IRELAND INFLUENZA ACTIVITY 2025-2026

Virology Report prepared for the WHO Consultation on the Composition of Influenza Virus Vaccines for the Southern Hemisphere 2027

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Summary

- The 2025–2026 influenza season in Ireland was characterised by an earlier-than-usual onset, with influenza activity rising above baseline levels in early November 2025 (week 45) and peaking during weeks 50–51/2025. Following the peak, influenza activity declined steadily through January and February 2026. By week 20/2026, influenza activity had returned to baseline levels, consistent with the end of sustained seasonal circulation. The season was associated with a substantial burden of hospitalisation and severe disease, particularly among older adults.

- In total, 24,948 laboratory-confirmed influenza cases were notified nationally, with the highest incidence observed in adults aged ≥80 years and children aged <5 years. Across the season, 5,935 hospitalised influenza cases, 183 ICU admissions and 301 deaths among laboratory-confirmed influenza cases were reported.

- Virologically, the 2025/26 season was dominated by influenza A(H3N2). Of 697 influenza-positive specimens genetically characterised at NVRL, 617 (88.5%) were A(H3), 72 (10.3%) were A(H1N1)pdm09 and eight (1.1%) were B/Victoria. Most A(H3) viruses (585/617; 94.8%) were subclade K (2a.3a.1), represented by A/Norway/8765/2025, consistent with the dominant A(H3N2) subclade reported across the EU/EEA.

- Antiviral susceptibility remained largely preserved. Neuraminidase susceptibility interpretations were available for 632/697 (90.7%) viruses, with all showing normal inhibition to oseltamivir and zanamivir. Six viruses carried PA substitutions associated with reduced susceptibility to baloxavir marboxil.

- Overall, the 2025/26 influenza season was characterised by an early onset and December peak, substantial clinical burden and marked predominance of A(H3N2) subclade K viruses. The virological pattern broadly mirrored that observed across the EU/EEA, highlighting the importance of continued integrated epidemiological and genomic surveillance.

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INTRODUCTION

Human influenza A and B viruses are the principal causes of seasonal influenza epidemics in humans. Influenza A viruses are defined by combinations of haemagglutinin (HA) and neuraminidase (NA) surface glycoproteins, with A(H1N1)pdm09 and A(H3N2) currently circulating. Influenza B viruses are of the B/Victoria lineage, while sustained detection of B/Yamagata has not been demonstrated in recent years. Seasonal influenza imposes a substantial global burden each year, causing millions of clinically apparent infections, with an estimated 3–5 million cases of severe illness and 290,000–650,000 deaths, disproportionately affecting older adults, young children, pregnant women, the immunocompromised, and those with underlying conditions. [1]

Prevention relies primarily on vaccination, complemented by antiviral therapeutics (neuraminidase inhibitors and the cap-dependent endonuclease inhibitor baloxavir marboxil) for treatment and outbreak control. Antigenic drift and waning immunity necessitate periodic updates to vaccine composition. The World Health Organization (WHO) convenes biannual vaccine composition meetings (VCMs), informed by antigenic and genomic data generated within the Global Influenza Surveillance and Response System (GISRS), to recommend strains for inclusion in Northern and Southern Hemisphere vaccines. [2, 3]

Continuous, integrated genomic and antigenic surveillance is essential to detect emerging variants, monitor clade dynamics, and assess antiviral susceptibility. Within GISRS, National Influenza Centres (NICs) undertake national laboratory surveillance, generate and share viruses and sequence data, and forward representative specimens to WHO Collaborating Centres (CCs) for detailed characterisation. [2, 4]

The National Virus Reference Laboratory (NVRL, Dublin) is Ireland's WHO-recognised NIC. NVRL provides national reference diagnostics, subtyping, sequencing and genetic/antigenic characterisation and forwards representative viruses to the WHO CC in London (Francis Crick Institute) in line with GISRS protocols. In collaboration with the Health Protection Surveillance Centre (HPSC), and the Irish College of General Practitioners (ICGP), NVRL underpins Ireland's integrated influenza surveillance system through: the sentinel general-practice surveillance programme (ILI/ARI swabbing for qPCR at NVRL), the Severe Acute Respiratory Infections (SARI) sentinel hospital network (confirmatory testing and characterisation of influenza-positive admissions), and routine laboratory reporting that informs HPSC's Integrated Respiratory Virus Bulletin and Data Hub. [5]

NVRL also contributes to multi-country evaluation platforms, including European Centre for Disease Prevention and Control (ECDC)-coordinated Vaccine Effectiveness, Burden and Impact Studies (VEBIS) in hospital SARI settings, and collaborates, where relevant, with European Influenza Monitoring Vaccine Effectiveness in Europe (I-MOVE) studies of influenza vaccine effectiveness in primary care and hospitals. Data from these programmes — together with NVRL outputs – are shared with ECDC and WHO to inform national and international situational assessments, vaccine-performance evaluation and preparedness. [6, 7]

This report describes the genetic characterisation of seasonal influenza viruses detected in Ireland during the 2025/2026 winter season, using whole-genome and HA/NA-focused analyses performed at the NVRL. The analyses correspond to specimens in the characterised dataset spanning weeks 40/2025 through 19/2026 enabling national monitoring of lineage/clade turnover, potential antigenic drift, and markers of antiviral resistance, in alignment with GISRS/ECDC assessments.

METHODS

Specimen testing and subtyping/lineage assignment

Respiratory specimens received for routine screening were tested using the NxTAG® Respiratory Pathogen Panel + SARS-CoV-2 assay (Luminex/MAGPIX), which detects influenza A (with subtype discrimination for H1 and H3) and influenza B, alongside other respiratory targets. Specimens referred to the NVRL for further characterisation underwent a laboratory-developed RT-qPCR procedure that confirmed influenza A subtypes A(H1N1)pdm09 and A(H3N2) and assigned influenza B viruses to lineage level (B/Victoria, B/Yamagata). Samples were deemed eligible for sequencing when they met pre-specified analytical criteria (NxTAG MDD ≥ 300 or RT-qPCR $C_t \leq 25$).

Whole-genome sequencing (WGS)

Eligible influenza-positive extracts were prepared using the Illumina Microbial Amplicon Prep—Influenza A/B (IMAP-Flu) workflow, an amplicon-based whole-genome protocol that targets all eight influenza segments and incorporates bead-linked tagmentation and unique dual indices. Libraries were pooled and sequenced on an Illumina NextSeq 1000 rendering a 2 × 150 bp paired-end run on a P1 flow cell with XLEAP-SBS chemistry.

Bioinformatics and quality control

FASTQ files were processed through the INSaFLU web platform, which performs reference-guided assembly, consensus generation, variant annotation and phylogenetic outputs tailored for influenza genomic surveillance. Consensus HA (and NA, when recovered) sequences were retained for downstream analyses; sequences failing platform QC checks or lacking unambiguous HA calls were excluded from clade assignment. Clades were determined using Nextclade and sequences were checked for amino acid substitutions associated with antiviral resistance or reduced susceptibility using GISAID FluSurver.

OVERVIEW OF THE 2025/26 SEASON

National surveillance indicated an earlier-than-usual influenza season in Ireland in 2025/26. Influenza activity increased during Autumn 2025 and peaked during weeks 50–51/2025. Activity declined following the December peak and remained at baseline levels by the end of the surveillance period. In week 20/2026, influenza incidence was 0.4 per 100,000 population, with 20 laboratory-confirmed cases notified nationally. [5]

Across the 2025/26 season, 24,948 laboratory-confirmed influenza cases were notified nationally, corresponding to a cumulative incidence of 484.5 per 100,000 population. The highest incidence was observed among adults aged >80 years (2,368.2 per 100,000), followed by children aged <1 year (1,488.0 per 100,000) and 1–4 years (1,287.8 per 100,000). The median age of notified influenza cases was 42 years (IQR: 12–74). Females accounted for 13,609 cases and males for 11,325 cases. [5]

The season was associated with a substantial burden of severe disease. A total of 183 influenza-associated ICU admissions were reported between week 40/2025 and week 20/2026, representing 3.1% of hospitalised influenza cases. In addition, 301 deaths among laboratory-confirmed influenza cases were reported, of which 271 (90.0%) occurred in adults aged ≥65 years. Excess pneumonia- and influenza-related mortality was observed for six consecutive weeks, from week 52/2025 to week 5/2026. [5]

Influenza A(H3) was the predominant influenza A subtype detected during the season. Among NVRL sentinel GP and non-sentinel respiratory specimens, 3,460 influenza-positive specimens were detected during the season to date, comprising 3,432 influenza A and 28 influenza B viruses. Among subtyped influenza A viruses, A(H3) predominated (n=2,868), compared with A(H1)pdm09 (n=520). This predominance of A(H3) was also reflected in the viruses selected for genetic characterisation at NVRL.

By ISO week 21/2026, influenza circulation had declined to very low levels. No influenza-positive specimens were detected through sentinel GP surveillance in weeks 20 or 21, while two influenza-positive non-sentinel respiratory specimens were detected in each of these weeks. Influenza positivity among SARI cases was below 10% in ISO week 21.

Overall, the 2025/26 influenza season in Ireland was characterised by an early peak in December 2025, substantial clinical burden and marked predominance of A(H3) viruses. Activity declined substantially following the peak and was at baseline levels by the end of the surveillance period. Continued integration of primary-care, hospital-based and laboratory surveillance supports timely assessment of seasonal severity, circulating influenza types/subtypes and the emergence of genetically-distinct viruses.

Table 1: Notified laboratory-confirmed cases of influenza by age, sex and HSE health region, from week 40/2025, to week 20/2026 Data source: HPSC. [5]

Number of Influenza cases (incidence per 100,000 population)	
Cases	24,948 (484.5)
Age groups (years)	
<1	860 (1,488.0)
1–4	3,060 (1,287.8)
5–14	3,020 (421.3)
15–44	5,899 (285.4)
45–64	3,131 (242.1)
65–79	4,689 (787.7)
>80	4,287 (2,368.2)
Median age (IQR)	42 (12–74)
Sex	
Male	11,325 (445.1)
Female	13,609 (522.5)
HSE Health Regions	
Dublin and North East	5,721 (548.2)
Dublin and Midlands	4,533 (420.6)
Dublin and South East	5,637 (580.5)
South West	3,416 (461.2)
Mid-West	1,776 (430.0)
West and North West	3,863 (508.5)

Figure 1: Number of notified cases of laboratory-confirmed influenza infection by notification week in Ireland between week 40/2025 and week 20/2026 Data source: HPSC. [5]

Genetic Characterisation of Influenza Viruses- Ireland

Genetic characterisation:

- A total of 697 influenza-positive samples were genetically characterised by whole-genome sequencing directly from original clinical specimens, comprising 196 sentinel General Practice specimens, 242 SARI sentinel hospital specimens and 259 other non-sentinel specimens.

- Of the 697 characterised viruses, 617 (88.5%) were A(H3N2), 72 (10.3%) were A(H1N1)pdm09 and eight (1.1%) were B/Victoria.

- Haemagglutinin sequences were obtained for 697/697 (100%) viruses and used for clade/subclade assignment, interpreted against the relevant ECDC/TESSy influenza virus characterisation guidance for the 2025–2026 Northern Hemisphere season. [8]

- Neuraminidase antiviral genotypic susceptibility interpretations were available for 632/697 (90.7%) viruses, comprising 70/72 A(H1N1)pdm09 and 562/617 A(H3N2) viruses. All 632 were reported as having amino acid-based normal inhibition (AANI) to both oseltamivir and zanamivir, with no established high-level neuraminidase-inhibitor resistance identified.

- Six A(H3N2) viruses contained PA substitutions interpreted as amino acid-based reduced susceptibility (AARS) to the cap-dependent endonuclease inhibitor baloxavir marboxil. All six contained the PA-I38L substitution, supporting continued surveillance of antiviral genotypic susceptibility markers.

Table 2: Sequenced samples by Influenza Subtype, Sample source and patient characteristics

Sample source	A(H1N1)pdm09	A(H3N2)	B/Victoria	Total
Sentinel	6 (8%)	184 (30%)	6 (75%)	196 (28%)
Non-sentinel	44 (61%)	213 (35%)	2 (25%)	259 (37%)
SARI	22 (31%)	220 (36%)	0 (0%)	242 (35%)
Total	72	617	8	697

Age, Years	A(H1N1)pdm09	A(H3N2)	B/Victoria	Total
0–4	10 (14%)	133 (22%)	1 (13%)	144 (21%)
5–18	7 (10%)	136 (22%)	2 (25%)	145 (21%)
19–44	11 (15%)	103 (17%)	4 (50%)	118 (17%)
45–64	10 (14%)	62 (10%)	1 (13%)	73 (10%)
65+	34 (47%)	183 (30%)	0 (0%)	217 (31%)
Total	72	617	8	697

Sex	A(H1N1)pdm09	A(H3N2)	B/Victoria	Total
Female	37 (51%)	345 (56%)	4 (50%)	386 (55%)
Male	35 (49%)	271 (44%)	4 (50%)	310 (49%)
Total	72	617	8	697

Influenza A(H1)pdm09 viruses

Whole-genome/HA analyses characterised 72 A(H1N1)pdm09 viruses, representing 10.3% (72/697) of all influenza viruses sequenced during the 2025/26 season. All 72 were assigned to the 5a.2a.1 (D) genetic group represented by A/Missouri/11/2025, with D.3.1 viruses comprising the A(H1N1)pdm09 population detected in Ireland. A/Missouri/11/2025 is the reference virus for subclade D.3.1 in the 2025/26 ECDC/WHO characterisation guidance. [8]

The D.3.1 subclade is characterised by the 5a.2a.1 (D) background substitutions together with T120A, I372V, I460T and V520A. The ECDC/WHO guidance identifies A/Missouri/11/2025 as the D.3.1 reference virus and as the recommended A(H1N1)pdm09 vaccine virus for the 2026 Southern Hemisphere season. [10]

This represented a marked change from the 2024/25 Irish season, when A(H1N1)pdm09 viruses were predominantly assigned to 5a.2a (C.1.9). In 2025/26, the characterised A(H1N1)pdm09 population was instead represented entirely by the 5a.2a.1 D.3.1/Missouri genetic reporting group, consistent with the emergence and expansion of this group internationally.

Neuraminidase antiviral susceptibility interpretations were available for 70/72 (97.2%) A(H1N1)pdm09 viruses. All 70 were reported as showing amino acid-based normal inhibition (AANI) to oseltamivir and zanamivir, with no established high-level neuraminidase-inhibitor resistance identified. The NA-S247N substitution was detected in 14 A(H1N1)pdm09 viruses. This substitution has been associated with reduced inhibition by oseltamivir in some contexts, but the viruses in the final dataset remained classified as AANI. Continued monitoring of this substitution is therefore warranted.

Overall, A(H1N1)pdm09 represented a relatively small component of influenza circulation in Ireland during 2025/26, with A(H3N2) substantially predominating. Continued genomic surveillance is important to monitor further diversification of the D.3.1 lineage and for the emergence of antiviral susceptibility markers.

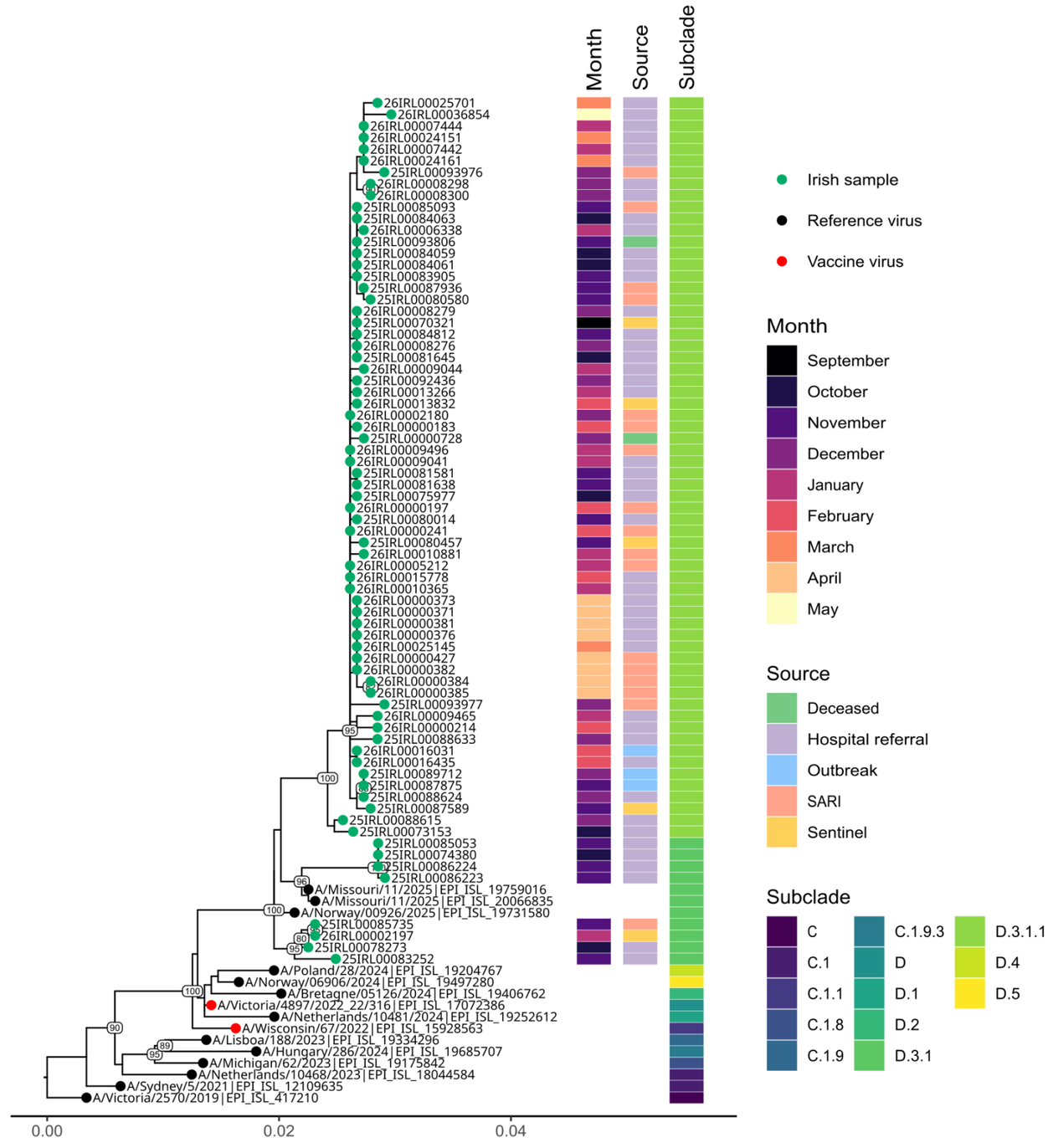

Figure 2: Maximum-likelihood HA phylogeny of Irish influenza A(H1N1)pdm09 viruses characterised during 2025/26 (n=72), shown with contemporary reference and vaccine viruses. Side bars indicate month, submission source and subclade. Scale bar shows substitutions per site. Bootstrap support values are shown at tree nodes.

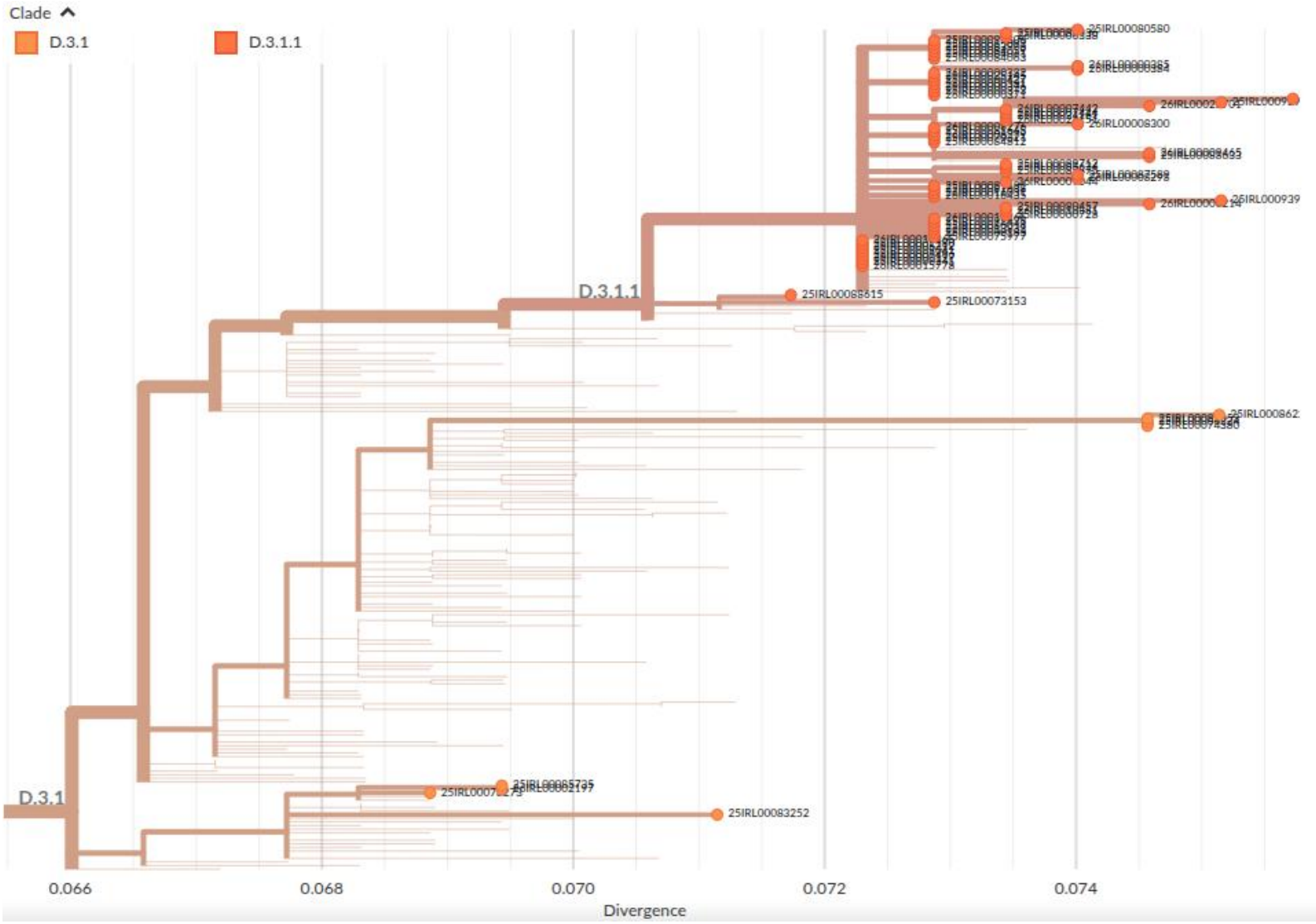

Figure 3: Nextclade phylogenetic tree of Irish influenza A(H1N1)pdm09 HA sequences characterised during 2025/26 (n=72). Irish viruses are shown as filled circles; colours indicate subclade assignment. Viruses clustered within D.3.1, including D.3.1.1. The x-axis represents genetic divergence.

Influenza A(H3) Viruses

Whole-genome/HA analyses characterised 617 A(H3N2) viruses, representing 88.5% (617/697) of all influenza viruses sequenced during the 2025/26 season. All belonged to clade 2a.3a.1, with the vast majority (585/617; 94.8%) assigned to the newly emerged subclade K (formerly J.2.4.1), represented by A/Norway/8765/2025. The remaining viruses were represented by A/Croatia/10136RV/2023 (J.2; n=19), A/Singapore/GP20238/2024 (J.2.4; n=12) and A/Netherlands/10685/2024 (J.2.3; n=1). The 2025/26 ECDC/WHO characterisation guidance defines subclade K by the additional HA substitutions K2N, S144N, N158D, I160K, Q173R, T328A and S378N and identifies A/Norway/8765/2025 as its reference virus. [8]

The marked predominance of subclade K in Ireland was consistent with the wider European pattern during 2025/26. Across the EU/EEA, 92% of genetically characterised A(H3N2) viruses belonged to subclade K, demonstrating the rapid emergence and expansion of this group during the season. [11]

Neuraminidase antiviral genotypic susceptibility interpretations were available for 562/617 (91.1%) of A(H3N2) viruses. All were reported as showing amino acid-based normal inhibition (AANI) to oseltamivir and zanamivir, with no established high-level neuraminidase-inhibitor resistance identified. However, six A(H3N2) viruses carried the PA-I38L substitution, which was interpreted as amino acid-based reduced susceptibility (AARS) to the cap-dependent endonuclease inhibitor baloxavir marboxil. Continued surveillance of antiviral target genes is therefore warranted.

The predominance of subclade K also represented a notable genetic change relative to the A(H3N2) component recommended for the 2025/26 Northern Hemisphere vaccine. The recommended A(H3N2) components were A/Croatia/10136RV/2023-like (egg-) and A/District of Columbia/27/2023-like (cell- or recombinant-based) viruses, both belonging to the J.2 genetic group. [9] The subsequent emergence and rapid predominance of subclade K therefore highlights the continuing genetic evolution of A(H3N2) viruses and the importance of genomic and antigenic surveillance for informing future vaccine strain selection.

Overall, the Irish A(H3N2) population during 2025/26 was characterised by strong predominance of the newly-emerged K subclade, closely mirroring the pattern observed across the EU/EEA. Continued genomic, antigenic and antiviral susceptibility surveillance is warranted to monitor further diversification of this rapidly expanding group.

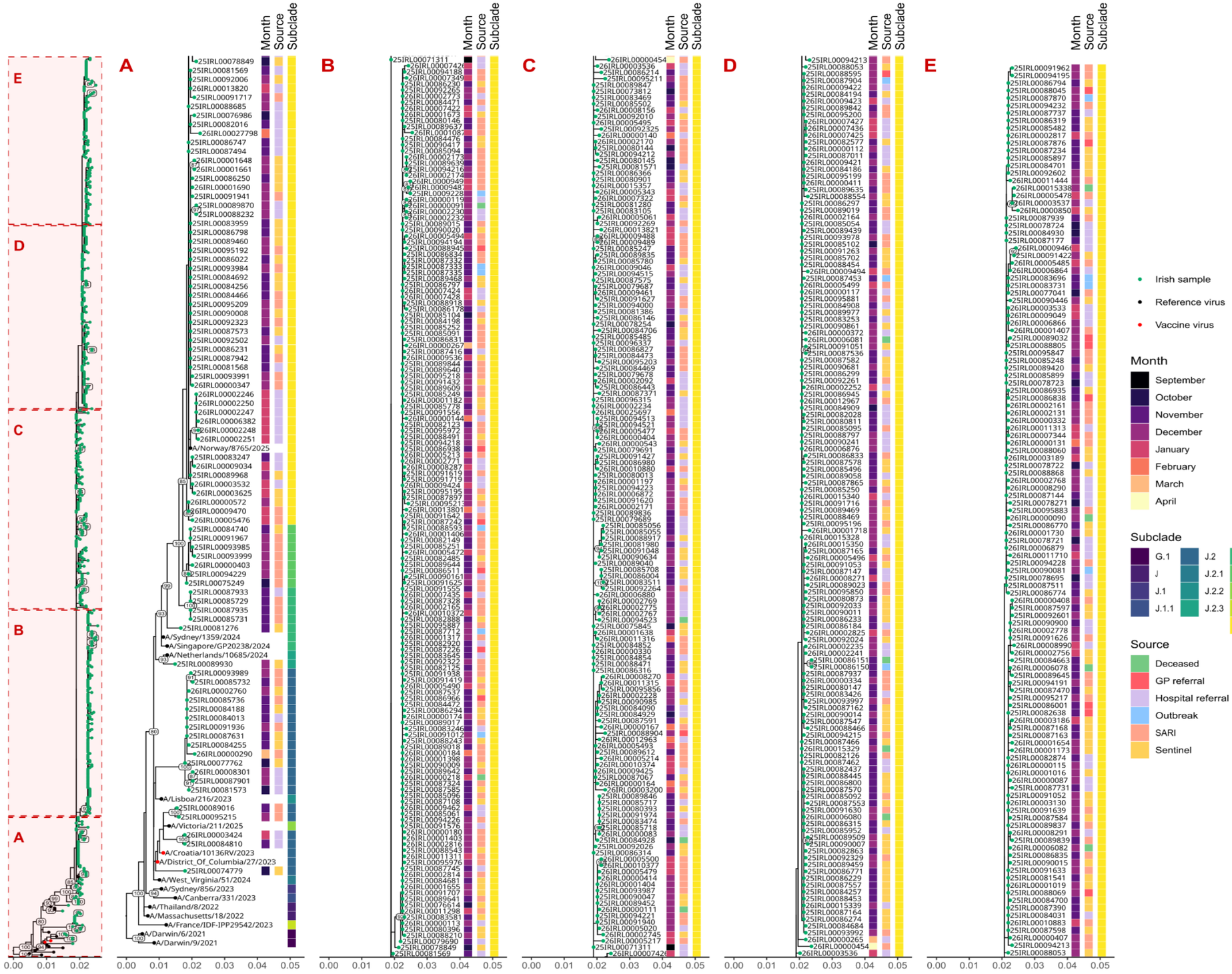

Figure 4: Maximum-likelihood HA phylogeny of Irish influenza A(H3N2) viruses characterised during 2025/26 (n=617), shown with contemporary reference and vaccine viruses. The tree is displayed in panels A-E for readability; side bars indicate month, submission source and subclade. Scale bar shows substitutions per site. Bootstrap support values are shown at tree nodes.

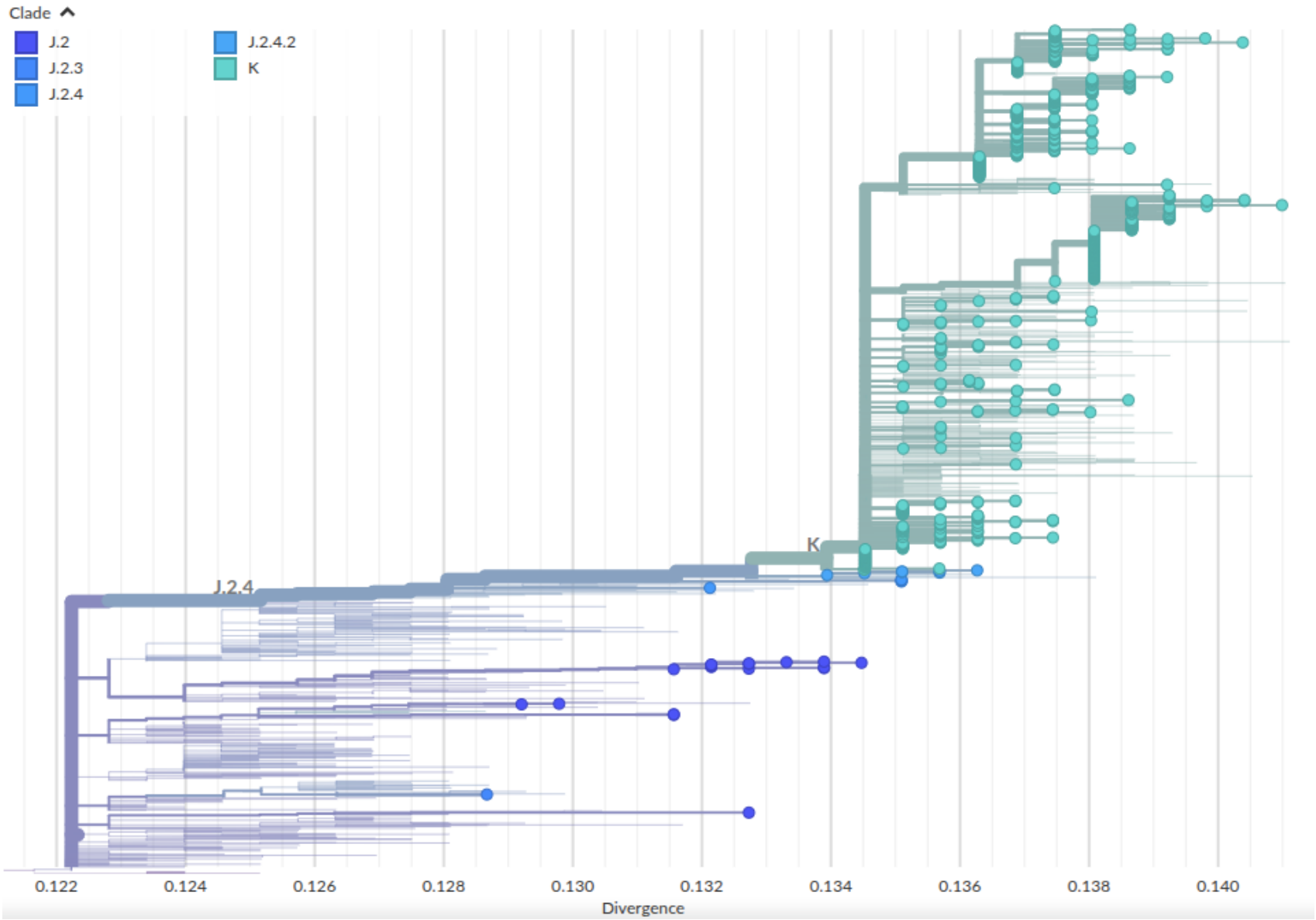

Figure 5: Nextclade phylogenetic tree of Irish influenza A(H3N2) HA sequences characterised during 2025/26 (n=617). Irish viruses are shown as filled circles against international sequence context; colours indicate Nextclade assignment. Irish sequences clustered predominantly within subclade K. The tree also shows J.2, J.2.3, J.2.4 and J.2.4.2 genetic context. The x-axis represents genetic divergence.

Influenza B Victoria lineage viruses

Haemagglutinin sequences were obtained from eight B/Victoria lineage viruses, representing 1.1% (8/697) of all influenza viruses genetically characterised during the 2025/26 season. All belonged to the V1A.3a.2 genetic group, although several subclades were represented. Three viruses were assigned to C.5.6.1, represented by B/ENG/120/2025; two to C.3.1, represented by B/Kanagawa/AC2414/2025; one to C.5, represented by B/Stockholm/3/2022; and one to C.5.6, represented by B/Switzerland/329/2024. One additional B/Victoria virus was assigned to a recognised subgroup not otherwise listed in the reporting categories. The ECDC/WHO 2025/26 guidance identifies these reference viruses within the contemporary B/Victoria genetic reporting framework. [8]

The characterised B/Victoria viruses therefore showed greater genetic diversity despite their small number, with no single subclade accounting for the entire Irish B/Victoria population. The 2025/26 Northern Hemisphere vaccine contained a B/Austria/1359417/2021-like virus, belonging to the broader V1A.3a.2 (C) group, for both egg-, cell- or recombinant-based vaccines. [9]

No neuraminidase antiviral susceptibility interpretations were available for the eight B/Victoria viruses in the final characterised dataset. Therefore, no conclusions regarding neuraminidase-inhibitor susceptibility should be drawn from the Irish B/Victoria sequence dataset for 2025/26.

Overall, B/Victoria circulation was limited in Ireland during 2025/26, accounting for only eight of the 697 genetically characterised viruses. Continued genomic surveillance is imperative to monitor the evolution of contemporary B/Victoria subclades to detect any changes in antigenic characteristics or antiviral susceptibility.

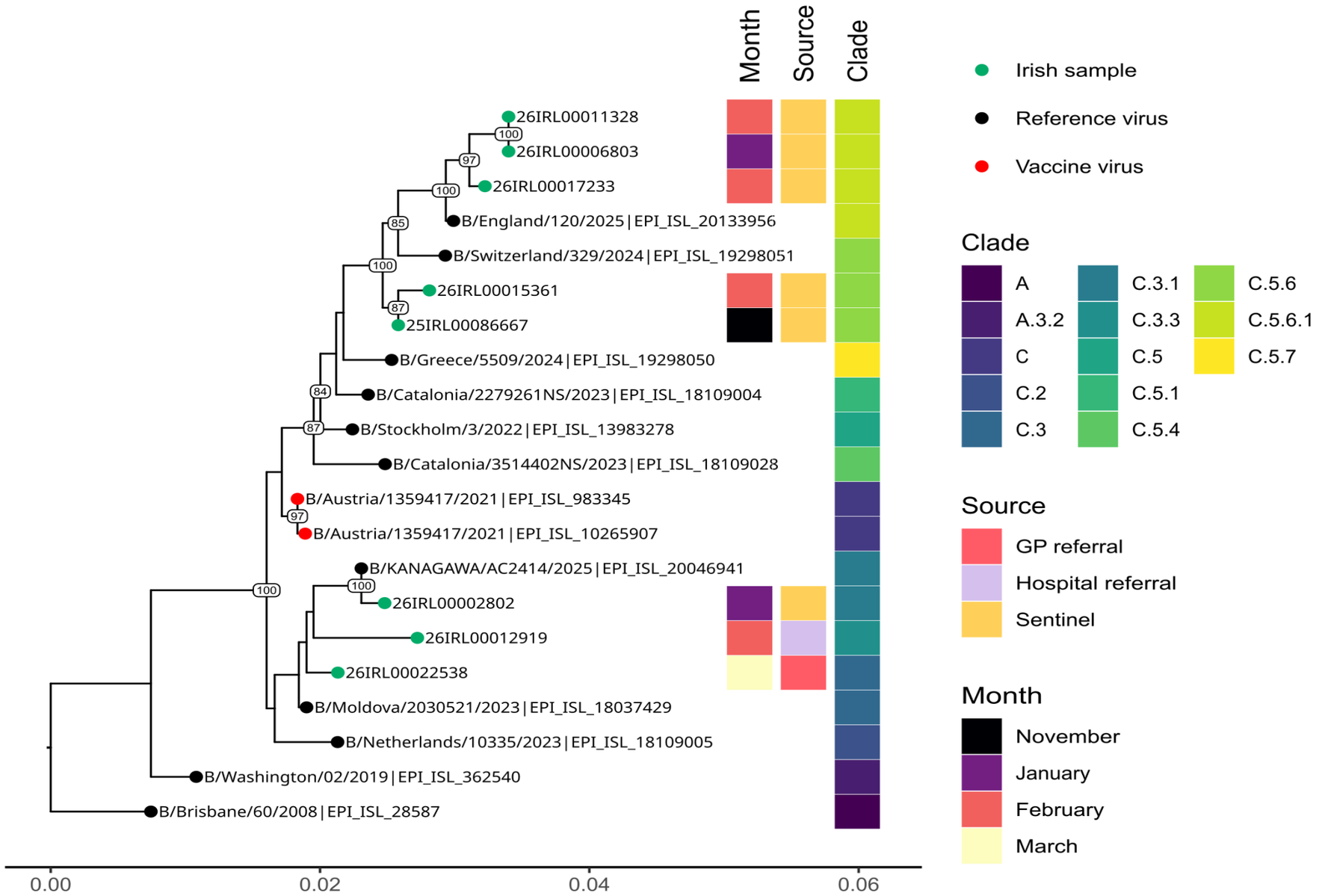

Figure 6: Maximum-likelihood HA phylogeny of Irish influenza B/Victoria lineage viruses characterised during 2025/26 (n=8), shown with contemporary reference and vaccine viruses. Side bars indicate month, submission source and clade. Scale bar shows substitutions per site. Bootstrap support values are shown at tree nodes.

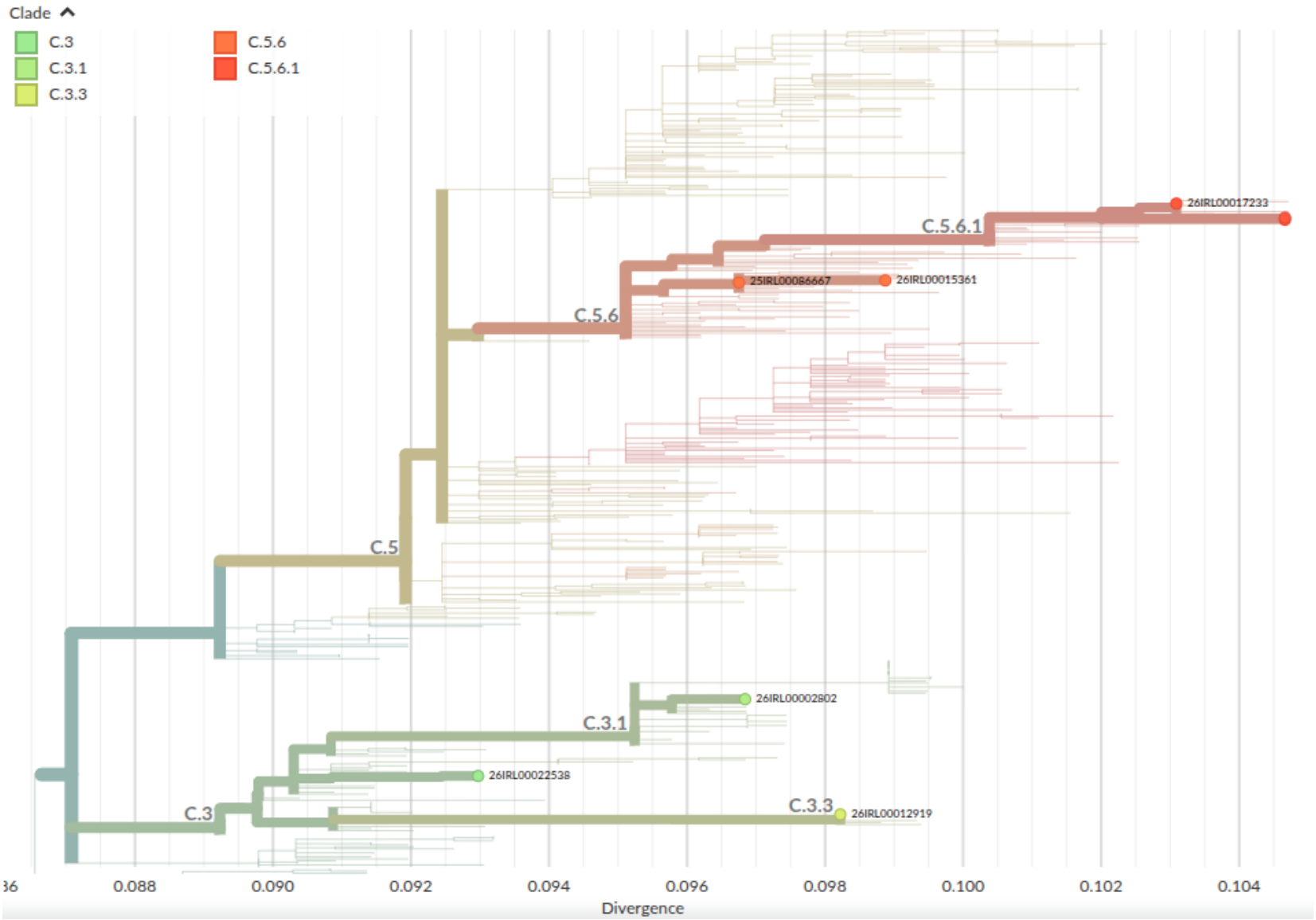

Figure 7: Nextclade phylogenetic tree of Irish influenza B/Victoria HA sequences characterised during 2025/26 (n=8). Irish viruses are shown as filled circles; colours indicate subclade assignment. Irish viruses occurred in C.3.1, C.3.3, C.5.6 and C.5.6.1. The x-axis represents genetic divergence.

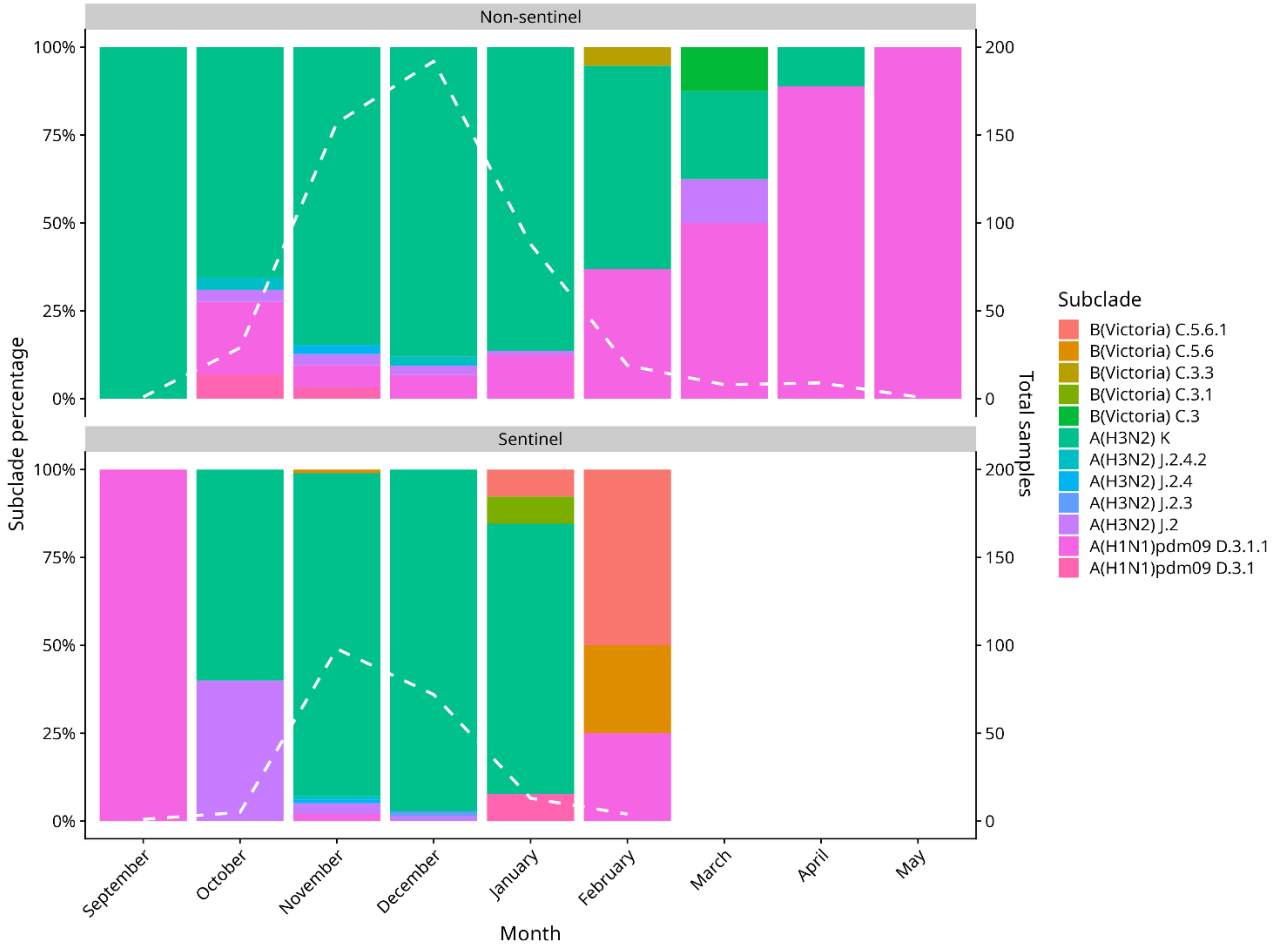

Figure 8: Monthly % composition of influenza HA subclades in Ireland, 2025/26, split by Non-sentinel (top) and Sentinel (bottom). Stacked bars show subclade shares (colours); white dashed line = number sequenced per month (right y-axis).

Conclusions

Influenza activity in Ireland began earlier than usual during the 2025/26 season, rising above baseline in early November 2025 and peaking during weeks 50–51/2025. Activity declined following the December peak and was at baseline levels by the end of the surveillance period. The season was associated with a substantial burden of severe disease, including 183 ICU admissions and 301 deaths among laboratory-confirmed influenza cases, with older adults particularly affected. [5]

Genetically, the season was strongly dominated by A(H3N2), which accounted for 617/697 (88.5%) characterised viruses. The majority of A(H3N2) viruses (585/617; 94.8%) belonged to the newly-emerged subclade K (2a.3a.1; former J.2.4.1), represented by A/Norway/8765/2025. This closely mirrored the wider EU/EEA pattern, where subclade K also became the predominant A(H3N2) group. [11]

A(H1N1)pdm09 accounted for 72/697 (10.3%) viruses and was represented by the 5a.2a.1 D.3.1/A/Missouri/11/2025 genetic group, while only eight B/Victoria viruses (1.1%) were characterised. The ECDC/WHO 2025/26 guidance identifies A/Missouri/11/2025 as the D.3.1 reference virus and A/Norway/8765/2025 as the reference virus for the newly emerged K subclade. [8]

Antiviral susceptibility remained largely preserved. Neuraminidase genotypic susceptibility interpretations were available for 632/697 (90.7%) viruses, all of which showed amino acid-based normal inhibition (AANI) to oseltamivir and zanamivir, with no established high-level neuraminidase-inhibitor resistance identified. However, six A(H3N2) viruses carried the PA-I38L substitution, interpreted as amino-acid-based reduced susceptibility to baloxavir marboxil, highlighting the importance of continued surveillance of antiviral target genes.

Overall, the 2025/26 season demonstrated the capacity for rapid changes in the genetic composition of circulating influenza viruses, particularly the emergence and predominance of A(H3N2) subclade K. Continued integration of epidemiological, genomic and antigenic surveillance will be important for monitoring viral evolution, informing vaccine-strain selection and detecting changes in antiviral susceptibility. Priorities should include maintaining timely and representative sampling across surveillance streams, maximising whole-genome sequence recovery and continuing collaboration with WHO, ECDC and European surveillance networks.

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We also acknowledge the support and technical advice of the WHO Collaborating Centre at the Francis Crick Institute, London, and the wider GISRS/ERLI-Net network for reference materials, antigenic outputs, and methodological guidance.

For weekly epidemiological updates and dashboards, please refer to the HPSC Integrated Respiratory Virus reports: https://respiratorydisease-hpscireland.hub.arcgis.com/

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