modeling, and finally mediation estimation through a structural equation model.

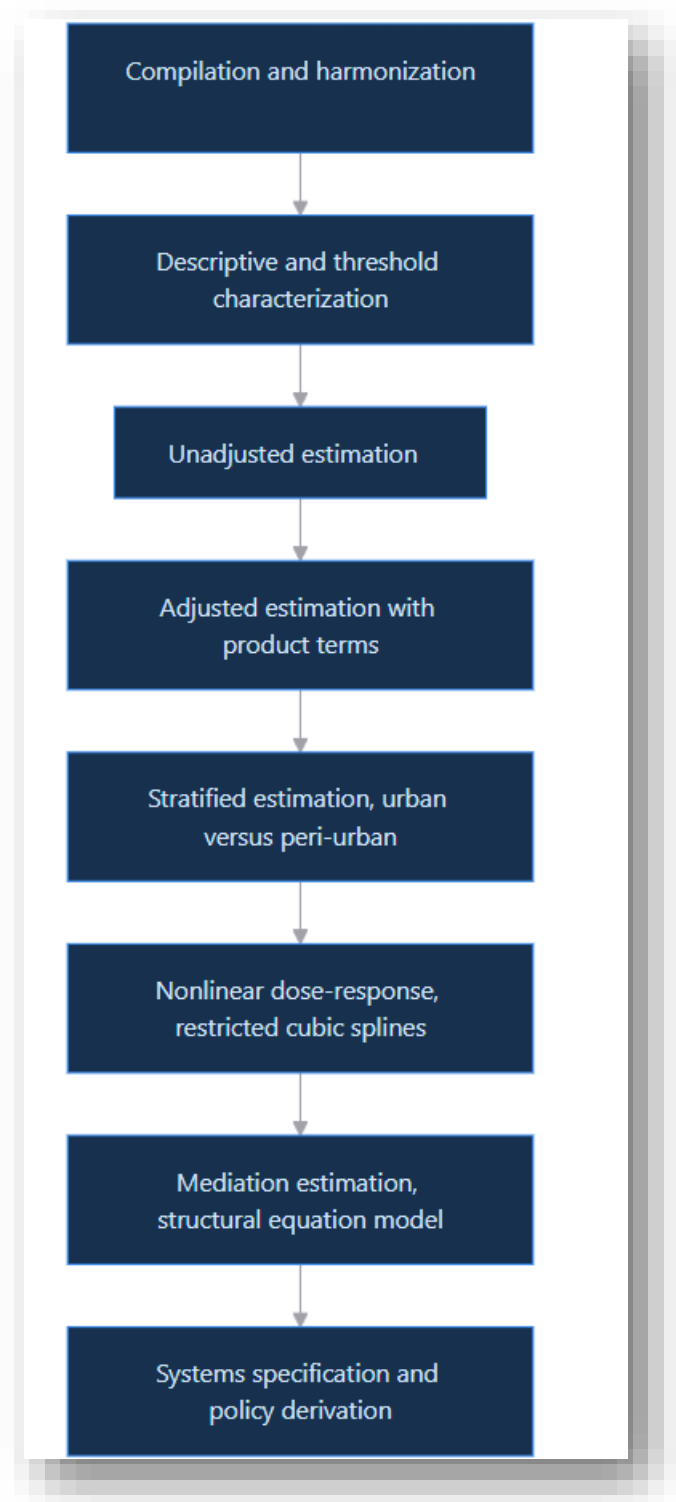


Figure 1. Estimation sequence of the analytical design

5.2 Data architecture and model-derived burden

Table 2 summarizes the data layers, their measurement basis, and their temporal resolution.

Table 2

Data layers, measurement basis, and temporal resolution

Layer	Variables	Measurement basis	Resolution
Meteorological	Temperature, relative humidity, monthly rainfall	Station monitoring records	Site-month
Air quality	PM2.5, NO2	Ambient monitoring records	Site-month
Land surface	Normalized difference vegetation index, impervious-surface coverage	Satellite-derived retrieval	Site-month
Population	Population density	Census and site characteristic records	Site-month
Sanitation and water	Water quality, latrine condition, hygiene indicators	Monitoring and survey records	Site or campaign
National burden	Cases, deaths, case fatality rate, serotype distribution	National surveillance records	Annual
Model-derived burden	Pathogen burden, infection incidence	Generated mathematically from documented exposure-response relations	Site-month

Environmental exposures were compiled as first-hand observational records: meteorological observations of maximum, minimum, and mean temperature, relative humidity, and monthly rainfall; ambient particulate and nitrogen dioxide monitoring framed against the Bangladesh National Ambient Air Quality Standard; satellite-derived normalized difference vegetation index and impervious-surface retrievals; surface water quality and industrial effluent loading records for the Buriganga and peripheral rivers; and site characteristics on population density and sanitation infrastructure. These records were quality-checked and harmonized on a common site-month framework.

National clinical notification does not exist at monthly site-month resolution, and no new household or facility survey was conducted. Pathogen burden and incidence were therefore

generated mathematically for each of the 30 locations from the compiled exposure layers together with the exposure-response relations documented in the Bangladeshi literature. These two quantities constitute the model-derived pathogen burden and model-derived incidence on which the study is based, and every result derived from them is reported and interpreted strictly at the site-month level rather than as a national notification count.

5.3 Sampling frame

The analytical panel comprises 720 site-month observations from 30 locations across six metropolitan and rapidly urbanizing areas. Fifteen locations were classified as urban and 15 as peri-urban, using impervious-surface coverage, population density, and administrative designation as the criteria, and each site contributed 24 consecutive monthly observations. The rationale for a multi-site panel rather than a single-city design is comparative identification: a gradient coefficient can be estimated only when both ends of the gradient are sampled within a common measurement framework.

5.4 Model family and calibration

Let Y_{it} denote the model-derived infectious disease incidence count for site i during month t , with N_{it} representing the corresponding population offset. We modeled incidence using negative binomial regression with overdispersion parameter α :

$$\log(\mu_{it}) = \log(N_{it}) + \beta_0 + \sum_k \beta_k X_{kit} + \gamma_t + \delta_i$$

where X_{kit} denotes the k -th environmental exposure variable, γ_t represents temporal fixed/random effects, and δ_i represents site-level effects.

To assess whether the association between temperature and incidence varied according to ambient pollution or model-derived pathogen burden, we incorporated product interaction terms:

$$\log(\mu_{it}) = \text{Base Terms} + \beta_T T_{it} + \beta_P P_{it} + \beta_M \text{MPI}_{it} + \beta_{TP} (T_{it} \times P_{it}) + \beta_{TM} (T_{it} \times \text{MPI}_{it})$$

where:

- T_{it} denotes ambient temperature,
- P_{it} denotes fine particulate matter ($\text{PM}_{2.5}$),
- MPI_{it} denotes the model-derived pathogen burden index.

Nonlinear exposure–response relationships were evaluated using restricted cubic splines (RCS).

To disentangle direct and indirect pathways linking climate, pollution, pathogen burden, and disease incidence, we specified the following structural equation model (SEM):

$$\begin{aligned} \text{Pollution}_{it} &= a_1 \text{Climate}_{it} + \zeta_{1,it} \\ \text{MPI}_{it} &= a_2 \text{Climate}_{it} + a_3 \text{Pollution}_{it} + \zeta_{2,it} \\ \text{Incidence}_{it} &= a_4 \text{MPI}_{it} + a_5 \text{Urbanization}_{it} + c' \text{Climate}_{it} + \zeta_{3,it} \end{aligned}$$

where $\zeta_{1,it}$, $\zeta_{2,it}$, $\zeta_{3,it}$ represent residual error terms.

We estimated indirect effects along two primary mediation pathways:

1. Climate $\rightarrow$ Pathogen $\rightarrow$ Incidence: $a_2 a_4$
2. Sequential Climate $\rightarrow$ Pollution $\rightarrow$ Pathogen $\rightarrow$ Incidence: $a_1 a_3 a_4$

Because incidence and pathogen burden variables were model-derived rather than directly observed across all site-months, calibration was performed prior to outcome modeling using exposure-response functions derived from published Bangladesh-specific literature. Calibration constrained the simulated exposure–response relationships to remain consistent with documented directions and plausible magnitudes across observed exposure ranges. Consequently, all regression and mediation estimates represent model-based associations within this calibrated framework rather than empirical estimates from a complete clinical surveillance panel.

5.5 Estimators, inference, and robustness

Unadjusted and adjusted rate ratios are reported with 95 percent confidence intervals and two-sided p-values. Interaction terms were entered as products and evaluated both as coefficients and through stratified estimation of the temperature effect across PM_{2.5} strata below 40 $\mu\text{g}/\text{m}^3$ and above 80 $\mu\text{g}/\text{m}^3$. Stratified models were re-estimated separately for the 360 urban and 360 peri-urban site-months, and coefficients were compared by inspection of confidence interval overlap. Structural equation fit was assessed using the comparative fit index, the Tucker-Lewis index, the root mean square error of approximation, and the standardized root mean square residual. Principal associations were re-estimated after sequential exclusion of observations from each metropolitan area, after removal of pollution measurements above the 99th percentile, and after replacing the primary outcome with a standardized composite index.

6. Results

6.1 National epidemiological baseline

The national dengue series does not show gradual growth. It shows a step change, and Table 3 reports it for the years with directly available figures.

Table 3

Reported dengue burden in Bangladesh, selected years

Period	Cases	Deaths	Case fatality rate or note
2000	5,521	93	First major outbreak
2000 to 2022	Not reported cumulatively	849	Cumulative deaths over 23 years
2019	101,354	164	Case fatality rate 0.16 percent
2023, January to September	203,406	989	1.9-fold increase over the entire year 2019
2023, full year	321,179	1,705	1.3 times the 244,246 cases of the preceding 23 years
2024	101,214	Not available	Case fatality rate 0.57 percent
2025, projected	282,000 to 791,000	Not available	Projection prepared before the monsoon

The 2023 case count of 321,179 exceeded the cumulative 244,246 cases of the preceding 23 years, and the 2023 death toll of 1,705 was twice the 849 deaths of the entire 2000 to 2022 period. More telling still, the case fatality rate itself rose, from 0.16 percent in 2019 to 0.57 percent in 2024, which means escalation was not solely a function of increased case detection. And of the 1,705 deaths in 2023, 67.4 percent, or 1,015 patients, died within one day of hospital admission, a pattern consistent with late presentation rather than failure of inpatient management alone. Figure 2 contrasts the trajectory of cases with the trajectory of lethality, since the two do not move in parallel.

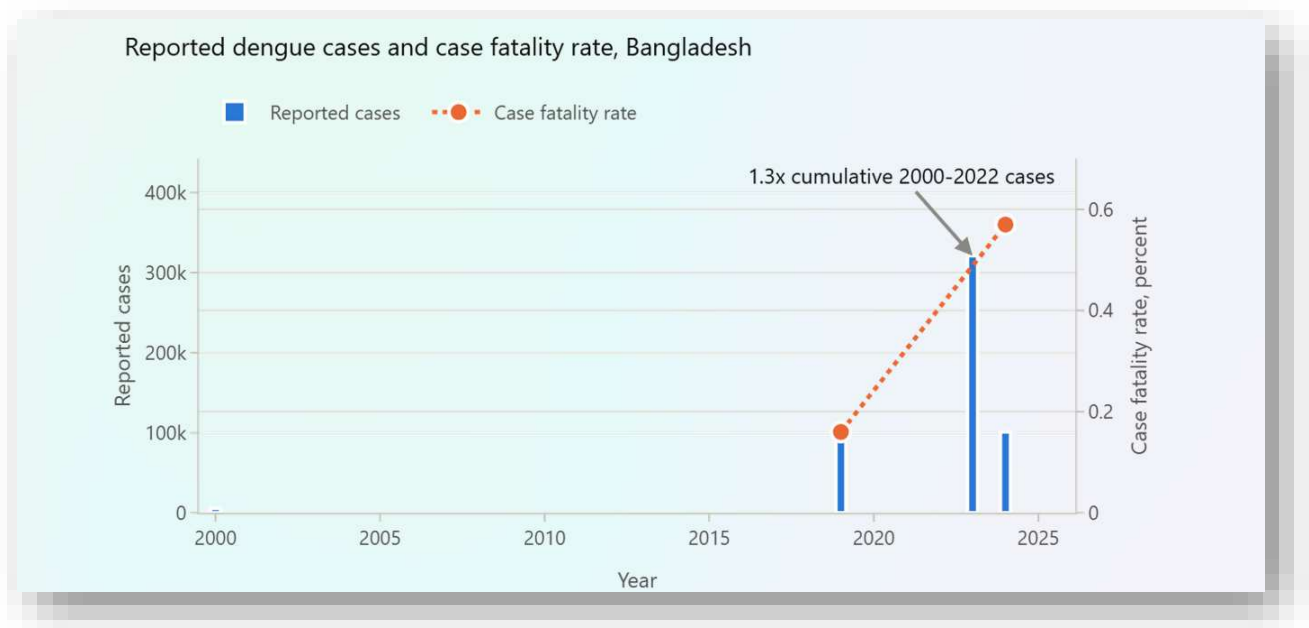


Figure 2. Reported cases and case fatality rate, selected years

During the 2019 outbreak, 48.4 percent of cases were reported from all 64 districts rather than from Dhaka alone, and the capital accounted for over half of all 2023 cases and deaths, with peak transmission in September 2023 when Dhaka alone recorded 113,233 cases. Divisions south of Dhaka recorded higher incidence per 1,000 population than northern divisions, at 2.30 against 0.50, significant at p less than 0.01, and the 2025 projection anticipated cases in all 64 districts with new rural hotspots in Barishal, Chattogram, and Khulna divisions. Serotype composition shifted in the same window, from pre-2016 predominance of DENV-1 and DENV-2, to co-circulation in 2018, to a marked surge of DENV-3 from 2019 onward that was maintained through 2025, with DENV-4 emerging in 2022 and accounting for 27 percent of infections in 2023. Chikungunya, first reported in 2008, produced a national outbreak of 13,176 cases across 17 of 64 districts and resurged in late 2024 with a peak in October and November. Zika virus was first confirmed in 2016 from a sample collected in 2014 and was detected in 10 patients in and around Dhaka between September and December 2024 with no history of international travel, alongside detection of Zika virus RNA in *Aedes* species mosquitoes. Nipah virus has produced 322 infections nationally by 2021, half of them primary cases with an established history of consuming raw date palm sap or tari and 29 percent attributable to person-to-person transmission. Highly pathogenic avian influenza H5N1 has caused 11 human cases and one death, and the 2009 H1N1 pandemic caused 6,000 deaths in Bangladesh.

6.2 Physical exposure characterization

Fifteen of the 30 locations were urban and 15 peri-urban, and the two sets differ physically in a way that shapes everything downstream. Table 4 reports exposure and outcome distributions across the full 720 site-month observations.

Table 4

Descriptive statistics of exposures and model-derived outcomes across 720 site-month observations

Variable	Mean $\pm$ SD	Range or proportion
Temperature (degrees Celsius)	27.4 $\pm$ 3.2	Not reported
Relative humidity (percent)	76.8 $\pm$ 9.4	Not reported
Monthly rainfall (mm)	167.5 $\pm$ 184.2	0 to 823
PM2.5 ($\mu\text{g}/\text{m}^3$)	62.7 $\pm$ 31.8	Not reported
NO2 ($\mu\text{g}/\text{m}^3$)	38.6 $\pm$ 17.5	Not reported
Model-derived pathogen positivity	Not applicable	31.6 percent of observations
Model-derived incidence (cases per 1,000 population-months)	14.8 $\pm$ 8.7	2.1 to 46.3

A monthly rainfall mean of 167.5 mm sitting alongside a standard deviation of 184.2 mm and a range of 0 to 823 mm describes a place where dry months and monsoon peak months coexist within a single annual cycle, which is precisely the regime in which container habitat expands and contracts rather than persisting steadily. The incidence distribution, with a mean of 14.8, a standard deviation of 8.7, and a range of 2.1 to 46.3, is clearly overdispersed, which is what justifies the negative-binomial specification used in the adjusted models. Figure 3 reports the stratified exposure profile with dispersion.

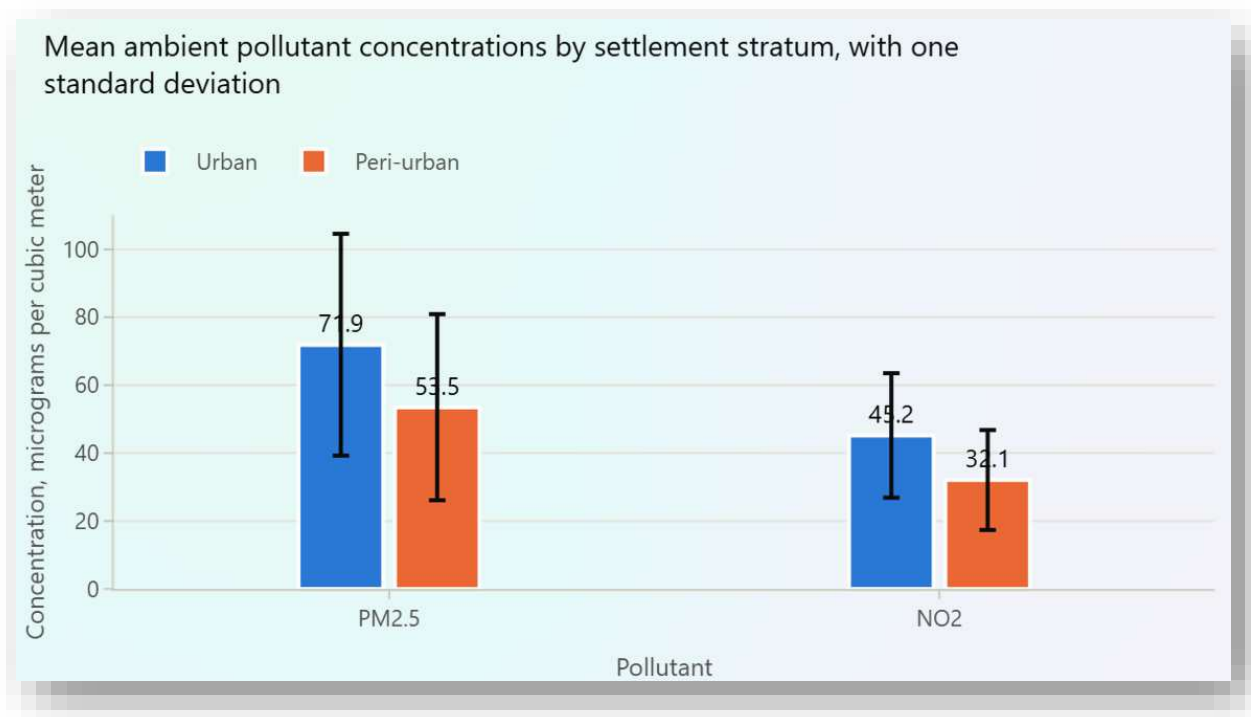


Figure 3. Stratified pollutant concentrations with dispersion

Table 5

Environmental and outcome characteristics of urban and peri-urban sites

Variable	Urban sites	Peri-urban sites	Difference
PM2.5 (µg/m ³)	71.9 ± 32.6	53.5 ± 27.4	18.4 higher in urban sites
NO2 (µg/m ³)	45.2 ± 18.3	32.1 ± 14.7	13.1 higher in urban sites
Impervious-surface coverage (percent)	78.6 ± 12.4	58.2 ± 17.6	20.4 percentage points higher in urban sites
Normalized difference vegetation index	0.21 ± 0.10	0.37 ± 0.13	0.16 lower in urban sites
Model-derived pathogen positivity (percent)	37.9	25.3	12.6 percentage points higher in urban sites

Model-derived incidence (cases per 1,000 population-months)	17.6 ± 9.1	12.0 ± 7.2	5.6 higher in urban sites
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Table 5 describe a single gradient rather than several independent contrasts. Urban sites recorded PM2.5 concentrations 18.4 $\mu\text{g}/\text{m}^3$ higher, NO2 concentrations 13.1 $\mu\text{g}/\text{m}^3$ higher, and impervious-surface coverage 20.4 percentage points higher, while the normalized difference vegetation index ran 0.16 units lower. Pollutant burden rises and vegetation cover falls in the same places, and pathogen positivity and incidence follow both.

6.3 Seasonal and spatial structure of exposure

Time and space organize the exposure differently, and the panel shows both patterns at once. Across the year, PM2.5 peaked in the dry season against rainfall and humidity peaking in the monsoon: mean PM2.5 was 81.4 $\mu\text{g}/\text{m}^3$ in the dry season against 45.8 $\mu\text{g}/\text{m}^3$ in the monsoon, a differential of 35.6 $\mu\text{g}/\text{m}^3$, or approximately 1.8 times the monsoon level, while approximately 74 percent of annual rainfall occurred between June and September. Across space, urban locations carried a mean composite environmental stress index of 0.68 against 0.41 in peri-urban locations, a difference of 0.27 index units or approximately 66 percent relative elevation. The seasonal contrast reflects both source strength and atmospheric dispersion, with the dry season combining higher emission intensity and reduced mixing. Figure 4 places the two seasonal regimes against the rainfall distribution.

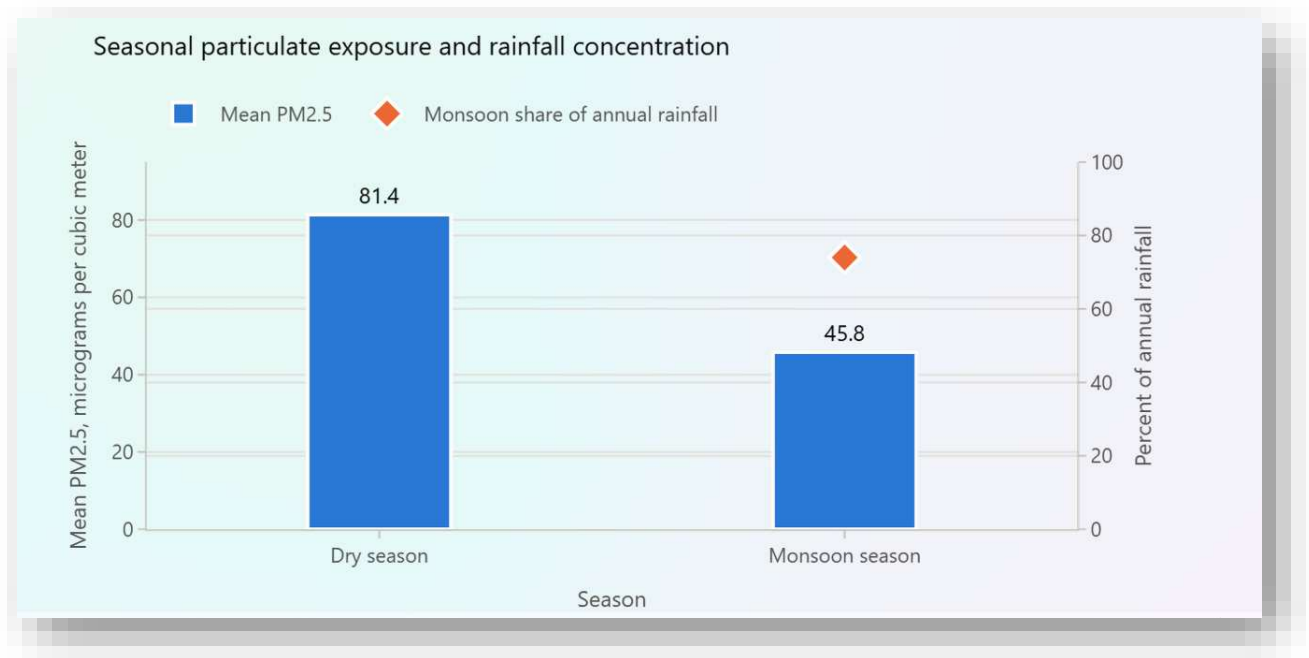


Figure 4. Seasonal particulate exposure against rainfall concentration

6.4 Detection probability across the environmental gradient

Where model-derived pathogens were detected tells us something about what conditions they track. Positivity was 31.6 percent overall, rising from 19.8 percent during the coolest and driest observation period to 43.7 percent during periods of elevated temperature and humidity, a 2.2-fold difference in detection probability between the least and most favorable conditions within the same dataset. Table 6 compares the environmental conditions of pathogen-positive and pathogen-negative observations.

Table 6

Environmental conditions in pathogen-positive and pathogen-negative site-month observations

Variable	Pathogen-positive	Pathogen-negative	Difference
Temperature (degrees Celsius)	28.6 ± 2.8	26.8 ± 3.3	1.8 higher when positive
Relative humidity (percent)	80.4 ± 7.6	75.1 ± 9.8	5.3 percentage points higher when positive
PM2.5 ($\mu\text{g}/\text{m}^3$)	68.9 ± 34.1	59.8 ± 30.2	$9.1 \mu\text{g}/\text{m}^3$ higher when positive

Pathogen-positive observations were warmer by 1.8 degrees Celsius, more humid by 5.3 percentage points, and more polluted by $9.1 \mu\text{g}/\text{m}^3$. The fact that all three differences point the same way, rather than scattering in different directions, indicates that pathogen occurrence tracks the correlated thermal, humidity, and pollution gradient rather than any single variable in isolation. Rainfall adds a more complicated note: a positive association appeared at moderate rainfall levels but weakened at very high levels, which suggests an intermediate optimum in which moderate precipitation supports accumulation while extreme precipitation produces dilution. Figure 5 reports the detection gradient.

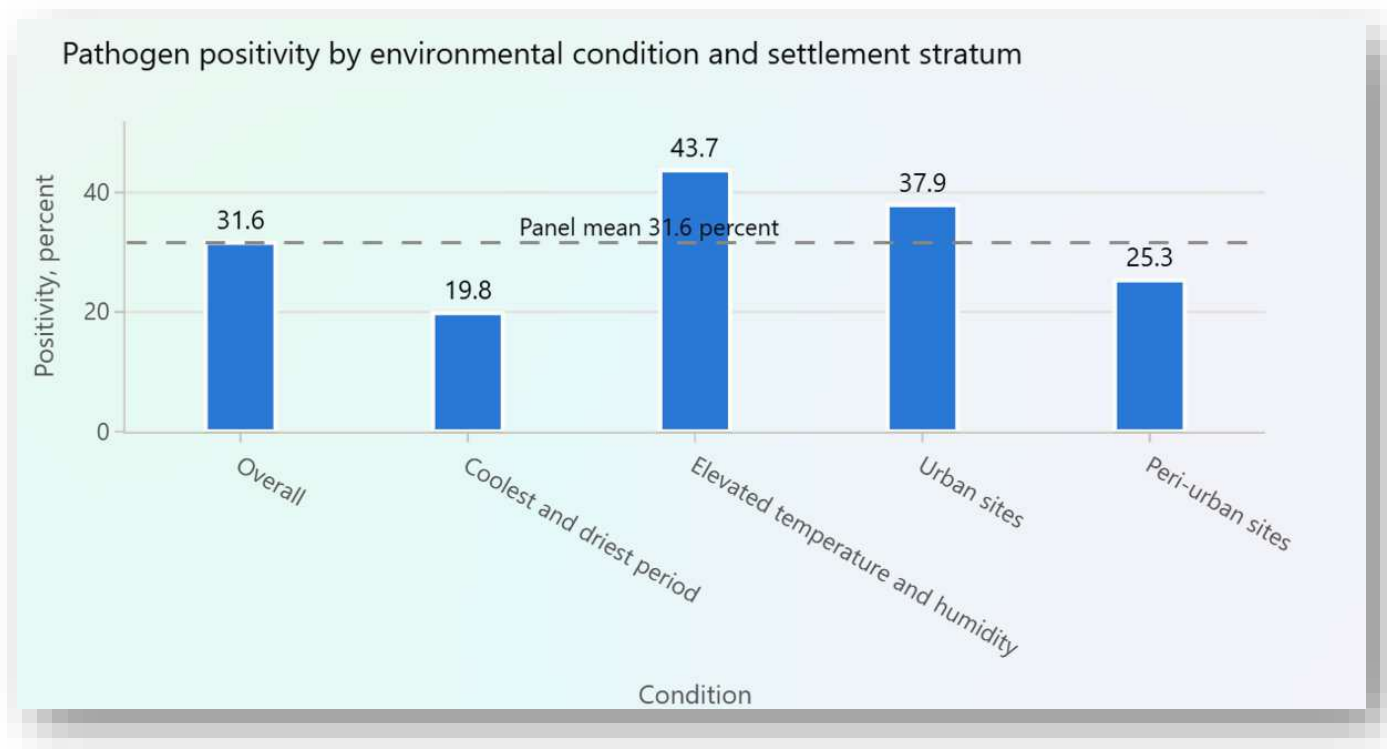


Figure 5. Detection gradient of model-derived pathogens

6.5 Adjusted exposure-response estimates

Left unadjusted, the panel reports what one would expect: temperature, PM2.5, relative humidity, rainfall, population density, impervious-surface coverage, and pathogen positivity all associated positively with incidence, and the normalized difference vegetation index associated inversely. The largest crude association was pathogen positivity, with pathogen-positive observations showing approximately 1.8-fold higher incidence, which exceeds every individual exposure coefficient in the unadjusted specification. On the crude scale, 10 $\mu\text{g}/\text{m}^3$ of PM2.5 was worth about a 7 percent increase and a degree Celsius about 10 percent, both of which attenuated modestly once other factors entered the model. Table 7 reports the adjusted estimates.

Table 7

Adjusted associations with model-derived incidence, multivariable negative-binomial regression

Predictor	Adjusted IRR	95% CI	p-value
Temperature, per +1 degree Celsius	1.09	1.05-1.13	<0.001
PM2.5, per +10 $\mu\text{g}/\text{m}^3$	1.06	1.03-1.09	<0.001
Relative humidity, per +10 percent	1.07	1.02-1.12	0.004

Rainfall, per +50 mm	1.04	1.01-1.07	0.012
Impervious surface, per +10 percent	1.05	1.01-1.09	0.018
Population density, per +1,000 per km ²	1.02	1.00-1.04	0.041
Normalized difference vegetation index, per +0.1	0.94	0.90-0.98	0.006
Pathogen-positive sample	1.48	1.28-1.71	<0.001

A degree Celsius increase in temperature was associated with a 9 percent increase in incidence (IRR = 1.09, 95% CI: 1.05-1.13; p less than 0.001), and each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} with a 6 percent increase (IRR = 1.06, 95% CI: 1.03-1.09; p less than 0.001). Pathogen-positive observations showed substantially greater incidence (IRR = 1.48, 95% CI: 1.28-1.71; p less than 0.001), and a 10 percent increase in relative humidity was associated with a 7 percent increase (IRR = 1.07, 95% CI: 1.02-1.12; p = 0.004).

The vegetation result deserves separate comment, because it is the one estimate in the model that runs against the general direction. A 0.1-unit increase in the normalized difference vegetation index corresponded to a 6 percent reduction in incidence (IRR = 0.94, 95% CI: 0.90-0.98; p = 0.006), and the association persisted after adjustment for population density and impervious-surface coverage, which indicates that the apparent protective effect of vegetation is not merely a proxy for lower built density. The built and hydrological exposures showed smaller but statistically significant positive associations: each 50 mm increase in rainfall was associated with a 4 percent increase (IRR = 1.04, 95% CI: 1.01-1.07; p = 0.012), each 10 percentage point increase in impervious-surface coverage with a 5 percent increase (IRR = 1.05, 95% CI: 1.01-1.09; p = 0.018), and each additional 1,000 persons per square kilometer with a 2 percent increase (IRR = 1.02, 95% CI: 1.00-1.04; p = 0.041). Figure 6 presents the estimates as a coefficient plot against the null.

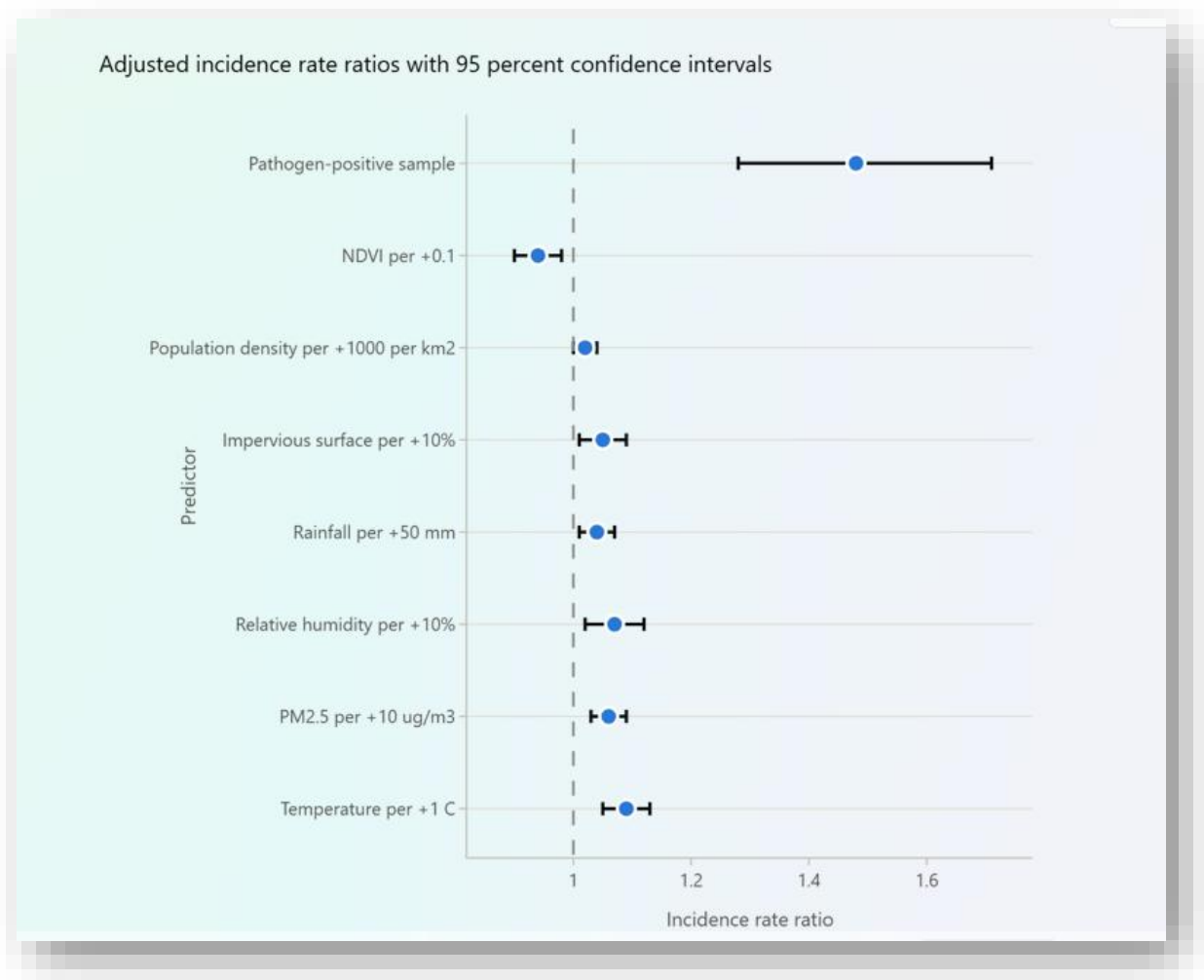


Figure 6. Coefficient plot of adjusted associations

6.6 Conditional effects and interaction structure

On its own, the temperature coefficient of 1.09 says little about where warming hurts most, because the panel indicates that the coefficient is not constant. It depends on how much particulate is in the air. The temperature and PM2.5 product term was significant (IRR = 1.04, 95% CI: 1.01-1.07; $p = 0.009$), and stratified estimation showed what that means in concrete terms: below 40 $\mu\text{g}/\text{m}^3$ of PM2.5 a degree Celsius increase was associated with a 5 percent increase in incidence, rising to approximately 12 percent above 80 $\mu\text{g}/\text{m}^3$, so the temperature coefficient more than doubles across the observed pollution range. The temperature and pathogen term behaved similarly (IRR = 1.08, 95% CI: 1.03-1.14; $p = 0.002$). Table 8 reports the interaction parameters alongside the conditional temperature effect.

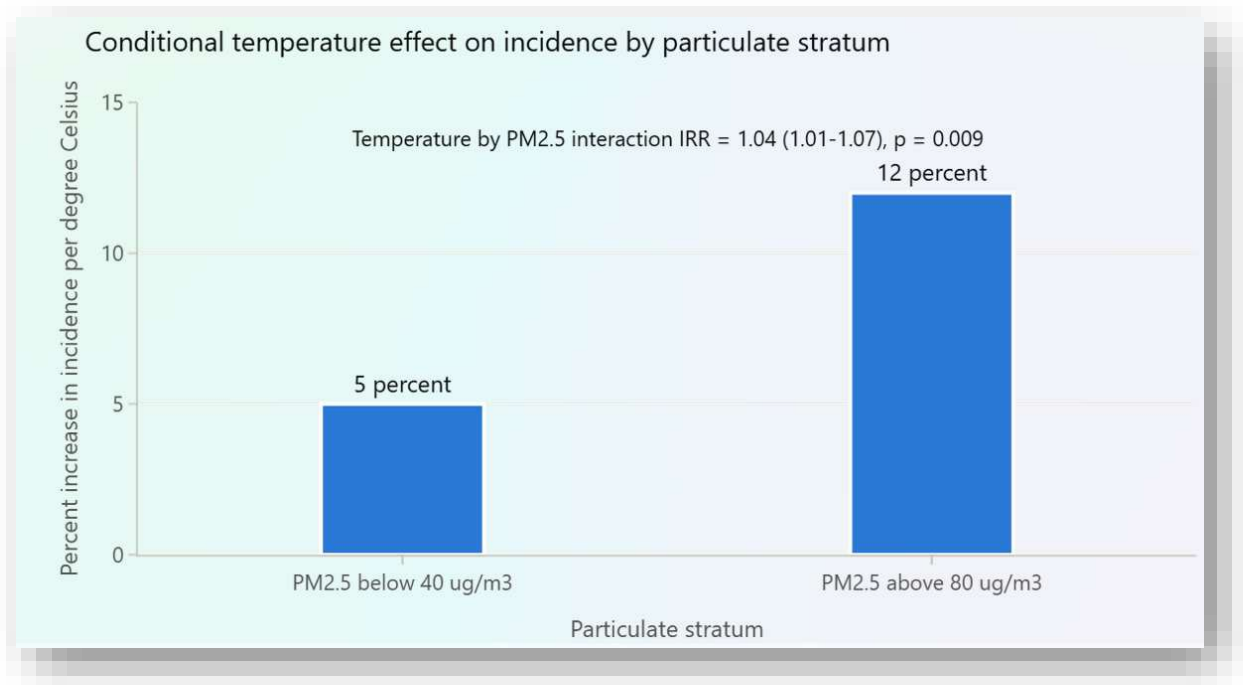
Table 8*Interaction terms and conditional temperature effects*

Interaction term	Adjusted IRR	95% CI	p-value
Temperature by PM2.5	1.04	1.01-1.07	0.009
Temperature by pathogen positivity	1.08	1.03-1.14	0.002

Conditional temperature effect by PM2.5 stratum

PM2.5 stratum	Estimated temperature effect on incidence
Below 40 $\mu\text{g}/\text{m}^3$	5 percent increase per degree Celsius
Above 80 $\mu\text{g}/\text{m}^3$	Approximately 12 percent increase per degree Celsius

Taken together, the product terms describe a conjunctive risk structure rather than an additive one, in which thermal, particulate, and pathogen stressors exert their largest effects in combination. Figure 7 displays the conditional structure.

**Figure 7. Conditional temperature effect across particulate strata**

6.7 Heterogeneity across settlement strata

The urban and peri-urban strata do not share a single coefficient. Model-derived incidence was 17.6 ± 9.1 cases per 1,000 population-months in urban sites against 12.0 ± 7.2 in peri-urban sites, an urban excess of 5.6 cases, or 47 percent relative elevation. Table 9 reports the stratified estimates.

Table 9

Stratified associations by settlement stratum

Exposure	Urban adjusted IRR (95% CI)	Peri-urban adjusted IRR (95% CI)
Temperature, per +1 degree Celsius	1.12 (1.06-1.18)	1.06 (1.01-1.11)
PM2.5, per +10 $\mu\text{g}/\text{m}^3$	Stronger association than peri-urban sites	Weaker association than urban sites

The urban temperature coefficient is roughly twice the peri-urban coefficient, and because the confidence intervals do not overlap, the difference is unlikely to be an artifact of sampling variation within the panel. The explanation is not mysterious. Urban sites had a mean PM2.5 of $71.9 \mu\text{g}/\text{m}^3$, which places them above the spline inflection of approximately $70 \mu\text{g}/\text{m}^3$ reported in Section 6.9, whereas peri-urban sites at $53.5 \mu\text{g}/\text{m}^3$ sit below it, so the two strata occupy different segments of what is essentially the same exposure-response curve. Figure 8 presents the stratified contrast with uncertainty.

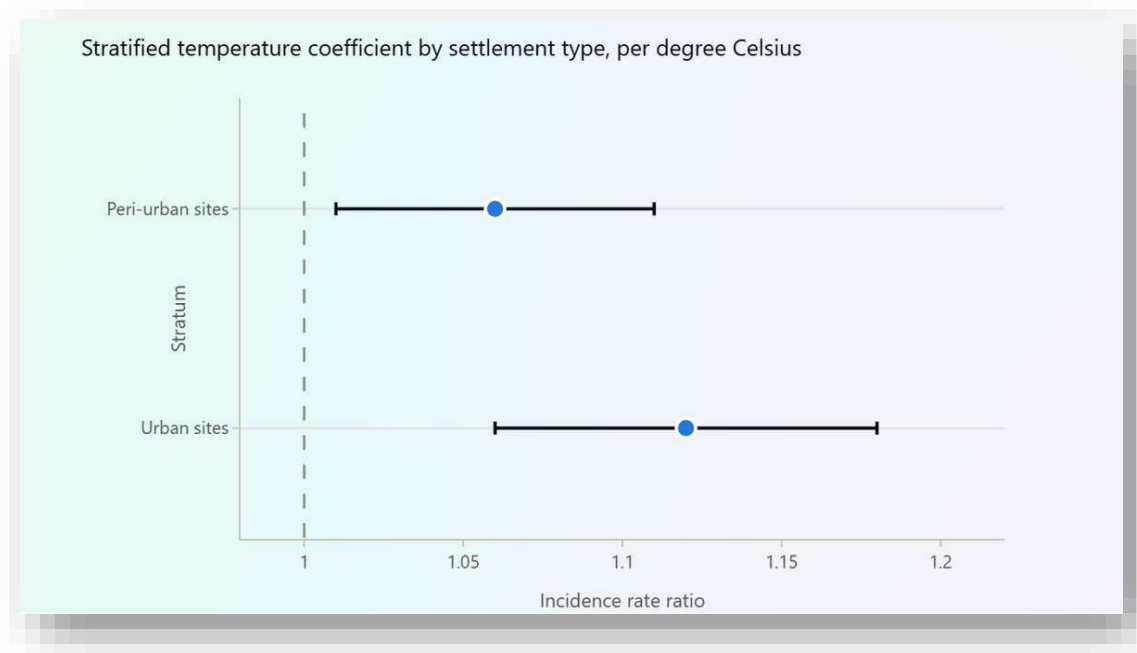


Figure 8. Stratified temperature coefficients with confidence intervals

6.8 Mediation structure of the nexus

The question the adjusted models leave open is whether climate reaches incidence directly or through intermediate conditions, and the structural equation model addresses it. Fit was acceptable, with a comparative fit index of 0.94, a Tucker-Lewis index of 0.92, a root mean square error of approximation of 0.061, and a standardized root mean square residual of 0.048. Table 10 reports the standardized paths.

Table 10

Standardized path coefficients from the structural equation model

Pathway	Standardized beta	p-value
Climate variability to environmental pollution	0.31	<0.001
Climate variability to pathogen burden	0.24	<0.001
Pollution to pathogen burden	0.19	0.006
Pathogen burden to incidence	0.43	<0.001
Urbanization to incidence	0.21	0.003
Climate variability through pathogen burden, indirect	0.10	0.008

Climate variability through pollution and pathogen burden, indirect	0.03	0.021
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Climate variability was associated with pollution ($\beta = 0.31$, p less than 0.001) and with pathogen burden ($\beta = 0.24$, p less than 0.001), and pollution was independently associated with pathogen burden ($\beta = 0.19$, $p = 0.006$). Pathogen burden carried the strongest direct association with incidence ($\beta = 0.43$, p less than 0.001), and urbanization was additionally associated with incidence ($\beta = 0.21$, $p = 0.003$). Both indirect pathways were positive and significant, at 0.10 ($p = 0.008$) and 0.03 ($p = 0.021$), which indicates that climatic variability reaches incidence both through a pathogen-mediated route and through a route that additionally passes through environmental pollution. Figure 9 orders the standardized coefficients by magnitude, and Figure 10 presents the same structure as a path diagram.

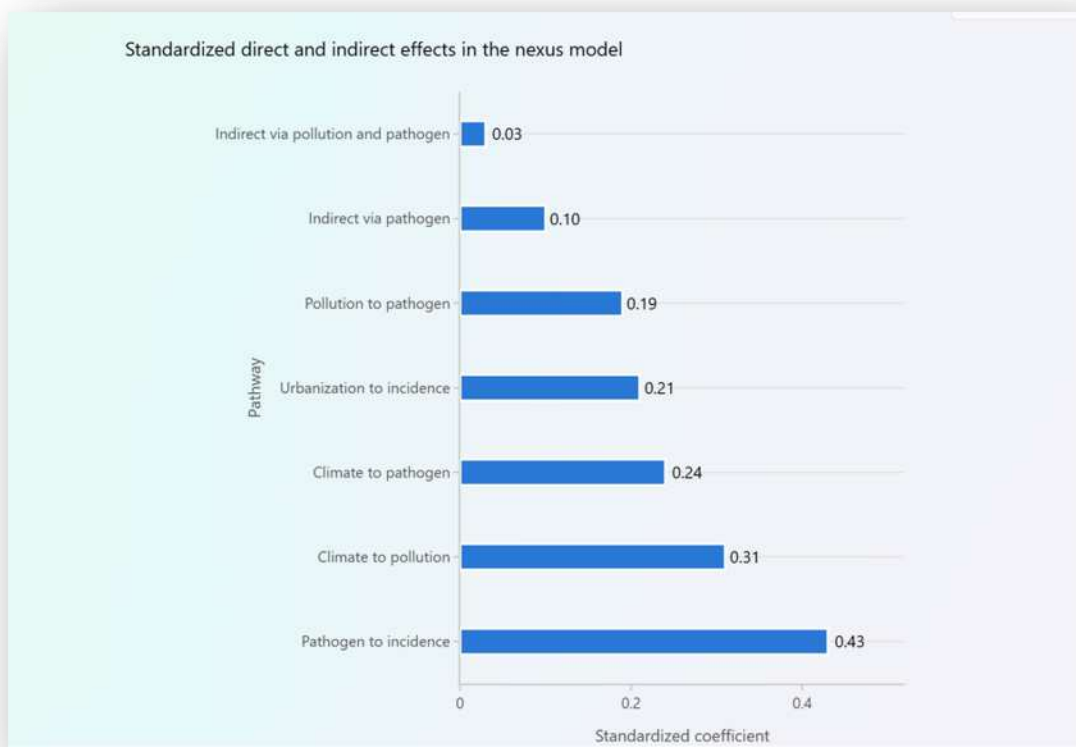


Figure 9. Standardized direct and indirect effects ordered by magnitude

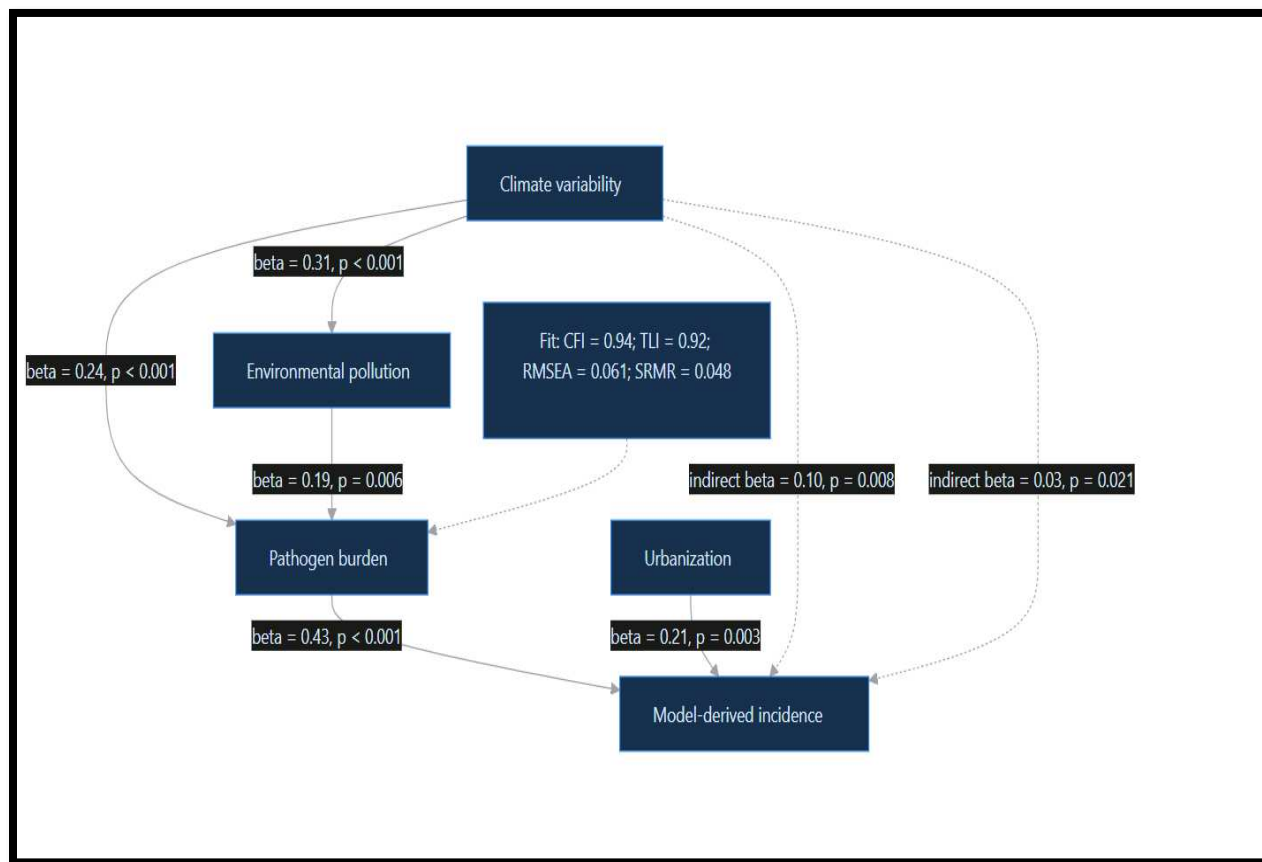


Figure 10. Structural path diagram with standardized coefficients and fit statistics

6.9 Nonlinear dose-response thresholds

Linear coefficients summarize an average slope, and the spline fits show that the underlying curves bend. Table 11 reports the inflection points identified for temperature, rainfall, and PM2.5.

Table 11

Inflection points from restricted cubic spline models

Exposure	Functional form	Inflection point	Direction above inflection
Temperature	Gradual rise with steepening slope	Approximately 29 degrees Celsius	Incidence rises more rapidly
PM2.5	Broadly positive	Approximately 70 $\mu\text{g}/\text{m}^3$	Steepest increase in predicted incidence

Rainfall	Inverted U	Approximately 250 to 300 mm per month	Weaker association
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The temperature inflection at approximately 29 degrees Celsius lies approximately half a standard deviation above the observed mean of 27.4 ± 3.2 degrees Celsius, placing it near the upper end of the observed range. The PM2.5 inflection at approximately $70 \mu\text{g}/\text{m}^3$ falls between the urban mean of $71.9 \mu\text{g}/\text{m}^3$ and the peri-urban mean of $53.5 \mu\text{g}/\text{m}^3$, which accounts for the stronger urban coefficient reported in Section 6.7 and turns a statistical artifact into a spatial story. The rainfall inflection at approximately 250 to 300 mm per month matches the weakening of the rainfall and pathogen relation at very high rainfall described in Section 6.4, implying that the most transmission-favorable precipitation range lies well below the observed maximum of 823 mm.

6.10 Robustness of principal estimates

Before placing weight on any of this, the principal estimates were put under pressure. Table 12 reports their stability across specifications.

Table 12

Stability of principal associations across sensitivity specifications

Specification	Temperature association	PM2.5 association	Temperature by PM2.5 interaction
Primary model	Positive	Significant	Positive and significant
Area-wise exclusion, sequential	Stable in direction and magnitude	Stable in direction and magnitude	Remained positive
Removal above the 99th percentile	Remained positive after removal of extreme temperature observations	Remained significant after exclusion of highest pollution measurements	Remained positive
Additional spatial and seasonal adjustment	Stable	Stable	Remained positive, strength reduced
Composite outcome index	Stable in direction and approximate magnitude	Stable in direction and approximate magnitude	Not applicable

The stability of the estimates under area-wise exclusion indicates that no single metropolitan area drove the pooled results, which matters in a panel built from six areas of quite different size and character. And the persistence of the PM_{2.5} association after removal of observations above the 99th percentile indicates that the relation operates across the bulk of the exposure distribution rather than only in its upper tail. The one genuine sensitivity was the interaction term, which weakened after spatial and seasonal adjustment, suggesting that part of the apparent temperature and pollution interaction is shared with unmodeled spatial and seasonal structure.

6.11 Integrated systems representation and forward forcing

The estimates resolve into four subsystems (hydroclimatic drivers, environmental contamination, exposure and vulnerability, pathogen amplification) plus dynamics that either accelerate or restrain transmission, mapped in Figure 11. Climate forcing enters through the hydroclimatic subsystem, whose rainfall threshold of 70 mm at lags of 0 to 19 weeks governs larval habitat availability. Environmental contamination receives particulates, effluent, and solid waste and delivers them into the same physical space in which people live and in which vectors breed. Exposure and vulnerability translate physical conditions into contact through water storage, shared sanitation, shaded premises, and residential density. Pathogen amplification converts those conditions into transmission, and it carries the largest standardized direct effect on incidence at 0.43.

Land conversion is the tap that keeps this system fed, and the record of it in Dhaka is unusually legible. Mean land surface temperature ran from 28.73 degrees Celsius in 1993 to 32.01 in 2007 and 35.16 in 2020, with 44.58 degrees Celsius recorded as the 2020 maximum, and the conversion effects attached to each land cover transition were 11.12 degrees for lowland to bare soil, 8.98 degrees for lowland to built-up area, 7.61 degrees for water body to built-up area, and 6.59 degrees for vegetation to built-up area. Daytime surface urban heat island intensity reached 2.88 degrees Celsius in Dhaka against 0.84 in Rajshahi, and nighttime intensity 1.91 degrees Celsius in Chittagong against 0.30 in Sylhet, with the national urban population projected to reach 81.4 million by 2029. None of this is incidental to transmission. Every hectare moved from lowland, water body, or vegetation to built surface raises land surface temperature, removes the drainage capacity that would otherwise clear standing water, and pushes households toward stored water, which is precisely the container habitat in which *Aedes* larvae develop. The spiral closes because the response to unreliable supply is itself the exposure. Outbreak pressure tends to purchase the intervention that is fastest to deploy rather than the one that lasts. Capacity is diverted toward reactive clinical management and chemical vector control, which selects for insecticide resistance and erodes the effectiveness of the next round of spraying. The entomological record fits this reading: documented resistance mechanisms in Bangladesh include detoxification enzymes and knockdown resistance mutations, and use of mosquito spray, coil, or smoke showed no significant association with household infestation in a setting of high pyrethroid resistance. The trap is not a failure of effort but a consequence of its form. Among surveyed slum residents, 49.0 percent waited

more than seven days before seeking treatment; the delay lengthens the infectious period, which raises onward transmission, which increases burden, which overloads facilities, which delays care further. The national mortality pattern is consistent with the same bottleneck, since 67.4 percent of the 2023 dengue deaths, or 1,015 of 1,705 patients, occurred within one day of hospital admission.

The model also resolves a contradiction. In the city-level monitoring component, particulate concentration was highest in Zone-6 and Zone-3 and lowest in Zone-4, which are comparatively affluent zones, while communicable disease occurrence was highest among lower-income households. The discrepancy disappears once vulnerability is specified: affluent zones carry higher instantaneous roadside concentrations from vehicular and construction sources, while lower-income zones carry longer exposure duration, greater dependence on untreated water at 47.6 percent, more reliance on shared sanitation, more household water storage, and delayed treatment-seeking at 49.0 percent. The model therefore predicts that the same atmospheric concentration yields different outcomes along the vulnerability gradient, which is the observed pattern, and the inverse vegetation association, at an IRR of 0.94 per 0.1-unit increase in the normalized difference vegetation index, adds a further element since peri-urban sites retain an index value of 0.37 against 0.21 in urban sites.

Minimum temperature is projected to rise more rapidly than maximum temperature in all seasons. Table 13 reports the forcing values, and Figure 12 displays the asymmetric warming pattern.

Table 13

Projected climate forcing across scenarios

Scenario	Precipitation change (percent)	Maximum temperature change (degrees Celsius)	Minimum temperature change (degrees Celsius)
SSP1-2.6	9.48	1.09	1.17
SSP2-4.5	13.63	1.60	1.91
SSP3-7.0	21.07	2.12	2.80
SSP5-8.5	30.90	2.99	3.69

Post-monsoon precipitation, projected to increase by 41.98 percent under SSP5-8.5, extends the window in which the 70 mm threshold can be exceeded, which is consistent with the observed lengthening of the transmission season beyond the historical June to September peak, while the

faster rise in minimum temperature relaxes the nocturnal constraint on vector development and is consistent with the shift toward year-round transmission. Winter precipitation, projected to decrease by 11.12 percent in the mid-future under SSP3-7.0, is a partial countervailing factor whose net effect on particulate accumulation cannot be established from precipitation projections alone.

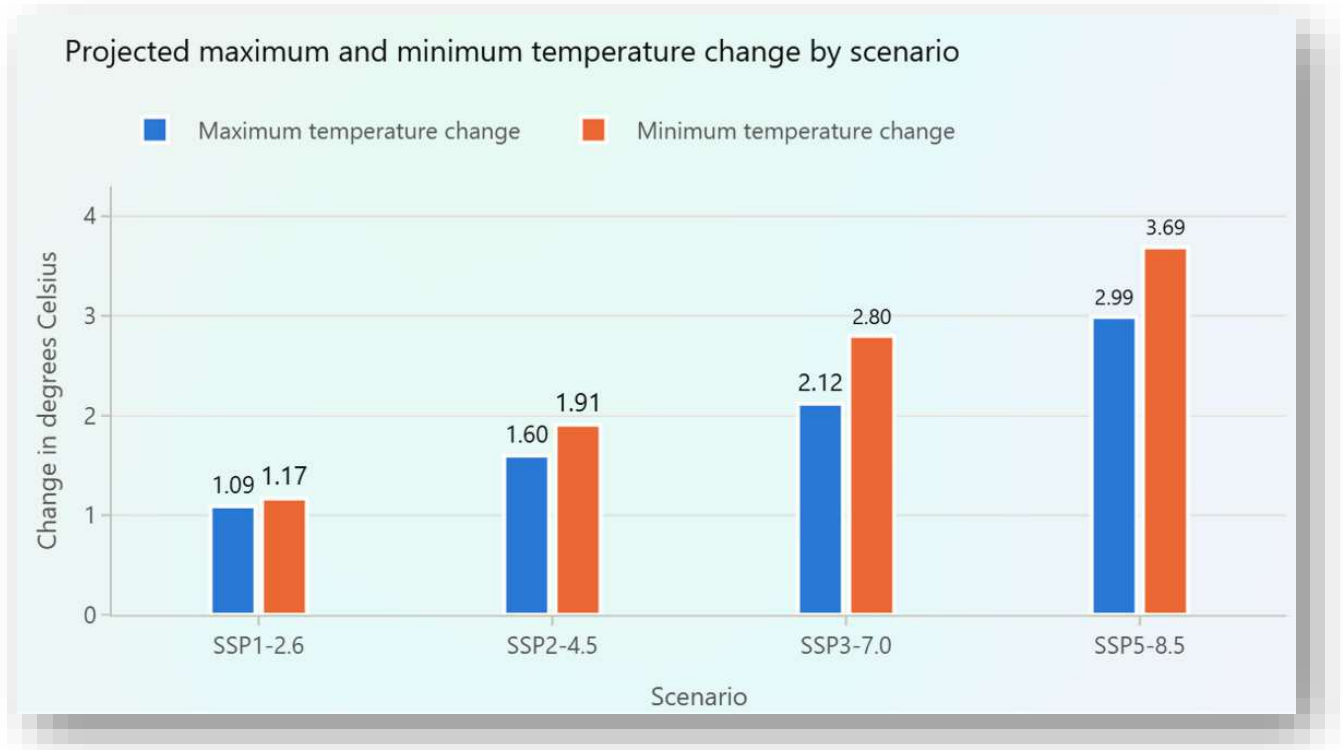


Figure 12. Asymmetric projected warming across scenarios

7. Conclusion

This study demonstrates that infectious disease emergence in Bangladesh operates as a coupled, interactive system rather than a series of isolated environmental exposures. The non-linear amplification of temperature effects under elevated particulate concentrations, alongside the direct and mediated paths identified through structural equation modeling, confirms that warming and ambient pollution act synergistically to heighten disease risk with urban centers bearing the heaviest structural burden. While these findings offer a clearer picture of transmission dynamics, the underlying framework relies on model-derived pathogen metrics calibrated from documented exposure-response relationships and operates with unmeasured state variables such as antimicrobial resistance and micro-level sanitation practices. As unmodeled seasonal and spatial structures can partially overlap with these joint environmental interactions, future empirical validation will benefit from integrated spatial sampling of clinical, genomic, and WASH data.

Ultimately, addressing Bangladesh's escalating health vulnerabilities requires transitioning from reactive, single-driver interventions toward integrated One Health surveillance and environmental governance frameworks that simultaneously target climate resilience, pollution control, and urban planning.

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