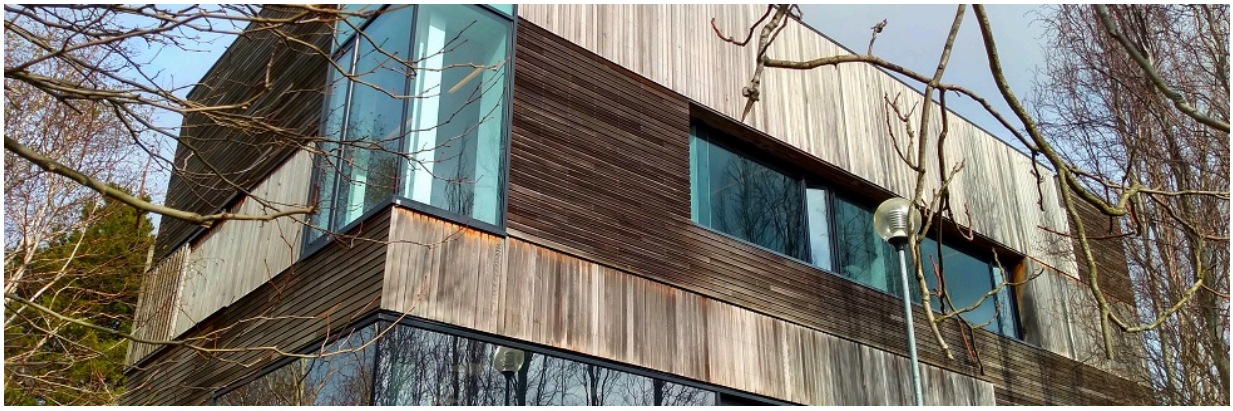

Genetic Characterisation of Respiratory Syncytial Virus (RSV) in Ireland

2025/2026 season

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Ireland

23rd September 2026

SUMMARY

- Both RSV-A and -B antigenic subgroups circulated in Ireland with near equal frequency in the winter season, 2025/2026.
- The NVRL sequenced 337 RSV samples, almost evenly split between RSV-A (53%) and RSV-B (47%).
- Samples were predominantly from individuals less than five years of age (58%), a cohort that was more frequently observed to be infected with RSV-A than RSV-B.
- Genetic lineages A.D.1.4, A.D.3.7, B.D.E.1, and B.D.E.1.2 were most prevalent.
- Irish RSV lineage dynamics were distinct from the rest of Europe; for instance, lineage A.D.3.11 comprised >35% of European cases but <10% in Ireland.
- No evidence suggests the passive immunisation programme was negatively impacted by circulating RSV genetic diversity; however, known nirsevimab resistance-associated mutations (I64T and P205S) were observed at a low frequency.
- Ongoing genomic surveillance is imperative to monitor RSV lineage dynamics and to evaluate binding site mutations in the context of continued population-scale immunisation strategies to mitigate disease.

CONTENTS

INTRODUCTION

Human respiratory syncytial virus (RSV) possesses two major antigenic subgroups (previously subtypes), termed RSV-A and -B. Both RSV subgroups exert a profound burden of lower respiratory tract infection, with an estimated incidence of about 33 million cases per annum in children under five years of age (Li *et al.*, 2022). Paediatric cohorts most susceptible to severe RSV disease include pre-term infants, neonates, and individuals less than one year of age. Importantly, the elderly and the immunocompromised of all ages are also severely affected and, while less common than influenza and COVID-19, RSV clinical disease is now thought to be greater in geriatric groups (Surie *et al.*, 2023). With the recent development and availability of both prophylactic vaccines and monoclonal antibodies, and the licensing of antiviral therapies in an increasing number of countries, this severe disease burden of morbidity and mortality is anticipated to lessen (Terstappen *et al.*, 2024).

The medium- and long-term effects of these pharmaceutical interventions on viral evolution and susceptibility to various treatment options are yet to be determined. In addition, continuous changes in both viral diversity and lineage turnover and replacement, arising primarily due to population-level immunity, necessitate public health surveillance employing viral pathogen genomics to enable an evidence-based assessment on possible impacts on RSV infections and to monitor for the possible emergence of vaccine-escape mutations (Rice *et al.*, 2025). Therefore, it has been recommended by the World Health Organization (WHO) to expand existing respiratory viral surveillance systems for influenza and SARS-CoV-2 to include RSV (World Health Organization, 2025).

This report describes the genetic characterisation of RSV by whole genome sequencing (WGS) in samples referred to the National Virus Reference Laboratory during the 2025/2026 winter season. This work builds on data from past seasons, to enable national monitoring of changes in the viral genome over time and as treatments are licensed.

METHODS

Respiratory samples received for routine screening were tested using the NxTAG Respiratory Pathogen Panel + SARS-CoV-2 assay (Luminex), which detects and discriminates between the RSV-A and -B subgroups. Additionally, samples referred to the NVRL for further characterisation were tested using a laboratory-developed reverse transcription quantitative polymerase chain reaction (qRT-PCR), which also allowed discrimination between RSV-A and -B. Samples with viral titres yielding Luminex multidimensional data (MDD) ≥ 300 or qRT-PCR $C_t \leq 25$ results were selected for sequence analysis. Samples meeting the case definition for severe acute respiratory infection (SARI), samples from infants admitted to paediatric intensive care units (PICU), and samples from individuals known to have previously received nirsevimab were also considered for sequencing regardless of MDD or C_t values.

Laboratory-confirmed RSV RNA-positive samples were processed for WGS with a next-generation sequencing (NGS) approach employing the New England Biolab NEBNext RSV Primer Module upstream of a tagmentation step and Illumina DNA library preparation. Sequencing was performed on the Illumina NextSeq 1000 platform using a P1 flow cell generating 2 × 150 bp paired-end reads. Reference-based genome assembly was performed using the INSaFLU platform (Santos *et al.*, 2024) with reference hRSV/A/England/397/2017 (EPI_ISL_412866) for RSV-A samples and reference hRSV/B/Australia/VIC-RCH056/2019 (EPI_ISL_1653999) for RSV-B samples. Samples were considered to be successfully sequenced if at least 85% of the genome was covered by a depth of 10 or more reads. Genome sequences generated were made publicly available on GenBank, Pathoplexus, and GISAID repositories.

For the purposes of this report, sequences were aligned using MAFFT (Katoh & Standley, 2013) and phylogenetic trees generated using IQTREE (Minh *et al.*, 2020). Trees were rooted using Gotree (Lemoine & Gascuel, 2021) and lineage nomenclature proposed by the RSV Genotyping Consensus Consortium (Goya *et al.*, 2024) was assigned using the Nextclade RSV datasets last updated on 2026-04-14 (Aksamentov *et al.*, 2021).

RESULTS

Sources of samples

A representative subset of suitable respiratory samples with RSV RNA detected by molecular methods underwent further genetic characterisation by WGS. Samples were referred to the NVRL from the following sources (Table 1):

1. GP Sentinel Surveillance Programme (n=109 successfully sequenced): representing samples with RSV RNA detected collected from patients attending general practice with clinical presentations meeting the case definitions for acute respiratory infection (ARI) and/or influenza-like illness (ILI).
2. SARI surveillance (n=209): samples meeting the case definition for SARI from the designated SARI hospital sites (St Vincent's University Hospital, St James's Hospital, University Hospital Limerick, and Children's Health Ireland at Crumlin).
3. Nirsevimab breakthrough infections (n=3): samples from individuals known to have previously received nirsevimab.
4. Typing request (n=16): samples submitted on the basis of a request from Public Health to acute hospital laboratories for characterisation of RSV samples from hospitalised patients.

Table 1: Successfully sequenced samples by RSV subgroup, sample source, and patient characteristics.

Sample source	RSV-A	RSV-B	Total
Sentinel	38 (21.5%)	71 (44.4%)	109
SARI	128 (72.3%)	81 (50.6%)	209
Nirsevimab breakthrough infection	3 (1.7%)	0 (0.0%)	3
Typing request	8 (4.5%)	8 (5.0%)	16
Sex			
Female	100 (56.5%)	79 (49.4%)	179
Male	77 (43.5%)	81 (50.6%)	158
Age			
<6 months	33 (18.6%)	19 (11.9%)	52
6-11 months	17 (9.6%)	13 (8.1%)	30
1-4 years	75 (42.4%)	39 (24.4%)	114
5-17 years	11 (6.2%)	10 (6.3%)	21
18-64 years	19 (10.7%)	38 (23.8%)	57
65+ years	22 (12.4%)	41 (25.6%)	63
Total	177	160	337

The majority of samples (62%) sequenced from the 2025/2026 winter season were sourced from SARI surveillance samples submitted to the NVRL, followed by samples referred through the GP Sentinel Surveillance programme (32%). Patients under five years of age contributed 196 (58%) of the total sequenced samples. A third of this cohort were patients born between March 2025 and February 2026 and therefore eligible for nirsevimab administration through the HSE RSV Immunisation Pathfinder Programme, either by immunisation during the season or by catch-up immunisation.

The dataset was reasonably balanced with successfully sequenced samples approximately equally represented from females (53%) and males (47%). Furthermore, while subgroups A and B were approximately equally represented in sequenced samples (53% RSV-A, 47% RSV-B), patients under the age of five years were more frequently observed with RSV-A and this trend was reversed in cases ≥18 years.

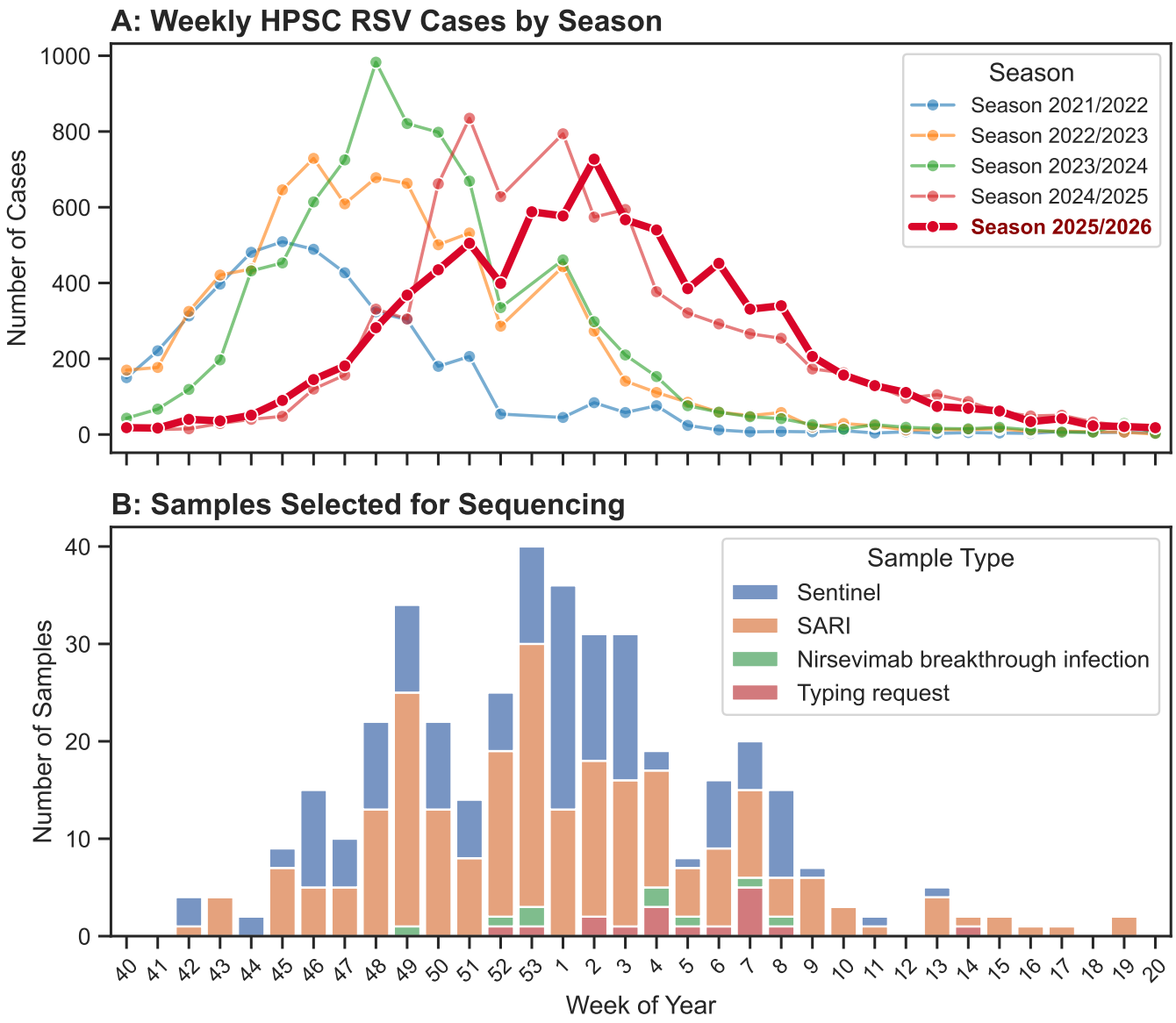

Figure 1: RSV cases and sequenced cases by epidemiological week.
A RSV cases in Ireland by epidemiological week. Data from the (Health Protection Surveillance Centre, 2026) (HPSC) Respiratory Virus Notification Data Hub. **B** Number of sentinel, SARI, nirsevimab breakthrough infections and typing requests selected for sequencing by epidemiological week. Note not all samples were successfully sequenced.

There were 8,020 confirmed RSV cases in Ireland between week 40, 2025 and week 20, 2026 as reported in the HPSC Respiratory Virus Notification Data Hub (Figure 1, A). The data show RSV peak activity in epidemiological week 2 of 2026, which, notably, was later compared to previous seasons with peaks in week 51 (2024/2025) and week 48 (2023/2024). Collection dates for samples selected for sequencing range from week 42 of 2025 to week 19 of 2026, and largely mirror the shape of confirmed cases, with several peaks around week 49, week 53, and week 7 (Figure 1, B).

RSV-A phylogenetic analysis

Clades

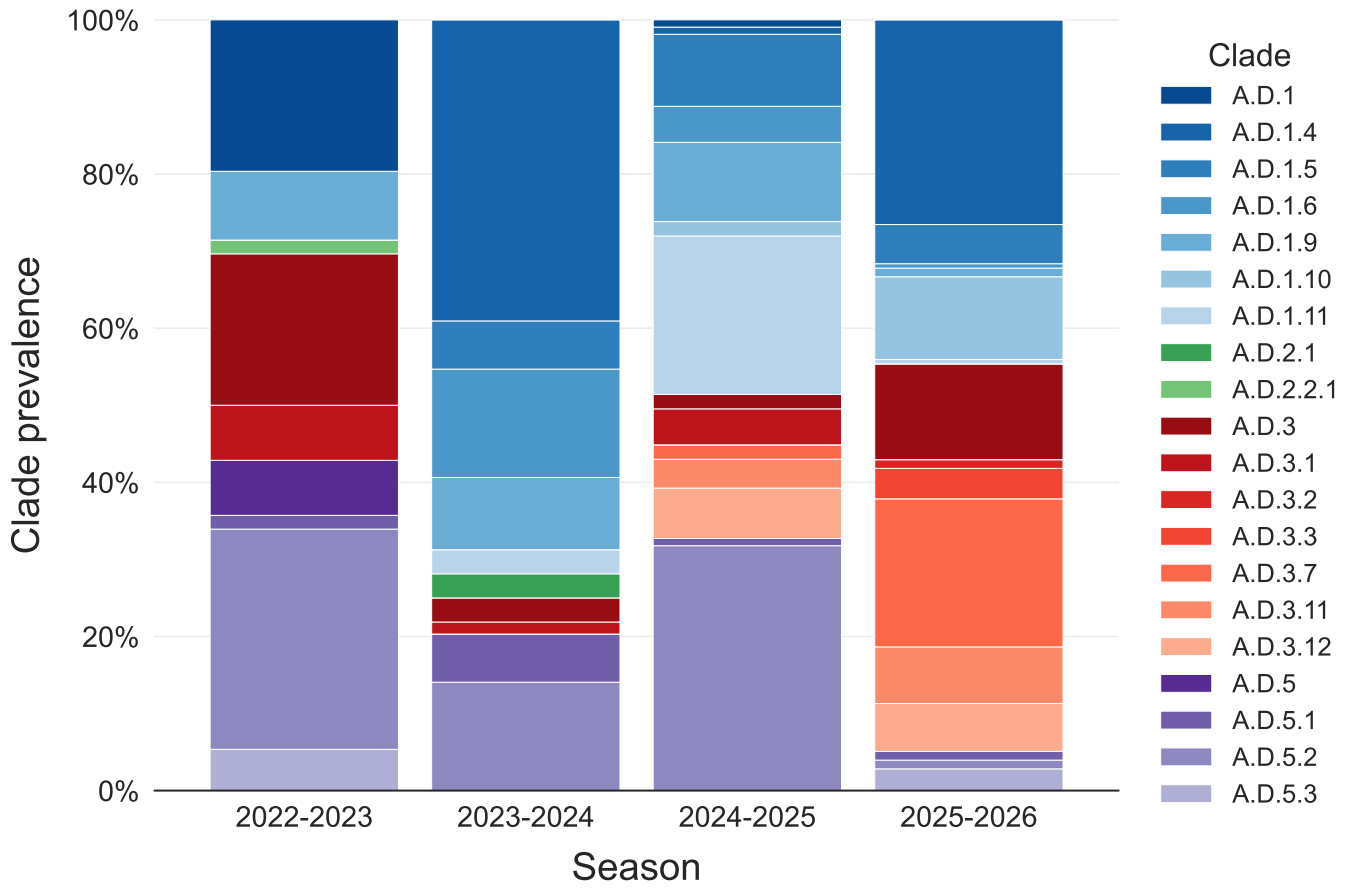

Figure 2: Comparison of RSV-A clades in Ireland by season.
RSV-A clades of Irish samples sequenced since 2022 grouped by the season in which they were collected. Sample sizes – 2022/2023: 56, 2023/2024: 64, 2024/2025: 107, 2025/2026: 177.

Of the 177 RSV-A samples successfully sequenced from the 2025/2026 season (2.2% of total RSV laboratory-confirmed cases nationally), 27% (n=47) were A.D.1.4, 19% (n=34) A.D.3.7, 12% (n=22) A.D.3, and 11% (n=19) A.D.1.10 (Figure 2). Compared to the previous season, there was a large decrease in A.D.5.2 (32% to 1%) and this season had the lowest proportion of A.D.5.X lineages (5%) of the four winter seasons from 2022-2026. Similar to 2023/2024, A.D.1.4 was again the most abundant clade (27%), albeit at a lower proportion. This season we observed the largest proportion of A.D.3.X lineages (50%) in the four seasons, particularly A.D.3.7.

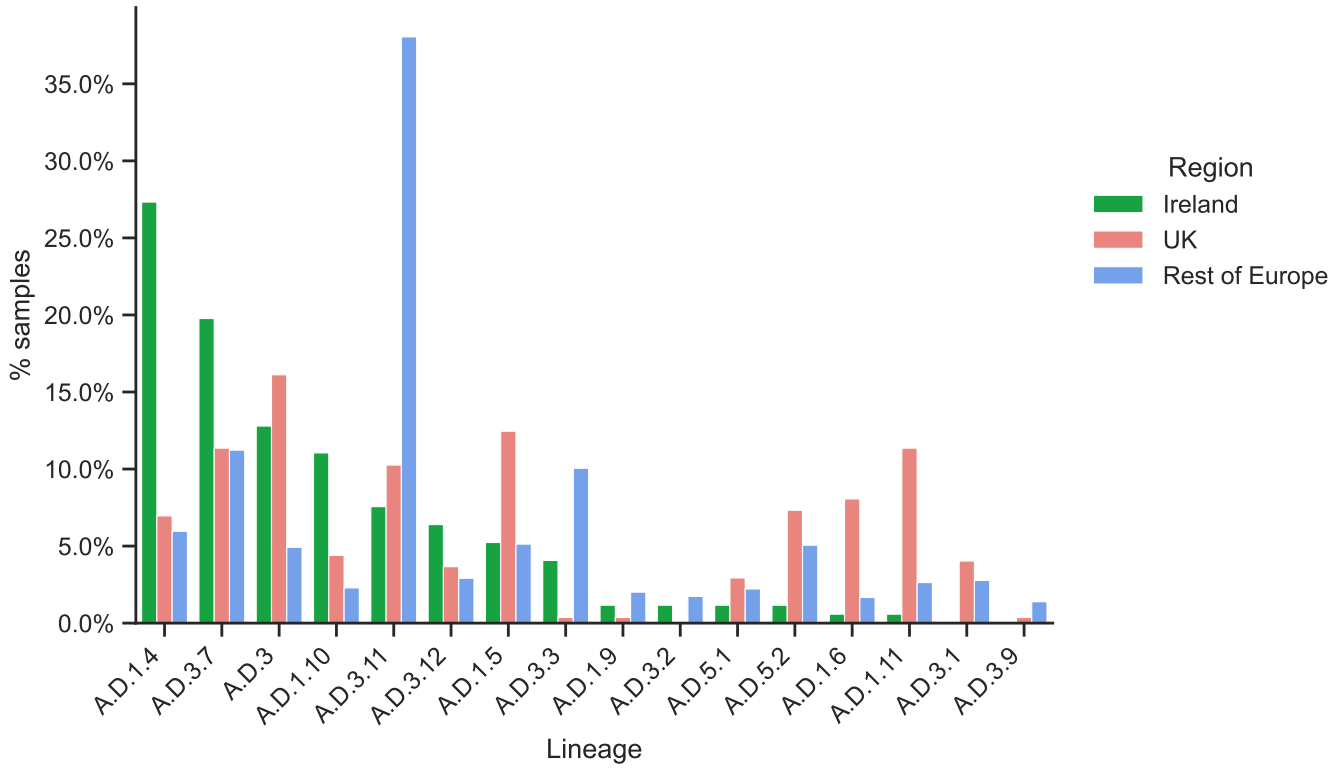

Figure 3: Comparison of RSV-A clades by region.
Clade frequencies of RSV-A sequences on GISAID for epidemiological week 40, 2025 to week 20, 2026 grouped by geographic region and ordered by prevalence in Ireland. Lineages with less than 10 sequences are excluded. Sample sizes – Ireland: 177, UK: 274, Rest of Europe 1,467.

Classifying European RSV-A sequences from this season that are publicly available on GISAID into clades and comparing them to Irish clades, we observe a number of differences (Figure 3). A.D.1.4 is particularly enriched among Irish sequences compared to sequences from the United Kingdom and the rest of Europe. A.D.3.11 is notable in that it comprises more than 35% of sequences in Europe, excluding Ireland and the UK, yet represents less than 10% of sequences in Ireland and the UK.

Phylogenetic tree

A phylogenetic tree of the Irish samples from the 2025/2026 season shows three large circulating clades that branch close to the root of the tree: A.D.1, A.D.3, and A.D.5 (Figure 4).

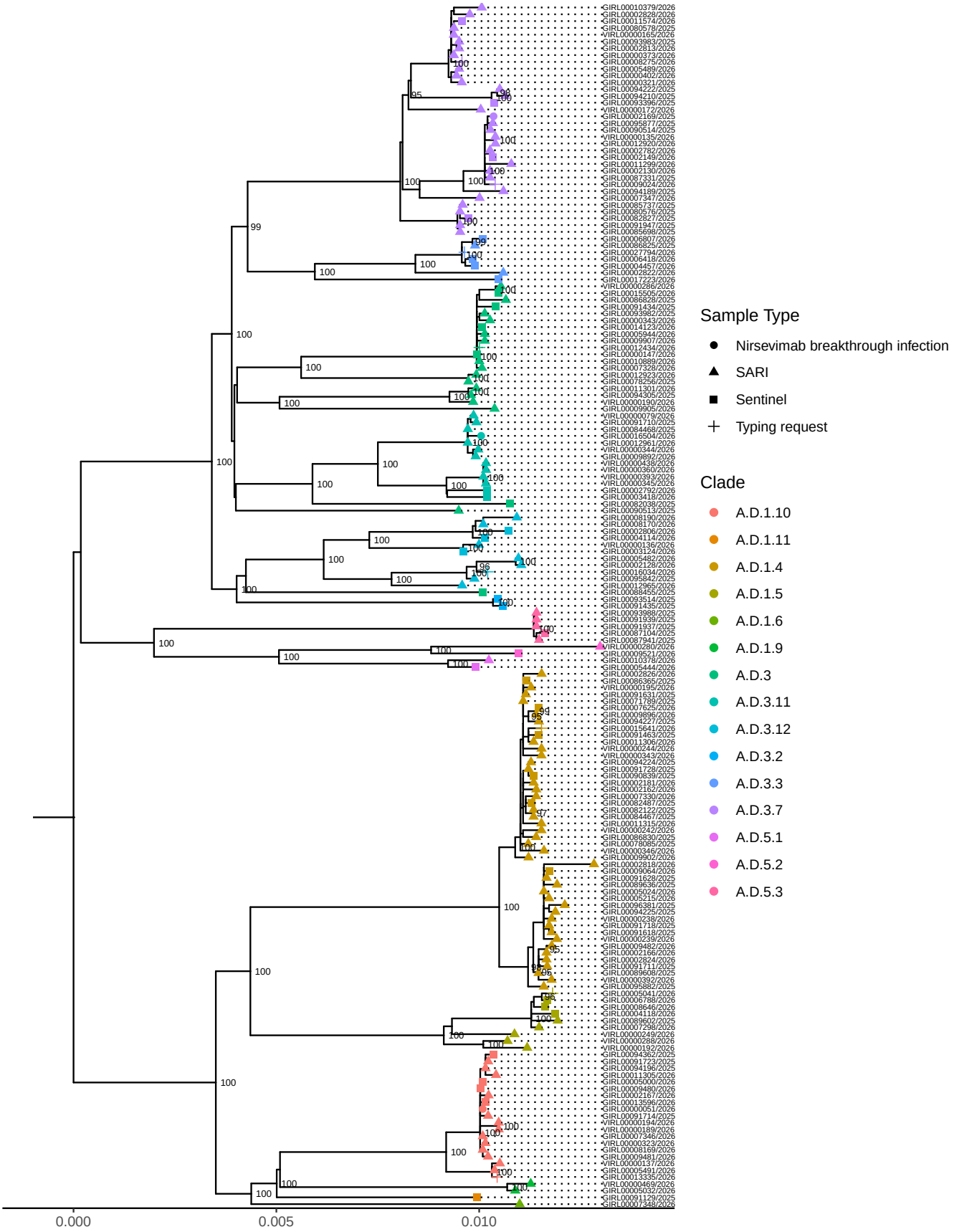

Figure 4: Phylogenetic tree of Irish RSV-A sequences from 2025/2026 season.
Maximum likelihood phylogenetic tree of Irish RSV-A whole genome sequences from 2025/2026 season. Tree tip colours represent assigned clades and tip shapes represent sample source. Branch support values shown where statistical support is high (ultrafast bootstrap approximation ≥ 95% and Shimodaira-Hasegawa-like approximate likelihood ratio test ≥ 80%).

RSV-B phylogenetic analysis

Clades

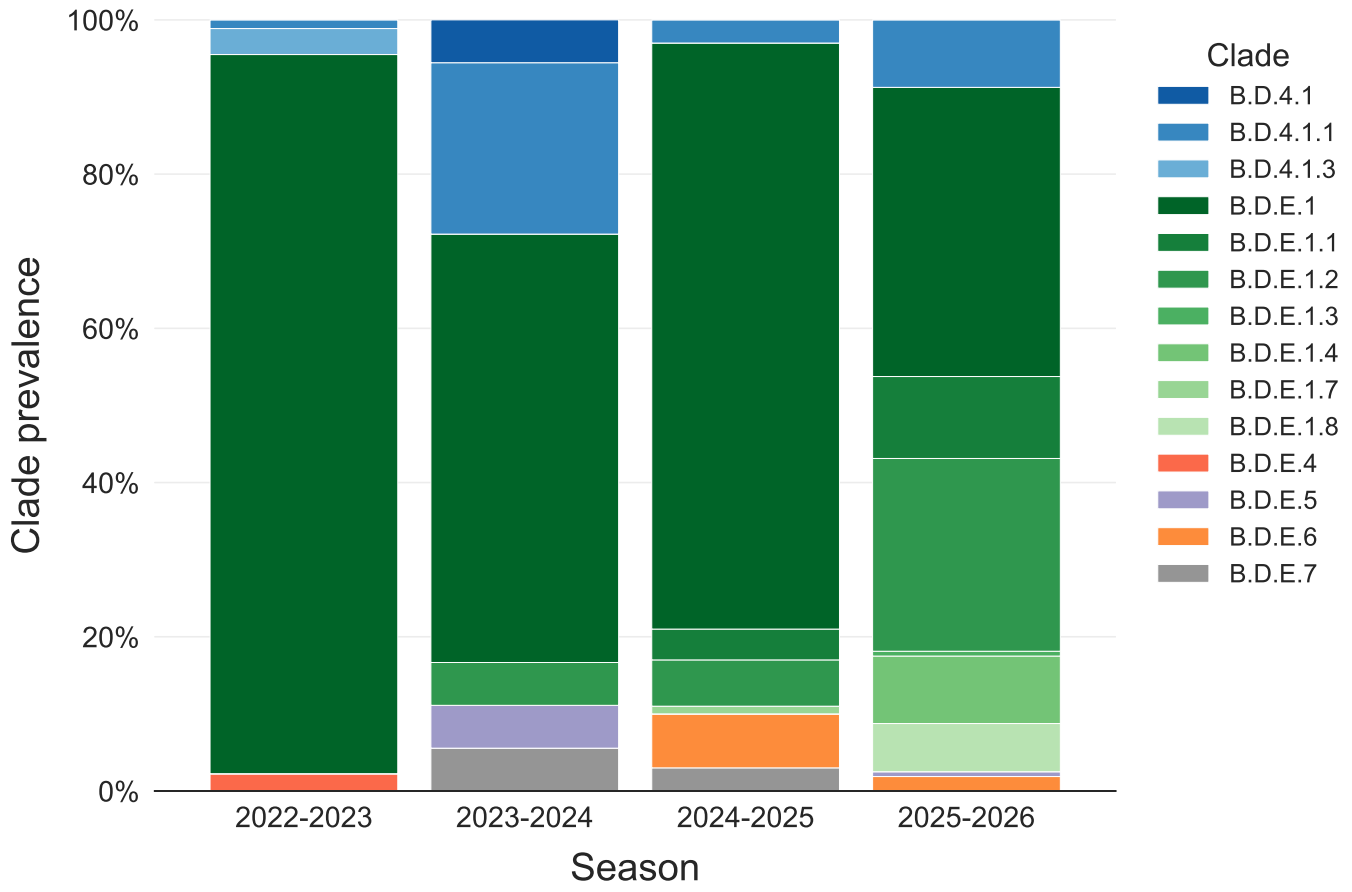

Figure 5: Comparison of RSV-B clades by season.
RSV-B clades of Irish samples sequenced since 2022 grouped by the season in which they were collected. Sample sizes – 2022/2023: 89, 2023/2024: 18, 2024/2025: 100, 2025/2026: 160.

There were 160 RSV-B samples sequenced from this most recent 2025/2026 winter season, 2.0% of the total number of RSV laboratory-confirmed cases nationally. These RSV-B samples mostly comprised three major genetic lineages: B.D.E.1 (n=60, 38%), followed by B.D.E.1.2 (n=40, 25%) and B.D.E.1.1 (n=17, 11%; Figure 5). Comparing the lineages circulating this season to previous seasons, we note an increase in the proportion of samples belonging to the B.D.E.1.2 lineage (6% to 25%).

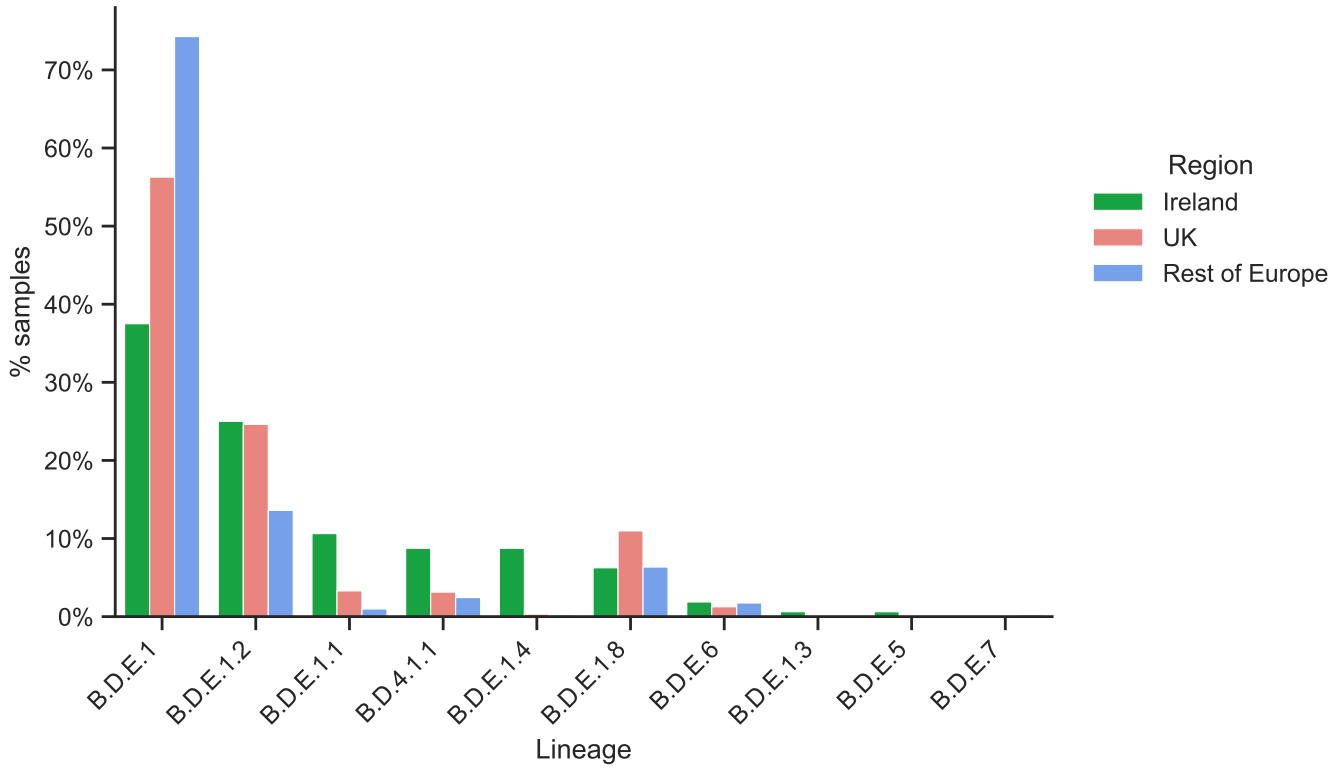

Figure 6: Comparison of RSV-B clades by region.
Clade frequencies of RSV-B sequences on GISAID for epidemiological week 40, 2025 to week 20, 2026 grouped by geographic region and ordered by prevalence in Ireland. Sample sizes – Ireland: 160, UK: 638, Rest of Europe 1,434.

Classifying European RSV-B sequences from this season that are publicly available on GISAID into clades and comparing them to Irish clades, we observe a few discernible differences (Figure 6). While B.D.E.1 is the most prevalent RSV-B lineage in Ireland, the UK, and the rest of Europe, it is much less predominant in Ireland (38%) compared to the other European regions (>50% in UK, >70% in rest of Europe). B.D.E.1.2 has comparable circulating frequencies in Ireland and the UK, however, despite B.D.E.1.4 accounting for 8.8% of Irish samples, it represented <1% in the UK and the rest of Europe.

Phylogenetic tree

A phylogenetic tree of RSV-B samples from this 2025/2026 season shows two major circulating lineages: B.D.4.1.1, and B.D.E.1 along with their respective genetic sub-lineages (Figure 7).

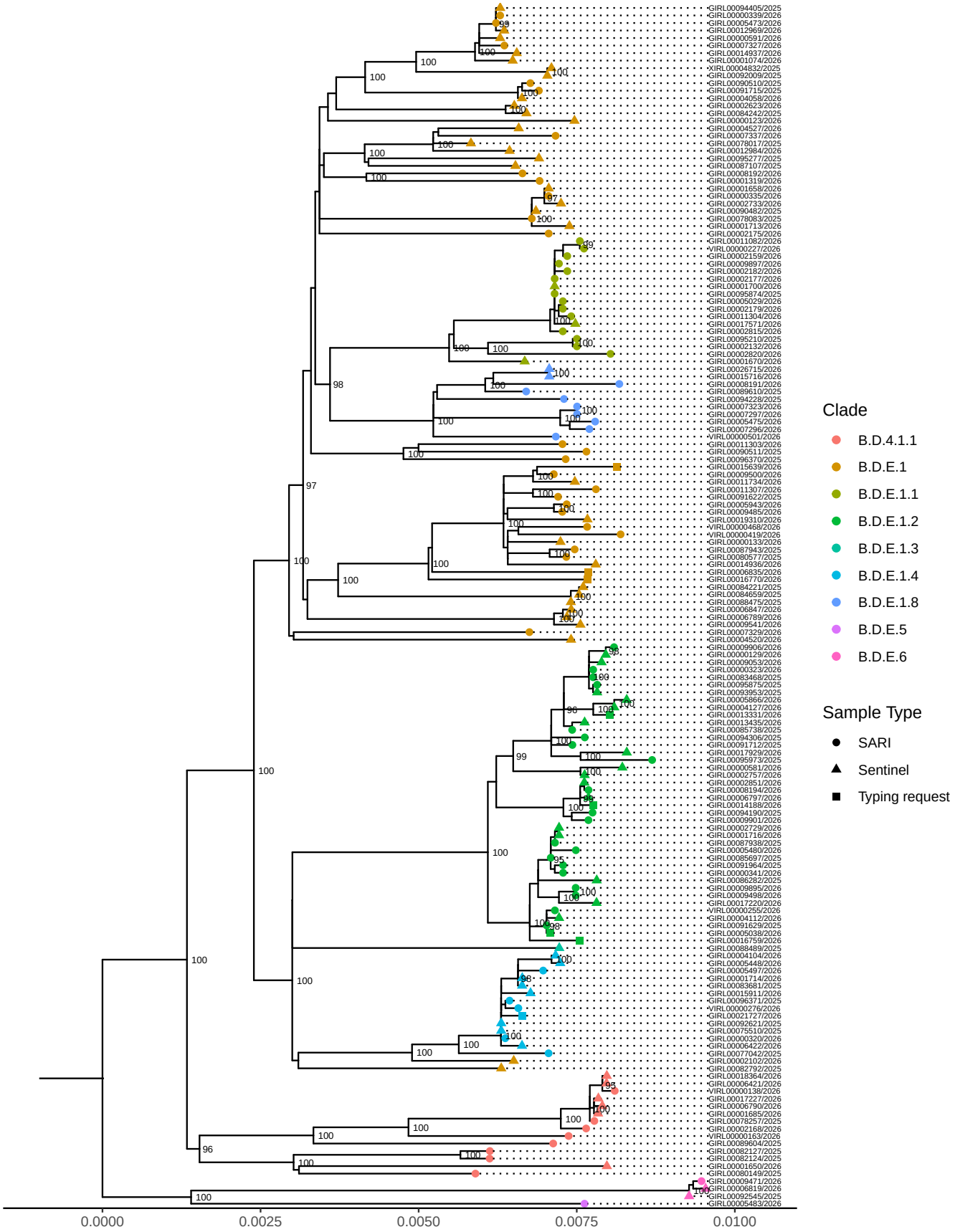

Figure 7: Phylogenetic tree of Irish RSV-B sequences from 2025/2026 season.
Maximum likelihood phylogenetic tree of Irish RSV-B whole genome sequences from 2025/2026 season. Tree tip colours represent assigned clades and tip shapes represent sample source. Branch support values shown where statistical support is high (ultrafast bootstrap approximation ≥ 95% and Shimodaira-Hasegawa-like approximate likelihood ratio test ≥ 80%).

Monoclonal antibody binding site mutations

Passive immunisation approaches with monoclonal antibodies, including palivizumab (Synagis), nirsevimab (Beyfortus), and clesrovimab (Enflonsia), represent a strategy to reduce the incidence of severe RSV disease (Drysdale *et al.*, 2023; The IMpact-RSV Study Group, 1998). They achieve this by binding to the RSV surface fusion (F) glycoprotein, thereby blocking viral fusion with host cells, and inhibiting viral entry (Wilkins *et al.*, 2023). Certain mutations at these binding sites have been observed to induce high levels of resistance in fusion-inhibition assays, for example, I64M/K65E and N208D exhibit >500-fold reduced susceptibility to nirsevimab (Fourati *et al.*, 2025). In Ireland, nirsevimab has been offered for two winter seasons to all infants born between September and February through the HSE RSV Immunisation Pathfinder Programme, in addition to a catch-up immunisation campaign for individuals born between March and August prior to the start of this RSV season (Health Service Executive, 2024). Therefore, it is imperative to identify circulating mutations that could affect the efficacy of the programme.

Nirsevimab

Nirsevimab binds to the F1 and F2 subunits at a discontinuous neutralising epitope in site Ø, located at amino acid residues 62-69 for F2 and 196-212 for F1. For RSV-A samples, two substitutions with no known resistance, N63S and V207I, were detected at low levels (<1.5%) this season (Figure 8). K209E, associated with low-level resistance and observed last season in Ireland, was not detected this season.

For RSV-B, a known resistance mutation, I64T, was observed in a SARI sample from an individual aged between 6-11 months. Notably, I64T has also been detected in one RSV-B sequence from Spain this winter season where nirsevimab is administered, but it was absent from all publicly available UK RSV-B sequences, where this form of prophylaxis is not used (Figure 10). Another known resistance mutation, P205S, was detected in an Irish SARI sample from an individual <6 months of age. P205S was observed in three European RSV-B sequences and no UK RSV-B sequences. R209Q and E66G were also both detected in Irish RSV-B sequences this season but are not associated with known resistance.

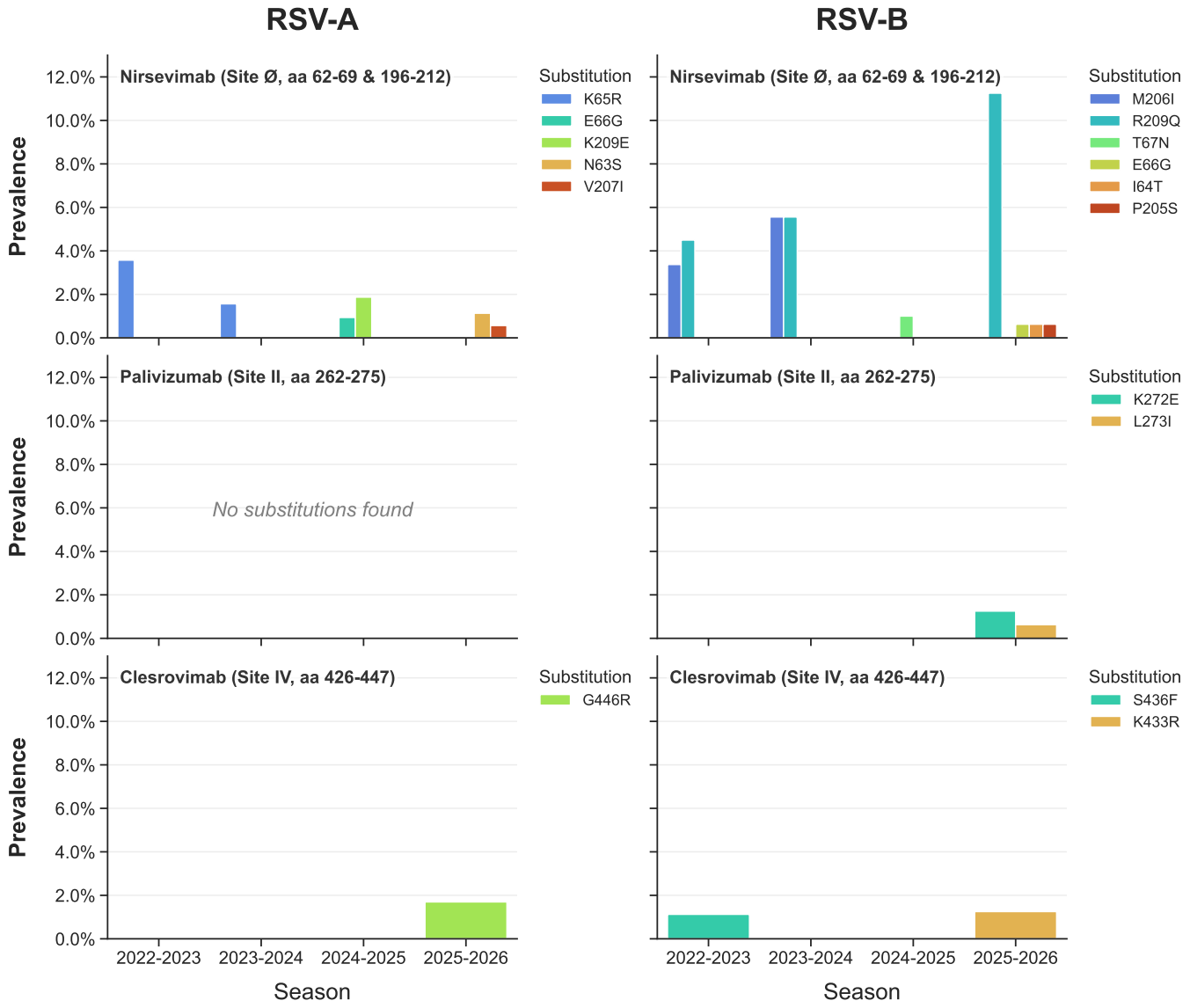

Figure 8: Prevalence of RSV F protein amino acid substitutions in monoclonal antibody target sites in Irish sequences by season (2022-2026).
Bar graphs illustrating the seasonal prevalence (%) of amino acid substitutions within the binding footprints of nirsevimab, palivizumab, and clesrovimab for RSV-A and RSV-B over four consecutive winter respiratory seasons (2022-2023 through 2025-2026).

Palivizumab

The palivizumab-neutralising epitope at antigenic site II is located at amino acid residues 262-275 in the RSV F surface glycoprotein. For RSV-B, two Irish samples had a known palivizumab resistance mutation, K272E (Figure 8). Both samples belong to the B.D.E.1.2 clade and cluster together in the RSV-B phylogenetic tree with three nucleotide differences (Figure 9). The earlier sample was collected as part of the Sentinel Surveillance Programme on December 23rd from an individual between 18-64 years of age. The second sample was collected as part of SARI surveillance on January 25th from an individual between 1-4 years of age. K272E was also not observed in the UK or other European RSV-B sequences (Figure 10).

L273I was also detected in an Irish sequence however this substitution is not associated with resistance to palivizumab. No further amino acid substitutions within the palivizumab binding region were observed in Irish RSV-A sequences.

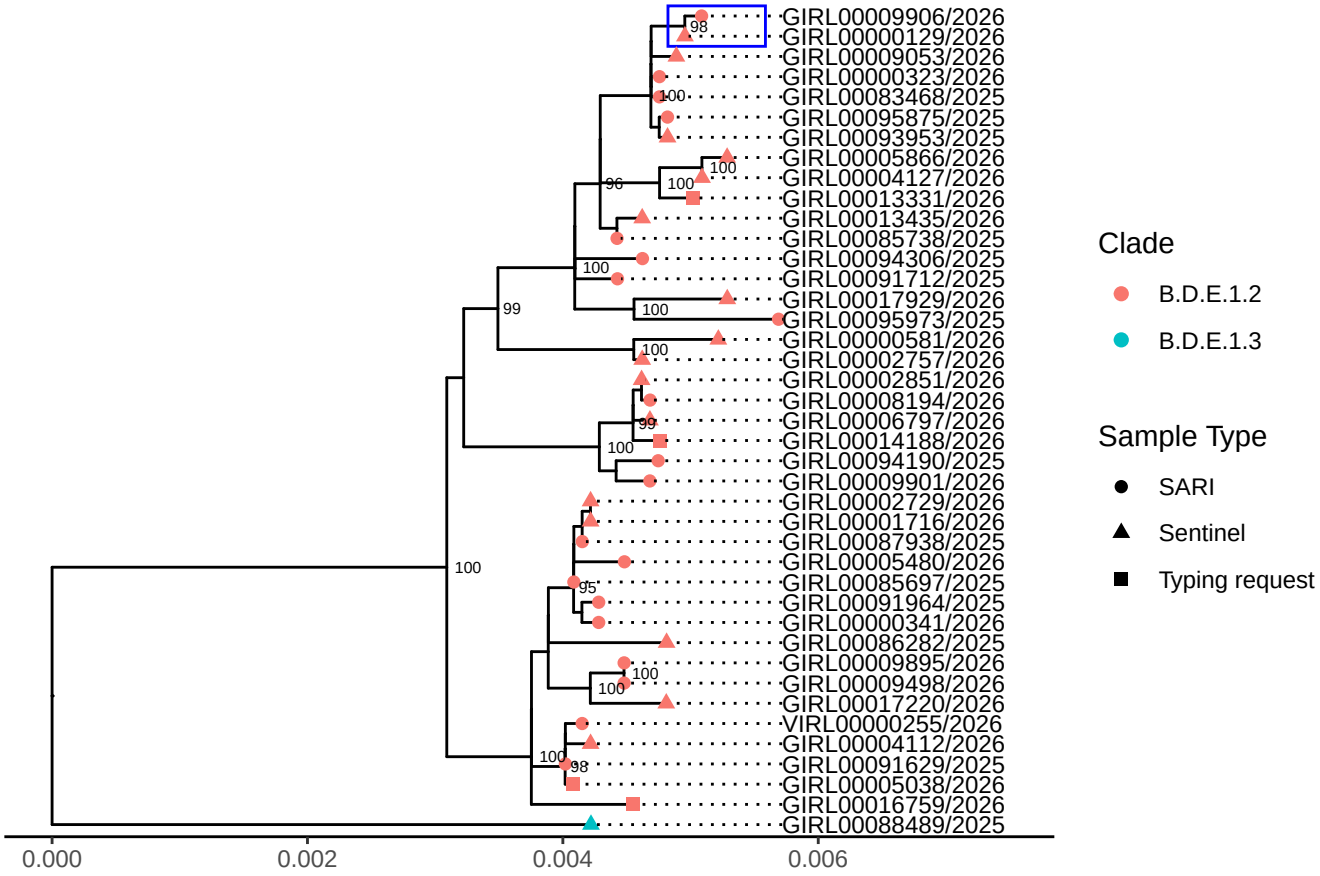

Figure 9: Phylogenetic tree of Irish RSV-B sequences belonging to the B.D.E.1.2 clade
This subtree highlights the genetic relationships among RSV-B whole genome sequences from the 2025/2026 season assigned to the B.D.E.1.2 clade. Tip shapes indicate the sample source. Notably, this clade contains two closely-related samples harboring the known palivizumab-resistance amino acid substitution, K272E, which cluster together with a genetic distance of only three nucleotides. Branch support values are displayed at key nodes where statistical support is high (ultrafast bootstrap approximation ≥ 95% and Shimodaira-Hasegawa-like approximate likelihood ratio test ≥ 80%). The scale bar indicates the number of nucleotide substitutions per site.

Clesrovimab

Clesrovimab targets antigenic site IV at amino acid residues 426-447 in the F surface glycoprotein. For RSV-A sequences, G446R, a known clesrovimab resistance-associated mutation, was detected in three sequences (Figure 8). These sequences do not cluster together and belong to three clades, A.D.3, A.D.3.3, and A.D.3.12. Two samples were collected as part of SARI surveillance and one as part of the Sentinel Programme. This substitution is not observed in other RSV-A sequences in Europe, however it was also detected in a single UK RSV-B sequence (Figure 10).

For RSV-B, we noted the presence of K433R in two Irish sequences though this does not represent a known resistance mutation.

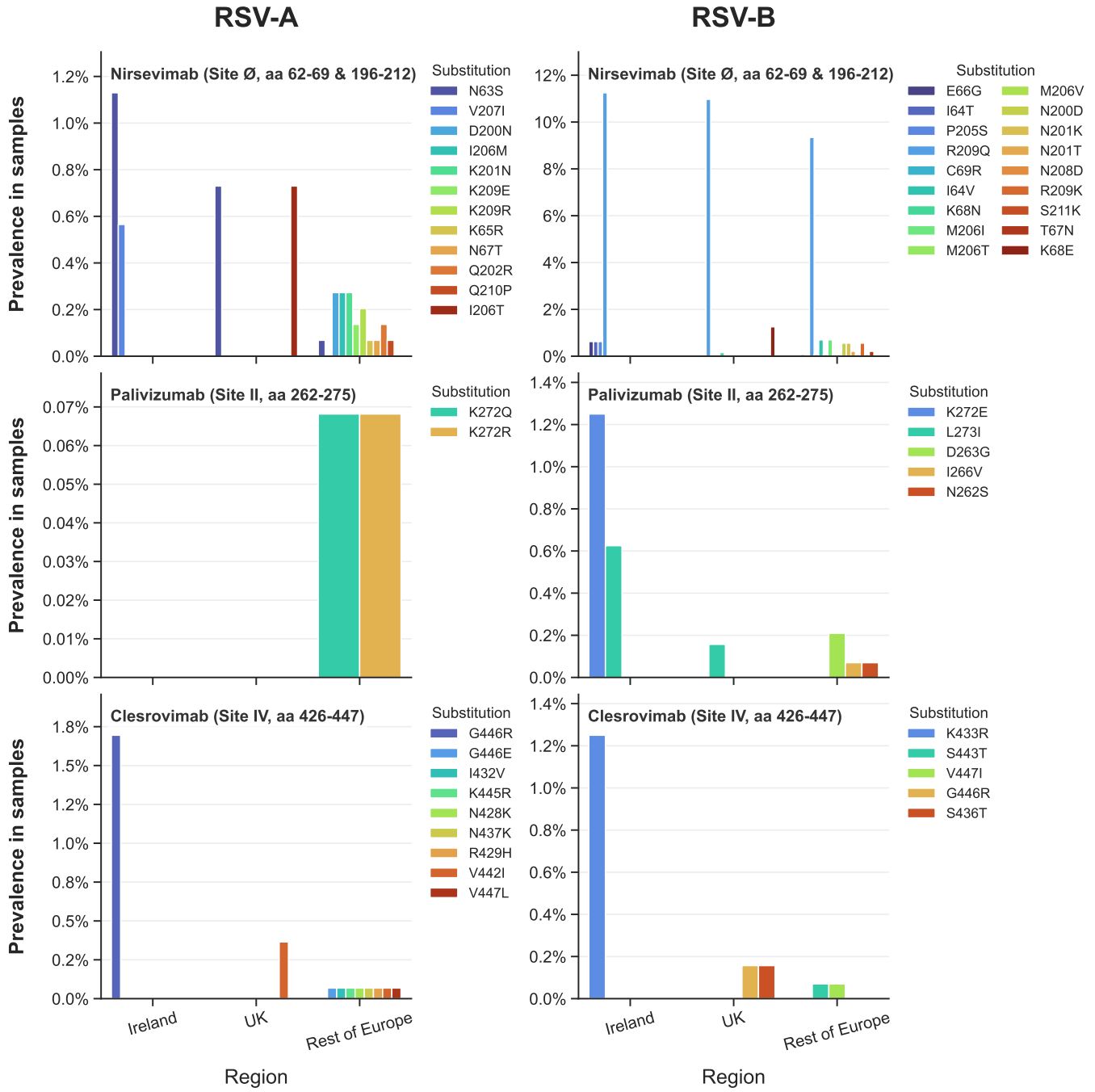

Figure 10: Prevalence of RSV F protein amino acid substitutions in monoclonal antibody target sites by geographic region.

Bar graphs detailing the prevalence (%) of observed amino acid substitutions within the specific F protein epitopes targeted by nirsevimab (Site Ø, aa 62-69 & 196-212), palivizumab (Site II, aa 262-275), and clesrovimab (Site IV, aa 426-447) for both RSV-A and RSV-B. Data is stratified by region: Ireland, the United Kingdom (UK), and the Rest of Europe. Note that Y-axis scales vary between panels to accommodate different prevalence magnitudes.

LIMITATIONS

It is important to acknowledge a number of limitations of this report on genetic characterisation of RSV in Ireland for the 2025/2026 season.

1. Linking genetic characterisation with clinical metadata, such as disease outcome, was not possible.
2. Potential nirsevimab exposure is inferred from the date of birth of the sample, unless explicitly indicated as a breakthrough infection, as data is typically unavailable with respect to exposure to monoclonal antibodies and nirsevimab uptake is reported as high (>82%).
3. While efforts have been made to normalise for collection date, age-range, geographic representation and level of care, the dataset is a proxy for RSV activity in Ireland throughout the winter season. A defined sampling strategy is not in place for RSV WGS in Ireland and the dataset may not be completely representative of the viral epidemiology throughout the season.

CONCLUSIONS

It is clear that RSV genomics has become an important tool to fully assess the dynamics of this pathogen's impact due to: 1) the recent introduction of vaccination and monoclonal antibodies, 2) more than 8,000 laboratory-confirmed RSV cases in Ireland between week 40 and week 20, and 3) a large proportion of these cases occurring in young children. The WHO recommends that each country participating in RSV genomic surveillance sequences a minimum of 46 RSV-positive specimens per year (World Health Organization, 2025). Furthermore, it acknowledges that countries in which RSV interventions are being used will need to increase this number to better evaluate extant genetic diversity and genomic changes relevant to interventions. This season, we met this recommendation by sequencing 337 samples that are broadly representative of the viral epidemiology of the RSV antigenic subgroups circulating in Ireland.

RSV pathogen genomics revealed that the frequencies of circulating lineages during the winter season 2025/2026 were both distinct from previous seasons in Ireland and, notably,

were also distinguishable from the circulating diversity in the UK and other European countries. The A.D.1.4 lineage, the single largest RSV-A lineage in the 2023/2024 season that was represented by only a single detection last season (2024/2025), returned as the largest lineage in 2025/2026, indicative of complex patterns of genetic lineage transmission. This high prevalence was also approximately five times the frequency observed elsewhere in Europe. Conversely, A.D.3.11 comprised more than 35% of European cases but less than 10% in Ireland.

For RSV-B, the predominance of the B.D.E.1 genetic lineage was much reduced this season in Ireland compared to last season and elsewhere in Europe. Other B.D.E.1.X lineages and B.D.4.1.1 were also more common compared to last season. Taking both RSV subgroups into account, RSV lineage dynamics in Ireland this winter season are noteworthy when compared either temporally or geographically, underscoring the need for ongoing pathogen surveillance here to provide an evidence base to better understand factors affecting transmission dynamics.

The introduction of the monoclonal antibody nirsevimab through the high uptake HSE Immunisation Pathfinder Programme during the 2024/2025 winter season creates a need to assess the impact of passive immunisation on disease burden and viral evolution over time. Pathogen genomics can contribute to one aspect of this assessment: identifying nirsevimab binding-site mutations that can reduce efficacy. A number of mutations were observed this season, two of which, I64T and P205S in RSV-B, are known to be associated with high levels of resistance. These mutations are currently observed at a low frequency and are therefore unlikely to affect the success of the immunisation programme. There is no current evidence to suggest circulating RSV diversity this winter season negatively impacted the passive immunisation programme. Future surveillance of these mutations and others arising will be necessary with the continued availability of population-scale immunisation strategies to mitigate morbidity and mortality in vulnerable groups.

ACKNOWLEDGEMENTS

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REFERENCES

Aksamentov, I., Roemer, C., Hodcroft, E., & Neher, R. (2021). Nextclade: clade assignment, mutation calling and quality control for viral genomes. *Journal of Open Source Software*, *6*(67), 3773. https://doi.org/10.21105/joss.03773

Drysdale, S. B., Cathie, K., Flamein, F., Knuf, M., Collins, A. M., Hill, H. C., Kaiser, F., Cohen, R., Pinquier, D., Felter, C. T., Vassilouthis, N. C., Jin, J., Bangert, M., Mari, K., Nteene, R., Wague, S., Roberts, M., Tissières, P., Royal, S., … HARMONIE Study Group. (2023). Nirsevimab for prevention of hospitalizations due to RSV in infants. *N. Engl. J. Med.*, *389*(26), 2425–2435. https://doi.org/10.1056/NEJMoa2309189

Fourati, S., Reslan, A., Bourret, J., Casalegno, J.-S., Rahou, Y., Chollet, L., Pillet, S., Tremeaux, P., Dossou, N. C., Gault, E., Salmona, M., Imbert-Marcille, B.-M., Mirand, A., Larrat, S., Moisan, A., Marot, S., Schnuriger, A., Veyrenche, N., Engelmann, I., … Ogoudjobi, S. (2025). Genotypic and phenotypic characterisation of respiratory syncytial virus after nirsevimab breakthrough infections: a large, multicentre, observational, real-world study. *The Lancet Infectious Diseases*, *25*(3), 301–311. https://doi.org/10.1016/S1473-3099(24)00570-X

Goya, S., Ruis, C., Neher, R. A., Meijer, A., Aziz, A., Hinrichs, A. S., Gottberg, A. von, Roemer, C., Amoako, D. G., Acuña, D., McBroome, J., Otieno, J. R., Bhiman, J. N., Everatt, J., Muñoz-Escalante, J. C., Ramaekers, K., Duggan, K., Presser, L. D., Urbanska, L., … Viegas, M. (2024). Standardized Phylogenetic Classification of Human Respiratory Syncytial Virus below the Subgroup Level. *Emerging Infectious Diseases*, (30). https://doi.org/10.3201/eid3008.240209

Health Protection Surveillance Centre. (2026,). *Respiratory Virus Notification Data Hub*. https://respiratorydisease-hpscireland.hub.arcgis.com/

Health Service Executive. (2024,). *New HSE immunisation programme to protect newborn babies against RSV*. https://about.hse.ie/news/new-hse-immunisation-programme-to-protect-newborn-babies-against-rsv/

The IMpact-RSV Study Group. (1998). Palivizumab, a humanized respiratory syncytial virus monoclonal antibody, reduces hospitalization from respiratory syncytial virus infection in high-risk infants. *Pediatrics*, *102*(3 Pt 1), 531–537.

Katoh, K., & Standley, D. M. (2013). MAFFT Multiple Sequence Alignment Software Version 7: Improvements in Performance and Usability. *Molecular Biology and Evolution*, *30*(4), 772–780. https://doi.org/10.1093/molbev/mst010

Lemoine, F., & Gascuel, O. (2021). Gotree/Goalign: toolkit and Go API to facilitate the development of phylogenetic workflows. *NAR Genomics and Bioinformatics*, *3*(3), lqab075. https://doi.org/10.1093/nargab/lqab075

Li, Y., Wang, X., Blau, D. M., Caballero, M. T., Feikin, D. R., Gill, C. J., Madhi, S. A., Omer, S. B., Simões, E. A. F., Campbell, H., Pariente, A. B., Bardach, D., Bassat, Q., Casalegno, J.-S., Chakhunashvili, G., Crawford, N., Danilenko, D., Do, L. A. H., Echavarria, M., … Nair, H. (2022). Global, regional, and national disease burden estimates of acute lower respiratory infections due to respiratory syncytial virus in children younger than

5 years in 2019: a systematic analysis. *The Lancet*, *399*(10340), 2047–2064. https://doi.org/10.1016/S0140-6736(22)00478-0

Minh, B. Q., Schmidt, H. A., Chernomor, O., Schrempf, D., Woodhams, M. D., Von Haeseler, A., & Lanfear, R. (2020). IQ-TREE 2: New Models and Efficient Methods for Phylogenetic Inference in the Genomic Era. *Molecular Biology and Evolution*, *37*(5), 1530–1534. https://doi.org/10.1093/molbev/msaa015

Rice, A., Gonzalez, G., Carr, M., Dean, J., O'Byrne, E., Aarts, L., Vennema, H., Banka, W., Bennett, C., Cleary, S., Domegan, L., O'Donnell, J., O'Leary, M., Goya, S., Presser, L., Meijer, A., Martin, G., Sawa, H., Waters, A., … Hare, D. (2025). Human respiratory syncytial virus genetic diversity and lineage replacement in Ireland pre- and post-COVID-19 pandemic. *Microbial Genomics*, *11*(3), 1379. https://doi.org/10.1099/mgen.0.001379

Santos, J. D., Sobral, D., Pinheiro, M., Isidro, J., Bogaardt, C., Pinto, M., Eusébio, R., Santos, A., Mamede, R., Horton, D. L., Gomes, J. P., Consortium, T., Bigarré, L., Fernández-Pinero, J., Pais, R. J., Marcacci, M., Moreno, A., Lilja, T., Øines, Ø., … Borges, V. (2024). INSaFLU-TELEVIR: an open web-based bioinformatics suite for viral metagenomic detection and routine genomic surveillance. *Genome Medicine*, *16*(1), 61. https://doi.org/10.1186/s13073-024-01334-3

Surie, D., Yuengling, K. A., DeCuir, J., Zhu, Y., Gaglani, M., Ginde, A. A., Talbot, H. K., Casey, J. D., Mohr, N. M., Ghamande, S., Gibbs, K. W., Files, D. C., Hager, D. N., Ali, H., Prekker, M. E., Gong, M. N., Mohamed, A., Johnson, N. J., Steingrub, J. S., … IVY Network. (2023). Disease severity of respiratory syncytial virus compared with COVID-19 and influenza among hospitalized adults aged g =60 years - IVY Network, 20 U.S. States, February 2022-May 2023. *MMWR Morb. Mortal. Wkly. Rep.*, *72*(40), 1083–1088. https://doi.org/10.15585/mmwr.mm7240a2

Terstappen, J., Hak, S. F., Bhan, A., Bogaert, D., Bont, L. J., Buchholz, U. J., Clark, A. D., Cohen, C., Dagan, R., Feikin, D. R., Graham, B. S., Gupta, A., Haldar, P., Jalang'o, R., Karron, R. A., Kragten, L., Li, Y., Löwensteyn, Y. N., Munywoki, P. K., … Mazur, N. I. (2024). The respiratory syncytial virus vaccine and monoclonal antibody landscape: the road to global access. *The Lancet Infectious Diseases*, *24*(12), e747–e761. https://doi.org/10.1016/S1473-3099(24)00455-9

Wilkins, D., Langedijk, A. C., Lebbink, R. J., Morehouse, C., Abram, M. E., Ahani, B., Aksyuk, A. A., Baraldi, E., Brady, T., Chen, A. T., Chi, H., Choi, E. H., Cohen, R., Danilenko, D. M., Gopalakrishnan, V., Greenough, A., Heikkinen, T., Hosoya, M., Keller, C., … Yun, K. W. (2023). Nirsevimab binding-site conservation in respiratory syncytial virus fusion glycoprotein worldwide between 1956 and 2021: an analysis of observational study sequencing data. *The Lancet Infectious Diseases*, *23*(7), 856–866. https://doi.org/10.1016/S1473-3099(23)00062-2

World Health Organization. (2025). *Respiratory syncytial virus sequencing considerations for an expanded Global Influenza Surveillance and Response System* (p. vi, 25 p.) [Publications]. World Health Organization. https://www.who.int/publications/b/75237