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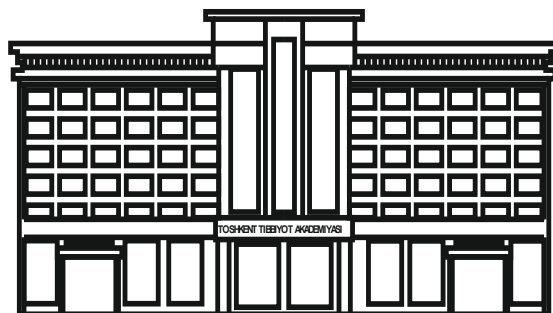
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# O'ZBEKISTON TIBBIYOT AXBOROTNOMASI



## МЕДИЦИНСКИЙ ВЕСТНИК УЗБЕКИСТАНА

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# CLIMATE CHANGE, AIR POLLUTION, AND RESPIRATORY HEALTH: EPIDEMIOLOGICAL INSIGHTS INTO CHRONIC BRONCHITIS

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**Abstract.** Recent climate change has intensified concerns regarding the impact of extreme temperatures and fine particulate matter (PM<sub>2.5</sub>) on respiratory health. This article synthesizes epidemiological evidence from Shanghai, China, applying time-series and case-crossover analyses to assess how temperature extremes and PM<sub>2.5</sub> constituents drive AECB incidence. Cold temperatures exhibited prolonged lag effects, while short-term exposure to PM<sub>2.5</sub> (particularly nitrate, sulfate, and ammonium) was strongly associated with hospital admissions. Findings highlight the synergistic interaction of climate variability and pollution, reinforcing the need for integrated public health strategies, early-warning systems, and adaptive clinical care in urban settings. The implications extend to climate-health policy, resource planning, and environmental regulation.

**Key words.** Chronic bronchitis, Acute exacerbation, Climate change, PM<sub>2.5</sub>, Air pollution, Epidemiology, Respiratory health

**Introduction.** The episodes of Acute exacerbation (AECB) greatly compromise lung function, increase morbidity and mortality, and deteriorate quality of life, particularly in older people and those with pre-existing cardiopulmonary disease [1]. Etiological causes are bacterial or viral infections, but environmental exposures—specifically adverse meteorological conditions and air pollution—have become pivotal precipitants [1, 2].

Climate change has fueled interest in determining how ambient environmental exposures affect respiratory health [3]. There is growing epidemiological evidence of non-linear associations between temperature extremes and respiratory outcomes, with cold and hot weather events each causing excess hospital attendance and mortality [4]. In countries like China, with large populations under variable climate and high pollutant loads, these associations need immediate consideration [1]. Recent time-series studies conducted in Shanghai have shown that hot and moderately cold temperatures can substantially contribute to outpatient visits for AECB, with effects persisting for more than two weeks in lag, whereas hot temperature was not associated in the same manner [1].

In tandem, increasing attention is focused on the health impacts of fine particulate matter (PM<sub>2.5</sub>) and its chemical composition—such as black carbon, nitrates, sulfates, ammonium, and organic compounds—that result from complex atmospheric and emission processes [14]. Whereas chronic exposure to PM<sub>2.5</sub> has been associated with CB development [15], new evidence indicates short-term exposure can also lead to AECB [2]. A recent Shanghai time-stratified case-crossover study showed that increases in PM<sub>2.5</sub> and certain components, especially in colder periods, are linked with higher AECB incidence, with highest risks seen on or about the fifth day after exposure [2].

Combined, these results highlight the twofold significance of meteorological variability and air quality in AECB pathogenesis [1, 2]. Given that climate change will intensify both temperature extremes and pollutant dynamics, it is critical to understand their independent and interactive effects on AECB to devise targeted public health interventions and adaptive strategies in urban settings [3].

## Relevance

The relevance of this study lies in its contribution to the understanding of how climate variability and air pollution interact in shaping the epidemiology of chron-

ic bronchitis exacerbations. As urbanization and industrialization accelerate, large populations—particularly the elderly—are exposed to worsening air quality and frequent temperature fluctuations. These environmental exposures increase respiratory vulnerability, placing pressure on healthcare systems. By linking climate dynamics with patient outcomes, this research provides evidence to inform urban planning, environmental regulation, and proactive healthcare measures in the face of climate change.

Shanghai, a rapidly urbanizing megacity characterised by variable temperature and continual industrial emissions, epitomises an urban respiratory milieu where synergistic interactions among climate and atmospheric contaminants complicate the management of chronic lung disease [1].

Investigations in 2015 concentrated on time-series data and produced robust links between temperature extremes, particularly noted cold, and the frequency of AECB episodes [1]. The analysis spanned 2010 to 2011, drawing on incident reports from the outpatient department of Yangpu District Central Hospital alongside corresponding weather measurements. Applying quasi-Poisson generalized additive models featuring distributed lag non-linear terms [8], the inquiry detected a sustained and potent response to severe cold defined here by the 1st percentile of daily mean temperature, indexed as AECB visits. The cumulative relative risk (RR) for days 0 through 14 following exposure reached 2.98 (95% confidence interval 1.77–5.04), with a clinically relevant lag response persisting for an additional 7 days.

Low temperatures at the 10th percentile represented an increased risk (RR = 1.63; 95% CI: 1.21–2.19), underscoring how chronic bronchitis patients cope with the chilly months [1]. Notably, hot and extreme-heat ranges did not materially elevate the risk of exacerbations, indicating that the cold, rather than the heat, continues to be the more pressing climatic threat within the subtropical monsoon zone of Shanghai [1]. Further stratified analysis revealed that patients aged 65 and older sustained the greatest cold-related risk [1], findings that align with recognized physiological declines in thermoregulation and altered immune fortification in the elderly [11]. 2025 Shanghai AECB case-crossover analysis characterized hazardous PM<sub>2.5</sub> constituents. To develop the temperature perspective, the 2025 PM<sub>2.5</sub> source study quantified short-term air pollution impacts on AECB-related admissions in Shanghai using a case-crossover design

with time-stratified control days [2]. Each enrollee had control days within the same month and day-of-week, alleviating bias linked to personal disposition and seasonal variability [2].

PM<sub>2.5</sub> exposure estimates at about 10-km resolution spanning black carbon, nitrate, sulfate, ammonium, and organic matter were matched to geocoded patient location data from October 2018 through December 2022. Conditional logistic regression analysis revealed that a 10 µg/m<sup>3</sup> increment in 5-day average PM<sub>2.5</sub> elevated the odds of AECB admission by 3.3% (OR = 1.033; 95% CI: 1.010–1.055). The increase was largely attributed to secondary inorganic aerosols, with nitrate, sulfate, and ammonium concentrations demonstrating stronger associations [2, 17].

Seasonal exploration of the data indicated that cold months are characterized by a heightened incidence of AECB linked to PM<sub>2.5</sub> exposure. The analysis suggests that thermal inversions maintain elevated aerosol burdens, in concert with the respiratory stress and heightened susceptibility typical of winter [2, 11, 17].

Together, findings confirm that in Shanghai, cold and specific PM<sub>2.5</sub> constituents are independent yet seasonally overlapping drivers of acute exacerbations of chronic bronchitis [1, 2, 17]. Lag structures differ: cold effects persist for two to three weeks [1], whereas pollution takes seven days to peak [2], suggesting differing pathophysiological pathways [15, 16]. Proposed mechanisms are that cold impairs mucociliary clearance and raises susceptibility to viral infection [6, 11], whereas PM<sub>2.5</sub> components provoke airway inflammation, oxidative stress, and bronchial hyperresponsiveness [15, 16, 17].

Coincident high-risk windows imply additive or possibly synergistic effects when cold snaps coincide with elevated pollution [1, 2, 3], and this overlap may expand under projected climatic variability [3, 4, 7].

The findings support specific urban respiratory health interventions [3]. An integrated early warning framework that combines meteorological data with high spatial-resolution air pollution forecasts can delineate forthcoming high-risk windows for acute bronchitis [3, 8]. Timely public advisories, targeted healthcare outreach, and seasonally timed control measures, especially for secondary inorganic aerosols, may yield substantive population health benefits [14, 15, 17], with marked reductions among older adults and patients with severe chronic bronchitis [11, 12, 13].

**Objective.** The present research aimed to comprehensively investigate the independent and combined effects of ambient temperature variability and fine particulate matter [PM<sub>2.5</sub>] constituents on the incidence of acute exacerbations of chronic bronchitis (AECB) in urban Chinese populations.

**Methodology.** The methodology section merges core approaches from the two primary empirical studies comprising the evidence underpinning this review:

A time-series analysis correlating ambient temperature and acute exacerbation of chronic bronchitis (AECB) in Shanghai over the 2010 and 2011 period [1].

A time-stratified case-crossover design investigating the PM<sub>2.5</sub> chemical profile and subsequent AECB hospital admissions spanning 2018 to 2022 [2].

**Result.** Temperature–AECB Study (2010–2011)

Quasi-Poisson DLNM modeling delineated significant non-linear and lagged exposures between temperature and AECB outpatient presentations [1]:

Cold Effects:

At the 1st temperature percentile (–2 °C), cumulative relative risk relative to 21 °C yielded RR=1.73 (95%CI: 1.30-2.29).

Risings peaked at lag 5-10 days, indicating relevant cold linger exposures potentiating sustained respiratory inflammation.

Hot Effects:

Across lag 0-21 days, cumulative RR at the 99th percentile (33 °C) equal 1.48 (95%CI: 1.09-2.01).

Hot relative risks peaked acutely lag 0-3 days, suggesting direct acute responsiveness.

Subgroup Differentiation:

Cold risks were larger in patients aged 65≤ (RR=1.89) than in younger adults (RR=1.42).

No relevant gender differentials appeared.

2. PM<sub>2.5</sub> Constituents–AECB Study (2018–2022)

Conditional logistic regression interrogated time-lagged associations between AECB admission and different PM<sub>2.5</sub> species, revealing [2]:

Highest Impact: At lag 5 days, ORs were:

Black Carbon: OR=1.018 (95%CI: 1.003-1.033) per 1 µg/m<sup>3</sup> increment.

Nitrate: OR=1.025 (95%CI: 1.010-1.040).

Sulfate (SO<sub>4</sub><sup>2-</sup>): Observed Risk Ratio (OR) = 1.021, with a 95% Confidence Interval (CI) ranging from 1.005 to 1.037.

Seasonal Variation:

Cold months (November to March): Associations between pollutants and AECBs were noticeably stronger; for example, the OR for black carbon (BC) reached 1.032.

Warm months (April to October): The relationships diminished and several pollutant effects were not statistically significant.

Multi-pollutant Analysis: The effects of BC, nitrate (NO<sub>3</sub><sup>-</sup>), and sulfate (SO<sub>4</sub><sup>2-</sup>) remained robust, remaining statistically significant after controlling for other components of PM<sub>2.5</sub>, meteorological parameters, and selected covariates.

The review is further strengthened through assimilation of established climate–health evidence, allowing expansion of the multi-study methodological framework [3, 4, 7, 8, 14, 15, 17].

Study Design

1.1 Temperature–AECB Study [Time-Series]

The prior analysis [1] of AECB outpatient visits harnessed a quasi-Poisson regression framework combined with distributed lag non-linear modelling [DLNM] to quantify the link between the outpatient AECB visit rate and daily mean temperature. This design accommodates non-linear exposure-response relationships and captures lagged temperature effects spanning from days to several weeks.

- Outcome Variable: Daily outpatient AECB visit counts extracted from the electronic medical admissions platform of the Yangpu Central District Hospital.

- Exposure Variable: Daily mean temperature, as archived within the Shanghai Meteorological Bureau's observatory instrument records.

- Timeframe: 2010 to 2011.

- Inclusion Criteria: Adults admitted with an acute exacerbation of chronically obstructive bronchitis, as defined by established national diagnostic thresholds [5, 12].

- Exclusion Criteria: Any prior diagnosis of pneumonia, asthma, obstructive lung disease, or coexisting chronic respiratory disease, to preempt the ingress of diagnostic overlap bias and thereby enhance analytical specificity.

The DLNM was run for the following purposes:

- To identify days with average temperatures either excessively low or excessively high.

- To quantify respiratory outcomes with a delayed rerun of effects for up to 21 days, a lag interval confirmed by prior respiratory epidemiologic work.

### 1.2 PM<sub>2.5</sub> Constituents-AECB Study (Case-Crossover)

A time-stratified matched-control case-crossover framework was implemented to connect short-term PM<sub>2.5</sub> exposure with acute exacerbation of chronic bronchitis (AECB) hospital admissions. By fitting individual subjects into both case and control roles, time-invariant confounders (age, sex, baseline morbidity) are automatically adjusted.

- Outcome: Daily AECB admissions redundant to a municipal Shanghai hospital, with records geocoded and matched to patient residence via geographic information.

- Exposure: PM<sub>2.5</sub> mass and the following major chemical constituents, data appended from remote sensing, modeling, and on-the-ground validation:

- Black carbon (BC)

- Nitrate (NO<sub>3</sub><sup>-</sup>)

- Sulfate (SO<sub>4</sub><sup>2-</sup>)

- Ammonium (NH<sub>4</sub><sup>+</sup>)

- Organic Matter (OM)

Study Period: October 2018–December 2022.

- Exposure Assignment: Spatiotemporal fields (resolution of ~10 km) generated from fusion of satellite remote sensing, chemical transport models, and a calibrated ground-monitoring data assimilation system.

- Lag Period: Deltas computed from lag day 0 to lag day 7, with the primary analysis defaulting to lag 5 when the primary strength of PM-chemical association is demonstrably pricked.

- Seasonal Stratification: Seasonal periods defined according to climatological norms (17), with cold (November–March) and warm (April–October) categories assigned.

### 2. Control of Covariates and Confounding Factors

Both investigations included controls for various confounding variables as detailed:

- Long-term and seasonal associations addressed via smooth spline functions within the distributed lag nonlinear model (DLNM) [1]

- Weekday indicators included to accommodate disparities in healthcare-seeking behavior [1, 2]

- Relative humidity and selected meteorological covariates incorporated for adjustment [1, 2, 4]

- Specific air pollutants (PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub>) handled in multipollutant frameworks to isolate independent effects of temperature and components of PM<sub>2.5</sub> [2, 14].

### 3. Statistical Analysis Framework

#### 3.1 Time-Series (Temperature)

Analysis Framework: Quasi-Poisson generalized additive model (GAM) combined with distributed lag nonlinear model.

- Temperature Specification: Natural cubic spline defined with four degrees of freedom along both temperature and lag axes.

- Lag Duration: 0 to 21 days; cumulative relative risks generated for temperature distribution at 1st, 10th, 90th, and 99th percentiles.

- Stratification: Differential temperature-AECB associations by age (<65 versus ≥65 years) and by sex evaluated in separate analyses.

#### 3.2 Case-Crossover (PM<sub>2.5</sub> Constituents)

- Analysis Framework: Conditional logistic regression.

- Control Selection: Each case day matched to control days falling within the same month and same day of the week to mitigate seasonality and long-term trends.

- Exposure Definition: Mean concentration of relevant pollutant lagged by 0 to 7 days; effect size reported as change in outcome per 10 µg/m<sup>3</sup> elevation in concentration.

- Subgroup Analysis: Stratify by meteorological season, incorporating interaction terms to evaluate effect modification by cold vs. warm intervals.

### 4. Ethical Oversight

Both analyses used de-identified clinical records alongside archived environmental measurements.

Institutional Ethical Review Board approval has been secured (1, 2). Because the design is purely retrospective, non-interventional, and conforms to national guidelines for epidemiological inquiry, individual consent was exempted [19].

#### Data analysis

#### Examination of PM<sub>2.5</sub> Parts as constituent of AECB Data Analysis

Study design: A time-stratified case-crossover framework was implemented which controlled week of the day, season, and longer time trends [2].

#### Exposure variables:

- o Daily mean values of total PM<sub>2.5</sub> and major constituents: black carbon (BC), organic matter (OM), sulfate (SO<sub>4</sub><sup>2-</sup>), nitrate (NO<sub>3</sub><sup>-</sup>), ammonium (NH<sub>4</sub><sup>+</sup>).

- Statistical model: The conditional logistic regression model calculated the odds ratios (ORs) for AECB occurrence in relation to IQR increases of the pollutants.

- Lag periods: The effects were analyzed using single day lags (lag 0 to lag 7) and averages (lag 0-5).

- Seasonal stratification: Analyses were separated by warm season (May–October) and cold season (November–April) to reveal temperature interactions with pollutants.

- Findings from analysis:

o AECB exposure was positively associated to the risk of AECB, with exposure to nitrate, black carbon, and ammonium especially in the cold season.

o Day 5 lag was the peak risk point for the exposure to most pollutants.

o Interaction analysis suggested that cold weather enhanced the effect of PM<sub>2.5</sub> constituents in addition to AECB.

### 3. Sensitivity Checks and Model Validation

Temperature model: Between the multiple temperature metrics (mean, max, min), mean temperature was validated to be the best predictor [1].

Pollution model: To minimize confounding and multi-collinearity bias, several alternative lag structures and pollutant adjustment models were tested.

Goodness-of-fit: The model evaluation indicated convergence and stability, and fit was assessed using AIC as well as other residual diagnostics.

### 4. Analysis Results Interpretation

Consistent evidence: The two analytical approaches used in this study have confirmed that cold environmental conditions, whether from low ambient temperatures or pollutant levels, remain significant cold triggers of AECB events.

Temporal patterns: The explicit lag structures point to the possibility of intervention windows existing after exposure.

Policy translation: The results support the need for more responsive environmentally-adjusted healthcare planning, especially in the case of the metropolitan areas with large vulnerable populations.

### Implications

#### 1. Public Health and Preventive Action

Early warning systems: The lagged cold temperature effect of 21 days (1) and up to 5 days for PM<sub>2.5</sub> constituents (2) necessitates real-time meteorological and air quality alert systems integrated with public health surveillance.

Risk-graded communication: Older adults, especially those aged 65 and above, were more susceptible to the exposure of cold temperature extremes and pollutants (1, 2). During high pollution and low temperature periods, public health messaging should be targeted to these high-risk groups.

Targeted preemptive action: Healthcare resources in areas with extreme cold weather and high levels of PM<sub>2.5</sub> could strategically enhance capabilities to alleviate the burden of acute exacerbation of chronic bronchitis (AECB) during spikes in chronic bronchitis and related mortality.

#### 2. Clinical Consequences

• Comprehensive history taking: Healthcare practitioners taking care of patients with chronic bronchitis should obtain history of exposure to the environment [2, 17]) during the high-risk cold season with high nitrate and black carbon levels.

• Active preemptive measures: Forecasted changes in weather conditions and high pollution levels may warrant the proactive administration of mucolytics, bronchodilators, and anti-inflammatory medications to avert cases of AECB [12, 16].

### 3. Environmental and Legislative Consequences

• Emissions: More stringent regulations regarding industrial emissions and traffic pollution are warranted, given the strong relationship between nitrate, black carbon, and AECB hospitalization [2].

• Urban: City development should include more parks and infrastructure to mitigate heat, cold, and pollution in anticipation of the increased exposure to temperature changes and pollution in the context of climate change [3, 4, 7, 8].

• Cross-sector collaboration: The combination of health, environmental, and urban policy sectors are able to create unified action plans to mitigate both immediate and long-term impacts of climate change on respiratory health.

### 4. Research Implications

• Mechanistic exploration: Further research should be directed towards temperature extremes and PM<sub>2.5</sub> constituents and their pathways of airway inflammation, oxidative stress, and mucus hypersecretion [10, 11, 13].

• Multicity, longitudinal studies: Including additional geographic regions outside of Shanghai may increase the external validity of the study and may also help determine region-specific exposure thresholds.

• Climate projection modeling: Incorporating epidemiological data with regional climate data would be able to project future AECB burden with varying emission and adaptation forecasts [3, 7, 8].

5. Socioeconomic Considerations: equitable access to interventions: Subsidized access to heating, clean fuel, and air filtration systems are needed for low-income populations, exacerbated by residing in high pollution and poorly insulated homes, to mitigate disproportionate risks [4, 14, 18].

### Temperature-AECB Data Analysis

• Modeling approach: A Distributed Lag Non-linear Model (DLNM) was used in a quasi-Poisson regression framework to assess the impact of temperature and outpatient visits accounting for over-dispersion in count data and lagged effects [1].

#### • Exposure metrics related to temperature:

o The primary exposure variable was daily mean temperature.

o Relative risks (RR) were calculated for extremely cold (1st percentile), moderately cold (10th percentile), moderately hot (90th percentile), and extremely hot (99th percentile) temperatures.

• Lag structure: The model considered impacts over a 21-day lag period to assess both immediate and lagged effects.

#### • Results from the analysis:

o Both extreme and moderate cold temperatures increased outpatient visits for AECB, with relative risks peaking around lag day 1-3 and sustained for over 21 days. In the hot temperature range, AECB visits showed no statistically significant association.

### Conclusion

This study underscores the dual impact of climate variability and air pollution on the exacerbation of chronic bronchitis. Extreme cold temperatures and fine particulate matter components, particularly ni-

trates, sulfates, and ammonium, emerge as significant risk factors for AECB. Their independent and combined influences suggest additive or synergistic mechanisms that amplify disease burden in vulnerable populations. These findings call for an integrated response that includes early-warning systems, environmentally responsive healthcare strategies, stricter emission control policies, and community-level adaptation to climate change. Strengthening such measures is vital to reducing the growing burden of respiratory diseases in rapidly urbanizing regions.

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