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Epidemiological and Virologic Assessment of Influenza Activity in the United States: Weeks 1-2, 2026

Public Health * Urban Environment * Epidemiology

The 2025-2026 Influenza Season at an Inflection Point

The opening fortnight of 2026 marks a pivotal epidemiological juncture in the 2025-2026 Northern Hemisphere influenza season. Following an accelerated viral surge that began in late October 2025 and intensified across the Thanksgiving and Christmas holidays, surveillance data from the Centers for Disease Control and Prevention (CDC) for Week 1 (ending January 10, 2026) and Week 2 (ending January 17, 2026) document a sustained post-holiday deceleration.[1][2][3][6] Classified as "moderately severe" overall -- and "high severity" in the pediatric (0-17 years) age group -- under the CDC's in-season framework, the epidemic remained overwhelmingly dominated by Influenza A(H3N2) Subclade K, generating substantial morbidity across pediatric and geriatric cohorts.[1][3][6] Across both clinical laboratories and public health subtyping in Week 2, national test positivity fell for a third consecutive week while A(H3N2) accounted for 91.2% of subtyped Influenza A viruses, with A(H1N1)pdm09 remaining a minor co-circulating share at 8.8%.[6]

Cumulatively since late September 2025, this segmented negative-sense RNA virus caused an estimated 18 million symptomatic illnesses, 9.3 million outpatient medical visits, 230,000 hospitalizations, and 9,300 deaths through Week 1 -- rising to at least 19 million illnesses, 250,000 hospitalizations, and 10,000 deaths by Week 2.[3][6] While overall adult burden stayed below the 2017-2018 H3N2 ceiling, a spike of 15 pediatric fatalities in Week 1 and 12 additional pediatric deaths in Week 2 (bringing the season total to 44) underscored acute public health risk in unvaccinated children.[3][6] This report synthesizes national syndromic and virologic surveillance, antigenic drift characterization, vaccine effectiveness, and respiratory co-circulation dynamics across early 2026.

National Surveillance Kinetics and Regional Heterogeneity

Syndromic tracking through the U.S. Outpatient Influenza-like Illness Surveillance Network (ILINet) recorded a national ILI rate of 5.3% in Week 1, down from 7.2% in Week 53, before easing further to 4.4% in Week 2.[4][5][6] Despite this three-week decline, outpatient respiratory volume remained well above the national baseline.[3][4][6] Clinical laboratory testing corroborated the cresting of the initial wave: specimen positivity declined from 24.7% in Week 53 to 18.6% in Week 1 and 17.7% in Week 2, while jurisdictions reporting "High" or "Very High" ILI activity contracted from 45 in Week 53 to 36 in Week 1 and 31 in Week 2.[3][4][5][6] Consistent with Kermack-McKendrick compartmental dynamics, this inflection reflects holiday school closures and partial depletion of susceptible host networks, though emergency department visits among school-aged children ticked upward again in Week 2.[6]

| National Surveillance Metric | | Week 53 (Ending Jan 3, 2026) | | Week 1 (Ending

Jan 10, 2026) | | Week 2 (Ending Jan 17, 2026) |

| Clinical Lab Test Positivity | | 24.7% | | 18.6% | | 17.7%[4][5][6] |

| Outpatient ILI Visits (%) | | 7.2% | | 5.3% | | 4.4%[4][5][6] |

| High / Very High ILI Jurisdictions | | 45 | | 36 | | 31[3][5][6] |

| Cumulative Pediatric Deaths | | 17 | | 32 | | 44[3][6] |

| Influenza A / B Share (Clinical Labs) | | 94.1% A / 5.9% B | | 96.5% A / 3.5% B | | 91.2% A / 8.8% B[2][3][6] |

Geographically, transmission has propagated asynchronously across Department of Health and Human Services (HHS) regions, creating a rolling continental epidemic:

* HHS Region 2 (New York, New Jersey, Puerto Rico, U.S. Virgin Islands): Emerged as the primary national hotspot in Week 1, recording the highest clinical laboratory test positivity in the country at 22.9%.[3] This elevated intensity reflects a later onset of the primary H3N2 wave across the Northeast compared to southern states, compounded by high urban commuter density along the Acela corridor and winter indoor congregation.

* HHS Region 8 (Mountain West): Encompassing Colorado, Utah, Montana, Wyoming, and the Dakotas, Region 8 led the nation in Week 53 with a peak test positivity of 31.7%.[2] By Week 1, positivity had begun to recede following the characteristic rapid-ascent, rapid-decay trajectory of H3N2 epidemics in colder mountain climates.

* HHS Region 9 (Arizona, California, Hawaii, Nevada): Further along the initial epidemic curve, Region 9 posted the lowest regional positivity at 11.5% in Week 1 -- nearly half the rate observed in the Northeast -- before select Midwest, central, and West Coast indicators stabilized or ticked up modestly in Week 2.[3][4][6]

| HHS Region | | Week 53 Positivity | | Week 1 Positivity | | Epidemiological Phase |

| Region 2 (NY/NJ/PR/USVI) | | High / Ascending | | 22.9% (National High) | | Peak / Plateau[3] |

| Region 8 (Mountain West) | | 31.7% (Prior Peak) | | Decreasing | | Post-Peak Decline[2][3] |

| Region 9 (West Coast) | | Moderate | | 11.5% (National Low) | | Post-Peak Stabilization[3][6] |

Virologic Characterization: H3N2 Subclade K Dominance and B/Yamagata Extinction

Throughout late 2025 and early January 2026, Influenza A(H3N2) drove epidemic activity, accounting for 81.5% to 89.7% of subtyped Influenza A specimens in Week 1 and 91.2% (581 of 637 subtyped Influenza A isolates) in Week 2.[3][6] Deep sequencing by the CDC revealed that 90.4% of 659 genetically characterized H3N2 viruses collected since September 28, 2025 belong to Subclade K (J.2.4.1).[1][6] Emerging in late summer 2025 after selection of the 2025-2026 vaccine's A/Croatia/10136RV/2023 (2a.3a.1) strain, Subclade K carries hemagglutinin head mutations that reduce neutralizing antibody binding affinity.[1][9]

By comparison, Influenza A(H1N1)pdm09 accounted for only 56 of 637 (8.8%) subtyped Influenza A specimens in Week 2 (11.7% cumulatively since Week 40), confirming that the 2025-2026 surge remained an overwhelmingly H3N2-driven epidemic rather than pivoting to an H1N1 second wave.[6]

| Influenza A Subtype | | Week 1 Share | | Week 2 Share (n = 637) | | Epidemiological Trajectory |

| A(H3N2) [Subclade K / J.2.4.1] | | 81.5% - 89.7% | | 91.2% (581 isolates) | | Sustained dominant strain (90.4% Subclade K)[1][3][6] |

| A(H1N1)pdm09 | | 10.3% - 18.5% | | 8.8% (56 isolates) | | Minor co-circulating subtype (11.7% cumulative)[3][6] |

Simultaneously, surveillance reaffirmed a landmark in viral evolution: 100% of lineage-typed Influenza B isolates belonged to B/Victoria, with zero B/Yamagata detections.[3][6] Undetected since March 2020 after pandemic interventions depressed its basic reproduction number below 1.0, B/Yamagata's functional extinction validated the transition to a trivalent 2025-2026 vaccine.[9]

Clinical Severity, Hospitalization Demographics, and Pediatric Mortality

FluSurv-NET cumulative hospitalizations reached 40.6 per 100,000 population by Week 53 and 55.4 per 100,000 by Week 2.[5][6] Weekly admissions peaked in Week 52 at 12.8 per 100,000 -- the second-highest overall peak since 2010-2011, and the highest peak weekly hospitalization rate observed in children under 18 since 2010-2011 -- before easing to 8.4 per 100,000 in Week 1 and 17,963 NHSN hospital admissions in Week 2.[3][4][6] Cumulative admission rates followed a U-shaped age curve: adults 65 years led at 85.0 per 100,000 in Week 52-53 snapshots, followed by children 0-4 years at 32.6 per 100,000, and adults 18-49 years at 11.9 per 100,000.[7]

| Clinical Outcome / Cohort | | Cumulative Burden (Through Week 2, Jan 17, 2026) | | Hospitalization Rate (per 100,000) |

| Symptomatic Illnesses / Visits | | 19,000,000 Illnesses (18M / 9.3M visits through Week 1) | | N/A[3][6] |

| Total Hospitalizations | | 250,000 | | 55.4 (Overall Cumulative by Week 2)[3][6] |

| Geriatric (65 yr) / Pediatric (0-4 yr) | | Highest Risk Strata | | 85.0 (65 yr) / 32.6 (0-4 yr) [Week 52-53][7] |

| Cumulative Deaths (All / Pediatric) | | 10,000 Total / 44 Pediatric (12 in Week 2) | | 1.9% of Week 1 US deaths (NCHS)[3][5][6] |

Critically, 15 new pediatric deaths were reported in Week 1 and 12 more in Week 2, raising the season total to 44 pediatric fatalities.[3][6][8] Among vaccine-eligible children with known immunization status, approximately 90% of pediatric deaths occurred in those not fully vaccinated.[4][6] Even against drifted H3N2 Subclade K, vaccination primes cellular immunological (T-cell) responses that restrict lower-respiratory replication and secondary bacterial pneumonia.

Trivalent Vaccine Effectiveness and Co-Circulating Respiratory Pathogens

With ~130 million trivalent doses distributed in the US, early UK data indicate vaccine effectiveness against hospitalization of 70-75% in children and 30-40% in adults despite H3N2 Subclade K drift, while the A/Victoria/4897/2022 component closely matches circulating A(H1N1)pdm09.[2][6][9]

| 2025-2026 Trivalent Component | | Reference Virus Strain | | Match Against Early 2026 Circulation |

| Influenza A(H1N1)pdm09 | | A/Victoria/4897/2022-like | | High antigenic match to minor (8.8%) H1N1 share[6][9] |

| Influenza A(H3N2) | | A/Croatia/10136RV/2023-like | | Drifted vs. Subclade K (90.4%); 70-75% pediatric VE vs. admission[1][6][9] |

| Influenza B (Victoria / Yamagata) | | B/Austria/1359417/2021-like (Yamagata excluded) | | 100% match to typed B isolates; 0 Yamagata detections[3][6][9] |

Simultaneously, co-circulating respiratory viruses complicate inpatient triage across CDC integrated respiratory surveillance channels: SARS-CoV-2 and RSV activity required concurrent monitoring alongside influenza.[10][11]

Clinical Preparedness for an H3N2-Dominated Season

Although national clinical laboratory positivity decelerated across Weeks 1 and 2, elevated emergency department visits among school-aged children and persistent A(H3N2) Subclade K circulation maintained high pediatric severity.[3][6] Mitigating severe morbidity relies on three evidence-based interventions: (1) rapid multiplex molecular diagnostics to distinguish influenza A/B from SARS-CoV-2 and RSV;[10][11] (2) early initiation of neuraminidase or cap-dependent endonuclease antiviral therapy in hospitalized, geriatric, pregnant, and high-risk pediatric patients coupled with close monitoring for secondary bacterial superinfections (Staphylococcus aureus, Streptococcus pneumoniae); and (3) continued administration of the 2025-2026 trivalent influenza vaccine to protect against severe pediatric outcomes and co-circulating A(H1N1)pdm09 and B/Victoria lineages.[6][9]

Updates (Nov 16, 2026): Corrected a critical source mismatch in Reference 6 that previously cited the 2025 Week 2 FluView report (ending Jan 11, 2025) instead of the 2026 Week 2 report (ending Jan 17, 2026), replacing erroneous 2024-2025 subtype figures (43.1% H1N1 / 56.8% H3N2) with verified Week 2, 2026 CDC figures (91.2% A(H3N2), 8.8% A(H1N1)pdm09, 17.7% lab positivity, 44 pediatric deaths). Removed unreachable Reference 10 (MakeMyTestCount). Note that as of November 2026, CDC's final retrospective end-of-season burden estimates for 2025-2026 remain pending post-season release.

Works Cited

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