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Temperature and Influenza in New York City: A Distributed Lag Nonlinear Modeling Study

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Abstract. Influenza continues to be a major seasonal public health issue, especially in large urban areas such as New York City. While prior studies have highlighted temperature as a crucial factor influencing influenza transmission, the detailed relationship between temperature variations and influenza incidence in NYC, including both immediate and delayed effects, remains insufficiently understood. This study uses the Distributed Lag Nonlinear Model (DLNM), which analyzes weekly laboratory-confirmed influenza cases from New York State Department of Health's open data portal, together with meteorological data obtained from John F. Kennedy Airport from January 2009 to May 2025. Results indicate a distinct nonlinear relationship between temperature and influenza risk, with sharply elevated risks at extremely low temperatures (e.g, -8 °C) and significantly reduced risks at warmer temperatures (e.g, around 16°C). Lag analysis further demonstrates that cold temperatures exert delayed effects on influenza incidence, with peak risks observed two weeks following exposure to low temperatures. These findings underscore the critical role of temperature in influenza transmission dynamics and suggest the need for targeted public health interventions during periods of cold weather.

Keywords: Influenza, Temperature, Distributed Lag Nonlinear Model (DLNM), Seasonal Epidemiology, New York City

1. Introduction

Influenza remains a leading seasonal infectious disease in the world, and it frequently causes epidemics. The Centers for Disease Control and Prevention (CDC) estimates at least 56 million flu-associated illnesses during the 2024-2025 season [1]. Globally, influenza outbreaks do impose substantial morbidity, mortality, and economic costs. Dense urban areas like New York City (NYC) are especially vulnerable, owing to frequent international traveling and variable weather conditions. Influenza presents difficulties to the medical system every year.

Previous studies suggested that meteorological factors, especially temperature, strongly affect influenza transmission. Research across various geographic regions indicates that low temperatures are generally associated with increased influenza activity. Notably, in China and Seoul, there was a clear correlation between low temperatures and high risk of influenza, and the distinct patterns were noted across different climatic regions [2]. Specifically, in northern China, the lower temperature was associated with a higher incidence of influenza, while in central and southern China, the

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relationship exhibited more complex patterns characterized by both low and high temperatures [3]. Therefore, region-specific studies are necessary for verifying these correlations, since meteorological factors and influenza incidence can substantially vary across climatic and geographic contexts.

Despite these findings, the specific relationship between meteorological variations and influenza incidence within New York City remains underexplored, notably with regard to both immediate and delayed effects that meteorological changes produce. This research seeks to fill this gap by using a Distributed Lag Nonlinear Model (DLNM) to analyze weekly influenza incidence data from January 2009 to May 2025 and meteorological data from John F. Kennedy Airport together, aiming to lay a solid foundation for characterizing the mechanism of influenza transmission and effective intervention in public health.

2. Methods

New York City, situated along the northeastern Atlantic coast of the United States, is the nation's most populous metropolis [4]. Its temperate climate features cold winters and hot, humid summers, and the marked seasonal swings in temperature and humidity foster respiratory illnesses like influenza.

2.1. Source of data

Influenza case data were accessed through the New York State Department of Health's open data portal, specifically the Influenza Laboratory-Confirmed Cases dataset. The dataset records influenza cases from January 2009 to May 2025, encompassing the entire influenza season within these years [5]. Across New York State, healthcare providers and hospitals reported weekly counts of laboratory-confirmed influenza cases categorized by influenza type (A, B, or unspecified) in the dataset. Reporting laboratories submit data through the Electronic Clinical Laboratory Reporting System (ECLRS), utilizing various laboratory tests, including Rapid Influenza Diagnostic Tests (RIDT), immunofluorescence assays (DFA, IFA), rapid molecular assays, reverse transcriptase polymerase chain reaction (RT-PCR), nucleic acid amplification tests, and viral culture. The total cases confirmed are divided by county according to the patient's place of address, the provider's place of address, or the facility's place of address [6].

The National Oceanic and Atmospheric Administration (NOAA) website was the primary source of meteorological data, focusing on the John F. Kennedy Airport weather station. Daily meteorological records, including average temperature (℃), wind speed (m/s), and precipitation (mm), were retrieved for the same study period. Daily weather data were averaged into weekly readings, which directly correlate with the weekly influenza case reporting periods, thus allowing for detailed temporal analysis and straightforward comparison with weekly influenza incidence data.

2.2. Statistical analysis

This paper assessed the monotonic relationships by linking weekly weather variables with influenza incidences using the Spearman rank correlation test [3, 7]. This non-parametric method was adopted because the case counts of influenza and the meteorological variables were not normally distributed. The Spearman test provided a preliminary understanding of the strength and direction of associations among variables.

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This article then employed a Distributed Lag Nonlinear Model (DLNM) to investigate how meteorological factors influence influenza over time [8]. According to the Spearman correlation, the average temperature had the highest degree of correlation with flu cases and was used in the DLNM. Weekly influenza case counts were assumed to follow a quasi-Poisson distribution to account for overdispersion. A cross-basis function was constructed to model the exposure–lag–response relationship. Temperature was modeled using a natural cubic spline with internal knots at the 25th, 50th, and 75th percentiles to capture its nonlinear effects. For the lag dimension, this article restricted the model to the 0–2 week range and used an integer (indicator) basis, which assigns a separate coefficient for each lag week. This avoids issues of overfitting and allows for direct interpretation of the lag-specific risks [3]. The final statistical model is as follows:

$$\log(\mathrm{E}[\mathrm{Y_t}]) = \alpha + \beta * \mathrm{cb} * (\mathrm{T_t}) + \mathrm{s(t)} + \varphi \ln(\mathrm{Y_{t-1}} + 1) \quad (1)$$

where $\mathrm{Y_t}$ the weekly count of influenza cases, α is the intercept term. cb is the cross-basis matrix for temperature, constructed using an indicator basis over lags 0–2. β is the vector of coefficients associated with temperature effects. $\mathrm{s(t)}$ is a smooth function of calendar time to control for seasonality and long-term trends, with 2 degrees of freedom per year. $\ln(\mathrm{Y_{t-1}} + 1)$ is a log-autoregressive term to adjust for short-term autocorrelation, and φ is its corresponding coefficient.

The relative risks (RR) and 95% confidence intervals (CI) were estimated across the range of temperatures and lags. All analyses were performed using R Studio, version 4.4.1.

3. Results

3.1. Data overview

A total of 826,176 laboratory-confirmed influenza cases were reported in New York City from January 2009 to May 2025. Table 1 summarizes the weekly distribution of influenza cases and meteorological variables during the study period. The mean weekly number of flu cases was 1560.83 ± 3405.13, with values ranging from 9.4 at the 5th percentile to 8424.6 at the 95th percentile. The weekly average temperature ranged from -1.38°C to 18.43°C, with a mean of 7.92 ± 6.16°C. Weekly wind speed and precipitation averaged 12.11 ± 2.34 m/s and 0.12 ± 0.12 mm, respectively.

Table 1. Summary statistics of weekly influenza cases and meteorological variables in NYC (January 2009 — May 2025)

	Weekly Average Temperature (°C)	Weekly Average Wind Speed (m/s)	Weekly Average Precipitation (mm)	Weekly Flu Incidence Case
Mean ± SD	7.92 ± 6.16	12.11 ± 2.34	0.12 ± 0.12	1560.83 ± 3405.13
5%	-1.38	8.63	0	9.4
25%	3.33	10.45	0.02	45
Median	7.22	11.94	0.09	329
75%	12.14	13.64	0.17	1214
95%	18.43	16.15	0.34	8424.6

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Figure 1 displays the weekly trend of laboratory-confirmed influenza cases in New York City from January 2009 to May 2025. Influenza activity exhibited a distinct seasonal pattern, with sharp peaks occurring primarily from December to March. These peaks became more pronounced in recent years, particularly after 2020.

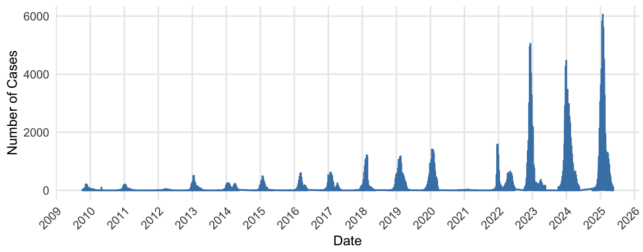

Figure 1. Seasonal patterns of weekly influenza incidence in NYC, January 2009 — May 2025

3.2. Spearman rank correlation

Spearman rank correlation analyses between influenza incidence and three meteorological variables: temperature, wind speed, and precipitation. Weekly influenza case counts showed a moderate correlation with temperature (r = -0.38) and wind speed (r = 0.16), indicating that lower temperatures and higher wind speeds were associated with increased influenza activity. In contrast, precipitation was only weakly correlated with flu incidence (r = 0.06). These results suggest that temperature is the most significant meteorological factor for modeling influenza trends in New York City.

Figure 2 presents the overall cumulative exposure-response relationship between weekly average temperature and the relative risk (RR) of influenza incidence, as estimated by the DLNM. The curve demonstrates a clear nonlinear pattern. The RR increases sharply at extremely low temperatures and gradually decreases with rising temperature, reaching its lowest point around 16°C. The estimated RR at -8°C was 39.54 (95% CI: 2.604–600.412), indicating a substantially elevated risk, though with high uncertainty due to sparse data. At moderate temperatures (e.g., 7.2°C), the RR was 1.109 (95% CI: 1.021–1.206), suggesting a statistically significant but modest increase in risk. At warmer temperatures (16.0°C), the RR dropped to 0.128 (95% CI: 0.061–0.269), indicating a strong protective effect.

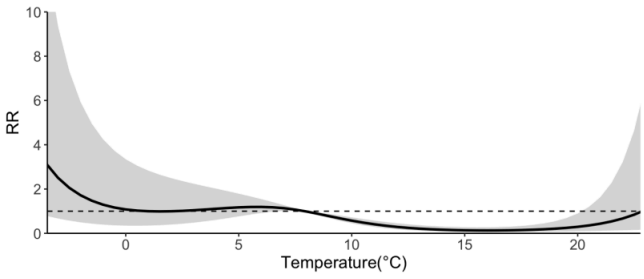

Figure 2. Nonlinear relationship between weekly temperature and influenza relative risk estimated by DLNM

Figure 3 illustrates the lag-specific effects of temperature on influenza risk across three temperature levels: -8 °C (low), 7.2°C (moderate), and 16°C (high). At -8°C, the RR was elevated at lag 0, dipped at lag 1, and peaked at lag 2 with an estimate of 1.409 (95% CI: 1.168–1.700),

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indicating the strongest delayed effect occurred two weeks after exposure. At 7.2°C, the RR showed a gradual increase across lags, reaching 1.062 (95% CI: 1.044–1.081) at lag 2. In contrast, at 16°C, the RR consistently decreased over time, falling to 0.617 (95% CI: 0.531–0.716) at lag 2.

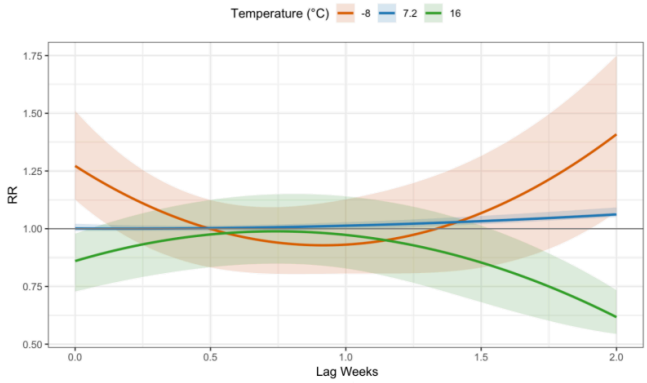

Figure 3. Effect of temperature on relative risk over lag weeks

4. Discussion

Over the years January 2009 to May 2025, the influenza cases in New York City exhibited a seasonal pattern with the weekly cases rising every winter, suggesting that colder periods contribute strongly to influenza outbreaks. Despite fluctuations in temperature during the study period, the highest influenza activity was consistently observed during the coldest temperatures.

4.1. Relationship between temperature and influenza

Spearman's rank correlation identified temperature as the meteorological variable most tightly linked to incidence, warranting its central role in the DLNM. The nonlinear relationship between temperature and influenza risk was observed, as illustrated in Figure 2. Risk increased sharply at lower temperatures, with the RR peaking at 39.54 at -8°C, though the wide confidence interval reflects limited data at this extreme. At more typical winter temperatures (7.2°C), risk remained modestly elevated (RR = 1.109), while at warmer temperatures (16°C), influenza risk decreased significantly (RR = 0.128), suggesting a protective effect.

Figure 3 demonstrates that cold temperature exposure is associated with delayed effects on influenza risk. For instance, at -8°C, the RR was highest at lag 2, indicating a possibility that low air temperature triggers a quick spike in respiratory viral infections and predisposes individuals or populations to secondary increases a week later. In contrast, at higher temperatures, the RR decreased consistently across all lag periods, reinforcing the protective role of warmth over time.

4.2. Mechanism of temperature affecting influenza

Influenza seasonality in temperate regions is primarily driven by enhanced respiratory droplet transmission under cold and dry conditions, with absolute humidity and temperature identified as key environmental predictors [9]. Low air temperature produces an environment for more stable viral materials in air and thus increases the duration of droplet existence in the air. It also hampers the impact of mucus membranes clearance, predisposing one to infections. These events probably encapsulate the known cause of the upsurge of transmission risk during and after a temperature drop.

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At the molecular level, temperature-sensitive molecular conformations of influenza mRNA secondary structural motifs have also been shown to be the basis for temperature sensitivity. Structural changes particularly affect the polymerase genes (PB1, PB2, and PA), which are essential for viral replication [10]. Environmental factors such as ambient temperature may cause the RNA configuration to snap back and forth, which also has an impact on the replication process. Such temperature-sensitive structural dynamics can help explain the immediate (lag 0) impact of cold weather on influenza incidence observed in our study.

5. Conclusion

In conclusion, this study shows that lower temperatures are strongly associated with increased flu morbidity among New York City. By browsing the DLNM framework, it was evident that there were not only instant but also lingering effects of cold conditions on flu incidents, while warm weather was related to flu-less incidence. These patterns support the role of temperature in physiological processes and hence the seasonal flux of flu cases. Such features as data coverage and model scope limit our analysis, which in turn concludes that the forecasting of the immunization against weather factors in health planning and recommends more detailed study involving other factors at a local level.

Despite significant contributions in this study, it is crucial to acknowledge the limitations. First, the flu data only include the lab-confirmed cases and lack the presence of the untested or unreported infections; hence the assumption that this data only reflects the total incidence. Variability in testing practices and test accuracy, such as false negatives, may also affect consistency. Second, all weather data came from JFK Airport, which may not reflect microclimate variation across NYC. Localized differences due to urban layout or coastal effects are not captured.

Future research should explore microclimatic differences around New York City by integrating data from multiple weather stations to understand influenza risk more precisely. Additionally, researchers could improve predictive accuracy for influenza outbreaks if they investigate combined effects of other meteorological factors such as humidity and air pollution.

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