

Before anyone Notices how Pandemic Viruses Spread across U.S. Cities

Pandemic [respiratory viruses](#) rapidly reach most metropolitan areas through distinct transmission pathways; however, unpredictable viral dynamics often limit their timely detection and containment. In a new study posted, researchers use high-resolution disease data to reconstruct the spatial spread of pandemic respiratory viruses across metropolitan areas throughout the United States.



Study

Several viral outbreaks have occurred in recent years due to the emergence of novel respiratory pathogens, such as A/H1N1 (H1N1pdm) influenza and the [severe acute respiratory syndrome coronavirus 2](#) (SARS-CoV-2), which have significantly increased morbidity, mortality, and socioeconomic disruption worldwide. Pandemic respiratory viruses, such as SARS-CoV-2, the causative agent of coronavirus disease 2019 (COVID-19), can rapidly adapt and spread, often crossing species barriers and geographic boundaries, thereby emphasizing the importance of early detection, surveillance, and coordinated public health responses.

Understanding how novel respiratory [pathogens](#) spread geographically is crucial for effective pandemic preparedness and response. Human mobility, including both long-distance air travel and short-range commuting, is critically involved in viral transmission rates; therefore, combining data on viral genetics and population movements could clarify global, national, and local trends.

In the United States, several factors influence the spread of both [influenza](#) and COVID-19. However, limited case reporting combined with the lack of detailed surveillance data makes it challenging to fully understand the early spread of these viruses at more granular geographic scales, particularly during the earliest stages of a pandemic when under-ascertainment is substantial.

Researchers combined city-level [influenza-like illness](#) (ILI) records from medical claims with estimates of daily SARS-CoV-2 infections at the county level. This included both reported and unreported cases to reconstruct the transmission pathways of H1N1pdm influenza and SARS-CoV-2 across U.S. metropolitan areas.

[Metropolitan Statistical Areas](#) (MSAs), as densely populated regions with strong social and economic connections, functioned as effective units for analyzing how respiratory pathogens spread. By mapping transmission networks among MSAs, spatial spread patterns of both viruses could be compared.

Although human mobility is directly implicated in the introduction of infections, the realized transmission network, which comprises the actual chain of who infected whom, represents only one of many ways an outbreak can develop. To quantify any uncertainty in early spatial dynamics, the current study proposed a process-based stochastic transmission model that incorporates inter-MSA air travel, [commuting patterns](#), and pathogen superspreading potential.

Results

A hypothetical novel respiratory virus originating in Minnesota was simulated for the current analysis to evaluate how stochastic transmission dynamics influence early spatial spread. For each MSA, the onset time of [local transmission](#) was estimated, and infection sources were identified based on the number of infections introduced from other MSAs.

Transmission links were based on identified infection sources and categorized by the dominant [mobility mode](#). The simulated transmission network exhibited a hub-and-spoke structure, which is consistent with previous simulation studies.

To assess variability in transmission [network structure](#), 100 simulations were performed, and transmission links were grouped by occurrence probability. The onset of the outbreak at the epidemic origin occurred after a median of three weeks, resulting in 670 infections.

Of 994 distinct [transmission](#) links, 56.9 % appeared in less than 20 % of simulations, indicating significant variation. In a single realization, 71.1 % of links were stable and present in over 80 % of simulations. Variability increased with lower transmissibility and greater superspreading potential.

A prediction-based inference framework for spatial infectious [disease](#) spread was validated using a simulated outbreak with known transmission networks. Due to model randomness, 100 independent inference realizations were performed to capture uncertainty.

The [algorithm](#) achieved 79.3 % precision and 78.2 % recall in identifying true transmission links for individual inference realizations. By aggregating results across inference runs and selecting transmission links based on their occurrence probability, overall inference performance improved substantially, with inference accuracy increasing as link occurrence frequency increased.

The inferred [SARS-CoV-2 transmission](#) network consisted of 304 links, featuring a hub-and-spoke pattern, with Seattle and New York identified as key sources of national spread through air travel. Nevertheless, regional areas like Chicago, Atlanta, New Orleans, and San Francisco facilitated local dissemination. Most inferred inter-metropolitan transmission events occurred between late February and mid-March 2020.

Network structure remained consistent across different thresholds, origins, and immunity durations. [Pandemic](#) influenza activity was measured by multiplying weekly local ILI incidence with A/H1N1pdm lab positivity rates to create the ILI+ metric. For pandemic influenza, early seeding locations were assumed to be San Diego, San Antonio, and New York, based on initial confirmed cases.

The reconstructed pandemic influenza network differed structurally from SARS-CoV-2 but shared a hub-and-spoke pattern. Most highly connected locations were considered major international

travel centers. However, some MSAs with high international travel, like Miami and Los Angeles, were not major transmission sources, suggesting that international travel volume alone does not predict spatial spread. Compared with SARS-CoV-2, fewer high-confidence transmission links could be inferred for [H1N1pdm influenza](#), likely due to the coarser temporal resolution and sparsity of available surveillance data.

Conclusion

Pandemic respiratory [pathogens](#) can rapidly establish widespread local transmission, often before detection or intervention is possible. Despite differences in transmission pathways, both pandemics analyzed in the current study occurred through common transmission areas and stochastic factors that complicated containment efforts.

Simulation results further suggest that early detection strategies, such as airport wastewater surveillance, may be most effective when deployed across a broad set of metropolitan hubs rather than a small number of major airports. Importantly, the benefits of expanded surveillance depend on pairing detection with interventions that meaningfully reduce onward transmission once infections are identified. Future studies are needed to refine simulation models that incorporate greater social and demographic details, as well as evaluate [pragmatic surveillance](#) and intervention strategies to enable the development of effective responses to future pandemics and mitigate their societal impact.

Source:

<https://www.news-medical.net/news/20260112/How-pandemic-viruses-spread-across-US-cities-before-anyone-notices.aspx>