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2025 Annual Australian Measles and Rubella Genotyping Surveillance Program Report

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Abstract

Molecular characterisation is a critical component of Australia's measles and rubella surveillance systems, supporting outbreak investigations, identifying sources of infection, and providing evidence for the continued maintenance of Australia's measles and rubella elimination status. This annual report summarises measles and rubella molecular epidemiology in Australia during 2025 and evaluates the utility of whole genome sequencing (WGS) for measles surveillance. In 2025, there were 181 confirmed measles cases notified in Australia, representing a 217.5% increase on the number notified in 2024. Measles virus genotypes B3 and D8 were detected, together with 21 vaccine-associated genotype A cases. Genotype B3 predominated during the first half of the year and was largely associated with importations from Vietnam, whereas genotype D8 predominated during the second half of the year and was primarily associated with importations from Indonesia. Thirty-one epidemiologically confirmed outbreaks were identified, comprising 13 genotype B3 outbreaks and 17 genotype D8 outbreaks. The discriminatory power and concordance with epidemiological data in the context of outbreak investigations was substantially greater for WGS data than for conventional N450-based genotyping. Viruses sharing the same N450-based distinct sequence identifier (DSid) differed by up to 33 single nucleotide polymorphisms (SNPs) for genotype B3 and 36 SNPs for genotype D8, whereas genomes obtained from epidemiologically linked cases differed by a maximum of four SNPs. Despite increased national case numbers in 2025, available molecular and epidemiological evidence indicated that cases were associated with importations or import-related transmission events, with no evidence of sustained endemic measles transmission. Rubella activity remained extremely low, with no rubella virus detections identified among 157 specimens tested by polymerase chain reaction (PCR) and only two notified cases nationally, neither of which had material available for genotyping. These findings support the continued absence of endemic measles and rubella transmission in Australia and highlight the value of WGS for enhancing measles surveillance and outbreak investigations.

Keywords: genomic surveillance; molecular epidemiology; outbreak investigation; public health surveillance

Background

Genotyping evidence for measles and rubella remains essential for verifying the interruption of endemic transmission in Australia. This evidence underpinned the verification of Australia's achievement of measles[1] and rubella elimination status in 2014 and 2017, respectively, and continues to support the country's ongoing maintenance of elimination. Globally, molecular surveillance of measles and rubella is supported through the World Health Organization Global Measles and Rubella Laboratory Network (GMRLN), which includes the MeaNS2 (Measles Nucleotide Surveillance)[2] and RubeNS2 (Rubella Nucleotide Surveillance)[3] databases. These international databases facilitate standardised reporting, molecular surveillance of measles and rubella viruses, and verification of the absence of endemic transmission within a given country.

This annual report provides a comprehensive overview of measles and rubella genotyping surveillance activities in Australia for 2025. Molecular genotyping is a critical component of the national measles surveillance system, supporting outbreak investigations, identifying sources of infection, and documenting the absence of endemic virus circulation. However, the reduction in circulating measles genotypes has limited the ability to differentiate between measles cases using the traditional N450-based Sanger sequencing genotyping method. Enhanced resolution of laboratory data is therefore required to assist with public health management of measles. In this report, in addition to providing a comprehensive overview of measles genotyping surveillance in Australia for 2025, a comparison is also made between the resolution provided by legacy Sanger sequencing (N450/genotyping) and by contemporary whole genome sequencing (WGS) to differentiate between confirmed cases of measles.

Methods

Real-time RT-PCR

All presumptive measles specimens received by the Victorian Infectious Diseases Research Laboratory (VIDRL) underwent confirmatory polymerase chain reaction (PCR) testing as follows. Total nucleic acid was extracted from clinical specimens using validated automated extraction methods according to the manufacturer's instructions. Measles virus RNA was detected using a modified real-time reverse transcription polymerase chain reaction (rRT-PCR) assay based on the protocol developed by the United States Centers for Disease Control and Prevention.[4] The assay targets a conserved region of the measles virus nucleoprotein (N) gene and is designed to detect all known measles virus genotypes.

Measles genotyping PCR and N450 Sanger sequencing

All measles rRT-PCR-positive specimens then underwent genotyping, which is performed using Sanger sequencing of the N450 region of the measles genome as follows. Measles genotyping PCR was performed using a modified version[5] of the World Health Organization- (WHO) recommended protocol.[6] The modification incorporated a second-round PCR using primers MeV216 and MeV214[7] to improve amplification sensitivity. The resulting amplicon, encompassing the 450-nucleotide region at the carboxyl terminus of the nucleoprotein gene (N450), was sequenced in both the forward and reverse directions as previously described.[5] The N450 sequences obtained were compared against reference sequences in the WHO GMRLN MeaNS2 database,[2] and distinct sequence identifiers (DSids) were assigned where applicable. N450 Sanger sequencing was performed at VIDRL on samples submitted by all jurisdictions except for Western Australia and Queensland. Both Western Australia and Queensland submit Sanger-derived N450 sequences from confirmed measles cases to VIDRL for the purposes of national surveillance and reporting.

Whole genome sequencing

Whole genome sequencing (WGS) was performed on samples from confirmed measles cases from all Australian states and territories, except Queensland and Western Australia, which performed Sanger sequencing and whole genome sequencing locally. Whole genome sequencing data generated by these jurisdictions was not submitted to VIDRL and thus could not be included in this report. Illumina whole genome sequencing data were analysed using the phac-nml/measeq[8] nextflow pipeline using default settings with the addition of the *--removeduplicates* flag. A minimum genome completeness of 85% was used as a cut-off for downstream analysis. For multiple sequence alignment and phylogenetic analysis, the augur toolkit (v33.0.0) was used.[9] Sequences were aligned using MAFFT[10] (v7.526, settings: *--reorder*; *--anysymbol*; *--nomemsave*; *--adjustdirection*) followed by phylogenetic analysis using IQTREE[11] (v3.0.1, settings: *-m GTR+G*; *-B 1000*; *-bnni*; *-alrt 1000*). Phylogenies were visualised using ggtree (4.0.5).[12] World Health Organization reference sequences (eg. NY.USA_94) and representative named strains obtained from the MeaNS2 database (eg. MVs/Victoria.AUS/09.25) were included in all phylogenies to provide context for Australian sequences. In addition, the reference sequence used for alignment/assembly is also included in the phylogenies for each genotype, and is labelled as such. The two reference sequences used for alignment/assembly (B3: PV686488 and D8: MH356236) were selected based on high sequence similarity with representative Australian sequences belonging to each genotype. Branch lengths are expressed as nucleotide substitutions per site.

Results and discussion

Measles molecular epidemiology, Australia, 2025

There were 2,121 samples tested for measles RNA by rRT-PCR at VIDRL during the reporting period, representing a 195.4% increase on the number of samples tested in 2024. The number of notified and confirmed measles cases rose from 57 in 2024 to 181 in 2025, an increase of 217.5%.

The rise in cases is attributed to a global decline in first-dose measles-containing vaccine coverage (84% in 2024), slightly below the 2019 level of 86%, resulting in a significant global resurgence of measles. An estimated 11 million cases were reported worldwide in 2024, an 8% increase compared with pre-pandemic levels in 2019.[13,14]

Within Australia during the reporting period, measles genotypes B3 and D8 were identified, as well as 21 vaccine-associated measles cases (genotype A). A distinct temporal pattern for measles genotypes B3 and D8 in Australia during 2025 was observed.

For inclusion in this report, Sanger N450 sequences were generated by VIDRL, or provided to VIDRL by all jurisdictions, for 163/181 confirmed measles cases (90.1%) detected in Australia in 2025. During the reporting period, WGS was attempted and successful for 70/75 measles samples (93%), referred from all jurisdictions except Western Australia and Queensland. WGS was attempted and unsuccessful for five of the 75 samples (7%) submitted to VIDRL during the reporting period.

Temporal and geographic distribution of measles cases in Australia

Measles cases were reported across multiple jurisdictions, with Victoria, New South Wales, Queensland and Western Australia contributing the majority of detections: 19.9%, 20.4%, 18.8% and 34.8% respectively. Other jurisdictions (South Australia, Tasmania, Northern Territory) contributed relatively few cases overall. The broad geographic distribution of confirmed measles cases (Figure 1) highlighted widespread but intermittent transmission linked to multiple introductions. Larger peaks were typically driven by a small number of jurisdictions at any given time, consistent with localised outbreaks rather than sustained nationwide transmission. The temporal separation between B3 (early 2025) and D8 (mid-to-late 2025) further supports the observation of distinct importation and transmission events for each genotype.

For genotype B3, cases were concentrated in the early part of the year, with activity beginning in the first quarter and peaking between approximately epidemiological weeks 10–15 (Figure 1A). During this period, multiple jurisdictions reported confirmed cases, with notable activity in Victoria, New South Wales and Western Australia. Following this peak, B3 detections declined rapidly, with only sporadic cases identified beyond mid-year and very limited activity in the latter half of 2025.

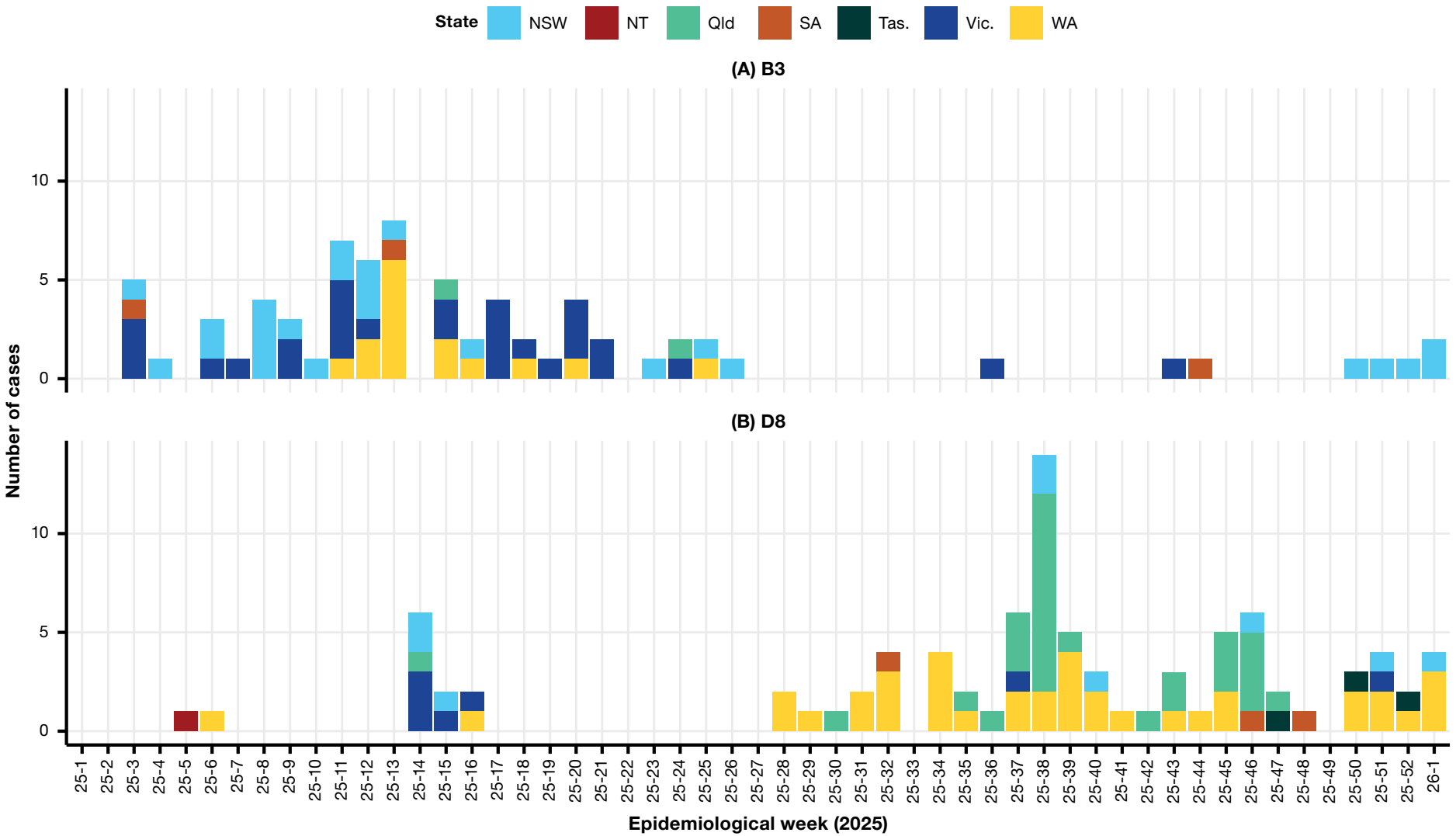
In contrast, genotype D8 showed minimal activity in the early months but increased substantially from mid-year onwards (Figure 1B). A clear rise in cases is observed from approximately week 30, with a pronounced peak around weeks 36–40. This surge was driven largely by cases in Queensland and Western Australia, with additional contributions from other jurisdictions. D8 activity remained elevated into the later part of the year, with continued, albeit lower-level, detections extending towards the end of 2025 and into early 2026.

Overall, the distribution of measles cases illustrates a shift in dominant genotype from B3 to D8 over the course of 2025, with outbreaks occurring sporadically across jurisdictions and with no evidence of continuous endemic transmission.

Measles genotype B3 in Australia

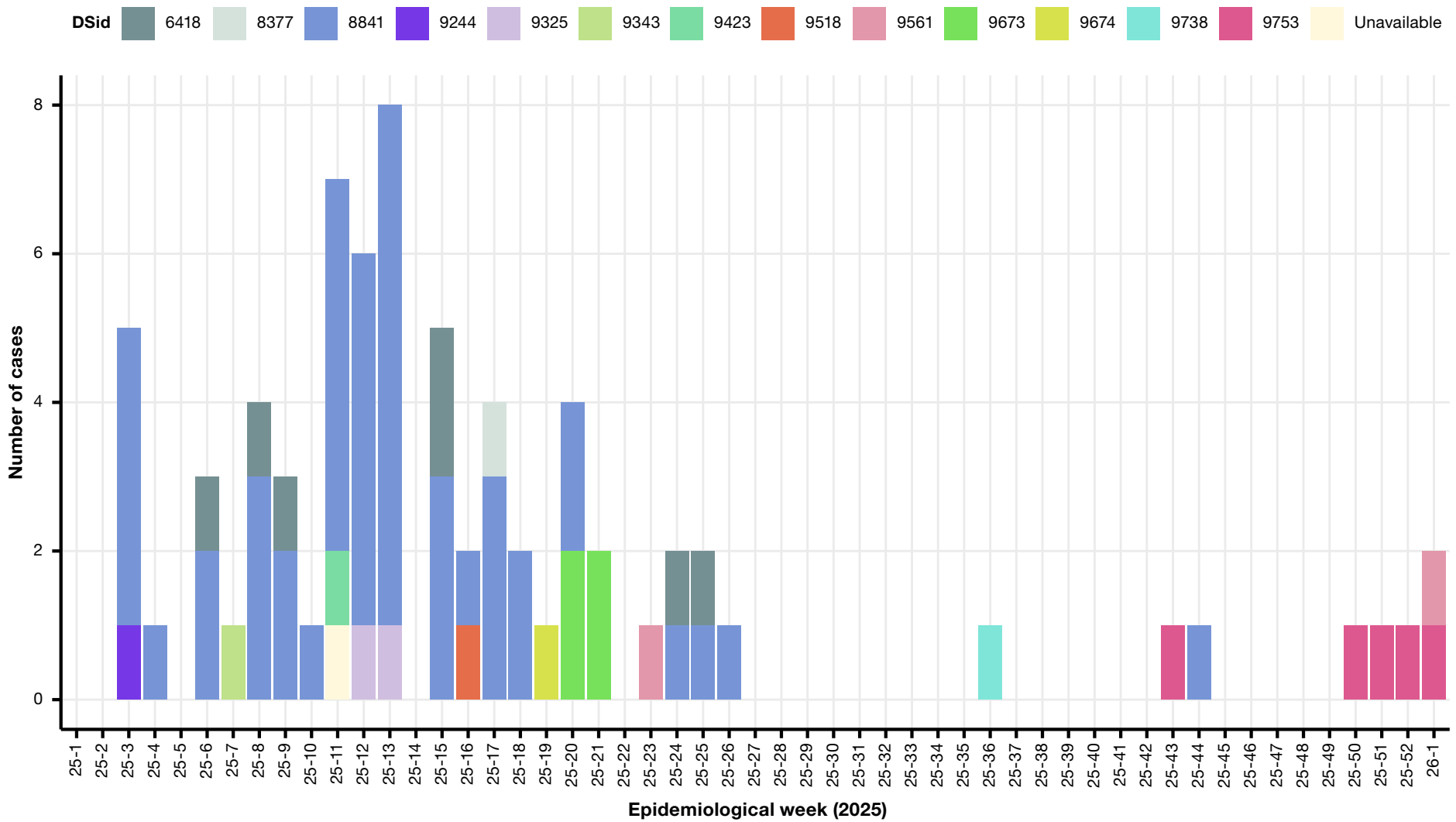
Measles genotype B3 was identified in 73 of 163 Sanger N450 sequenced confirmed cases (45%) and was predominantly detected in the first half of 2025 (Figures 1–3). Genotyping was unsuccessful for a further five cases; however, these were epidemiologically linked to outbreaks caused by genotype B3 (data not shown).

Figure 1: Geographical distribution of measles genotypes (A) B3 and (B) D8 by jurisdiction,[a] Australia, 2025



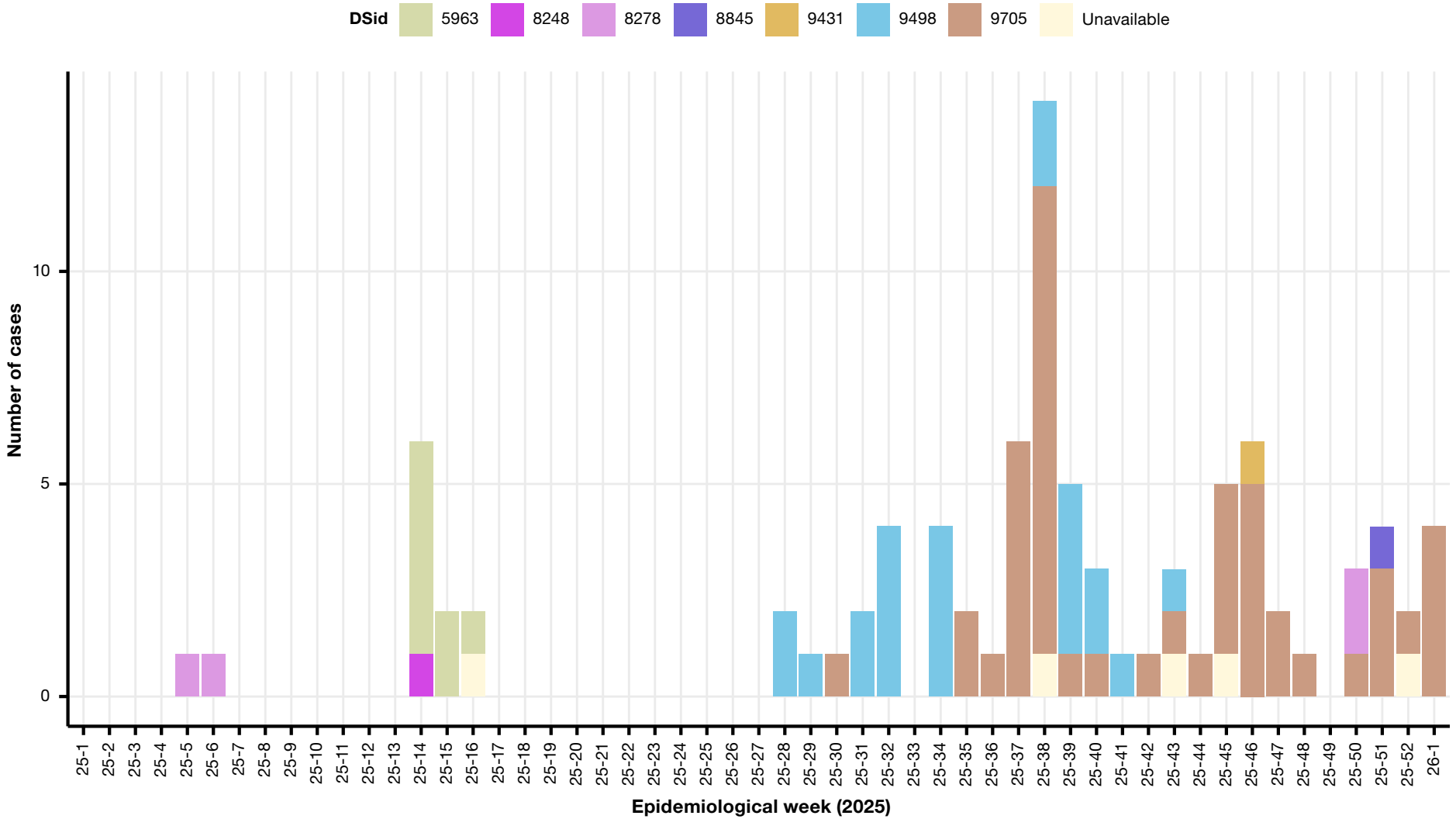
a The temporal distribution of laboratory-confirmed measles cases in Australia in 2025 by epidemiological week, stratified by genotype (B3 and D8) and jurisdiction (New South Wales (NSW), Northern Territory (NT), Queensland (Qld), South Australia (SA), Tasmania (Tas.), Victoria (Vic.) and Western Australia (WA)) is shown. Weekly case counts are displayed on the y-axis, with stacked bars representing contributions from each jurisdiction. Separate panels are shown for genotype B3 (A: upper panel) and genotype D8 (B: lower panel), allowing comparison of temporal trends and geographic distribution between the two circulating genotypes.

Figure 2: Temporal distribution of measles genotype B3 by distinct sequence identifier (DSid),[a] Australia, 2025



a Stacked histograms show the number of laboratory-confirmed measles cases (y-axis) by epidemiological week (x-axis), stratified by genotype B3. Bars are coloured by DSid, representing distinct genomic clusters, with 'Unavailable' indicating cases where a DSid was not available.

Figure 3: Temporal distribution of measles genotype D8 by distinct sequence identifier (DSid),[a] Australia, 2025



a Stacked histograms show the number of laboratory-confirmed measles cases (y-axis) by epidemiological week (x-axis), stratified by genotype D8. Bars are coloured by DSid, representing distinct genomic clusters, with 'Unavailable' indicating cases where a DSid was not available.

Among confirmed and epidemiologically linked B3 cases, 42 (53.8%) were acquired overseas, with importations predominantly from Vietnam (63.4%), followed by Indonesia (14.6%) and Pakistan (9.8%). Additional single imported cases were associated with Afghanistan, England, Morocco, Thailand, and the Philippines.

Measles genotype B3 diversity in Australia

On the basis of N450 Sanger sequencing data, the 73 genotype B3 viruses belonged to 14 distinct sequence identifiers (DSids; Figure 2). Sequences belonging to DSid 8841 predominated (n = 45; 57.7%) and were primarily associated with importations from countries within the WHO Western Pacific Region, particularly Vietnam. WGS revealed substantial diversity among DSid 8841 sequences, with up to 33 SNP differences observed (Table 1). Of the remaining 13 DSids identified amongst genotype B3 viruses, DSid 6418 accounted for 9.5% of cases, followed by DSid 9753 and 9673, representing 6.8% and 5.5% respectively. The genomic diversity among sequences belonging to each of these DSids is shown in Table 1.

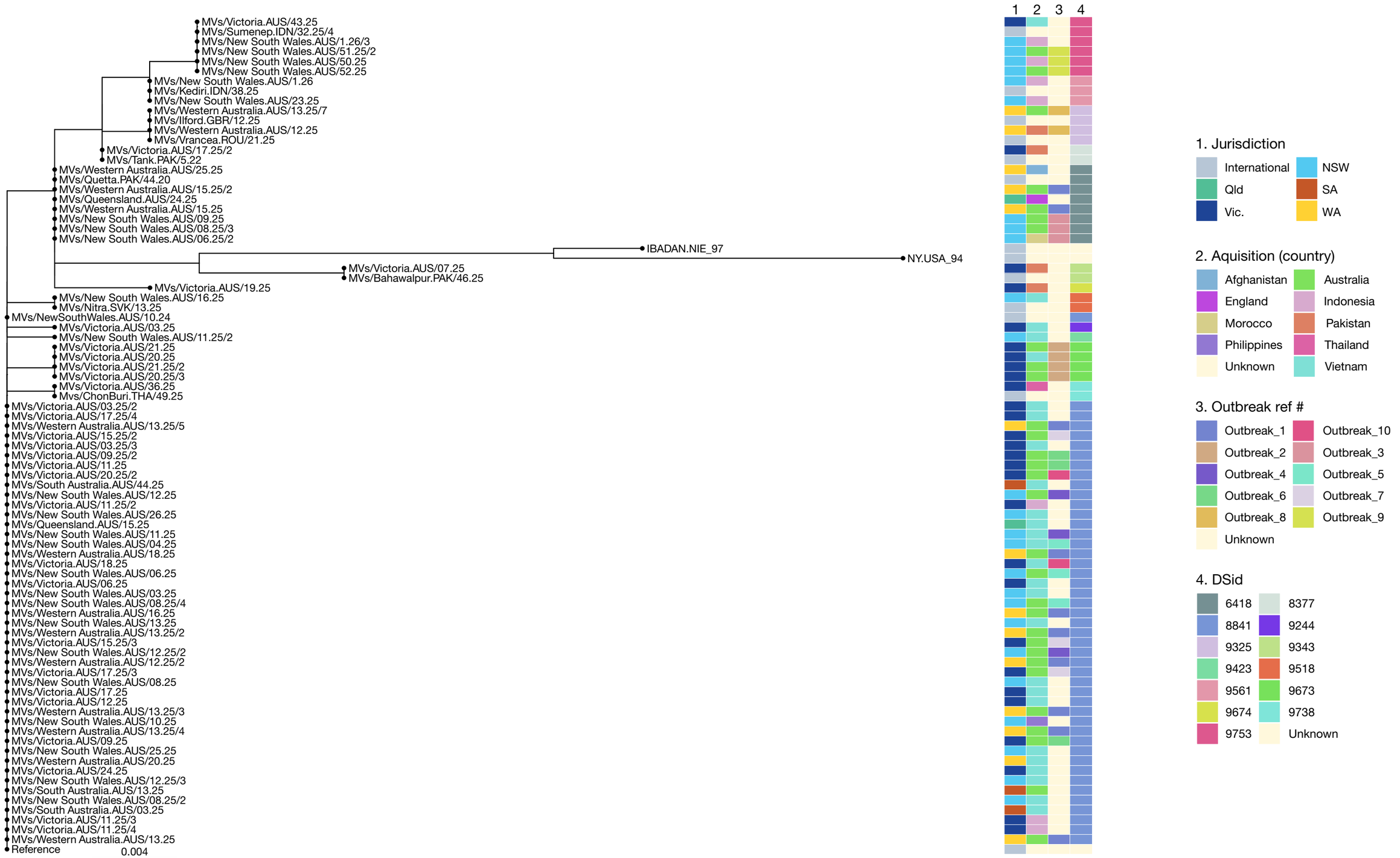
Table 1: Number of single nucleotide polymorphism (SNP) differences obtained using whole genome sequencing within measles genotype B3 and D8 samples classified by distinct sequence identifier (DSid),[a] Australia, 2025

Genotype	DSid	# genomes	Min. SNPs	Max. SNPs	Figure
B3	8841	34	0	33	5
B3	6418	4	0	15	5
B3	9673	3	0	0	5
B3	9753	3	0	16	5
D8	5963	10	0	36	7
D8	9705	10	0	14	7
D8	8248	4	0	37	7

a Only DSids from which two or more genomes were available for comparison are shown.

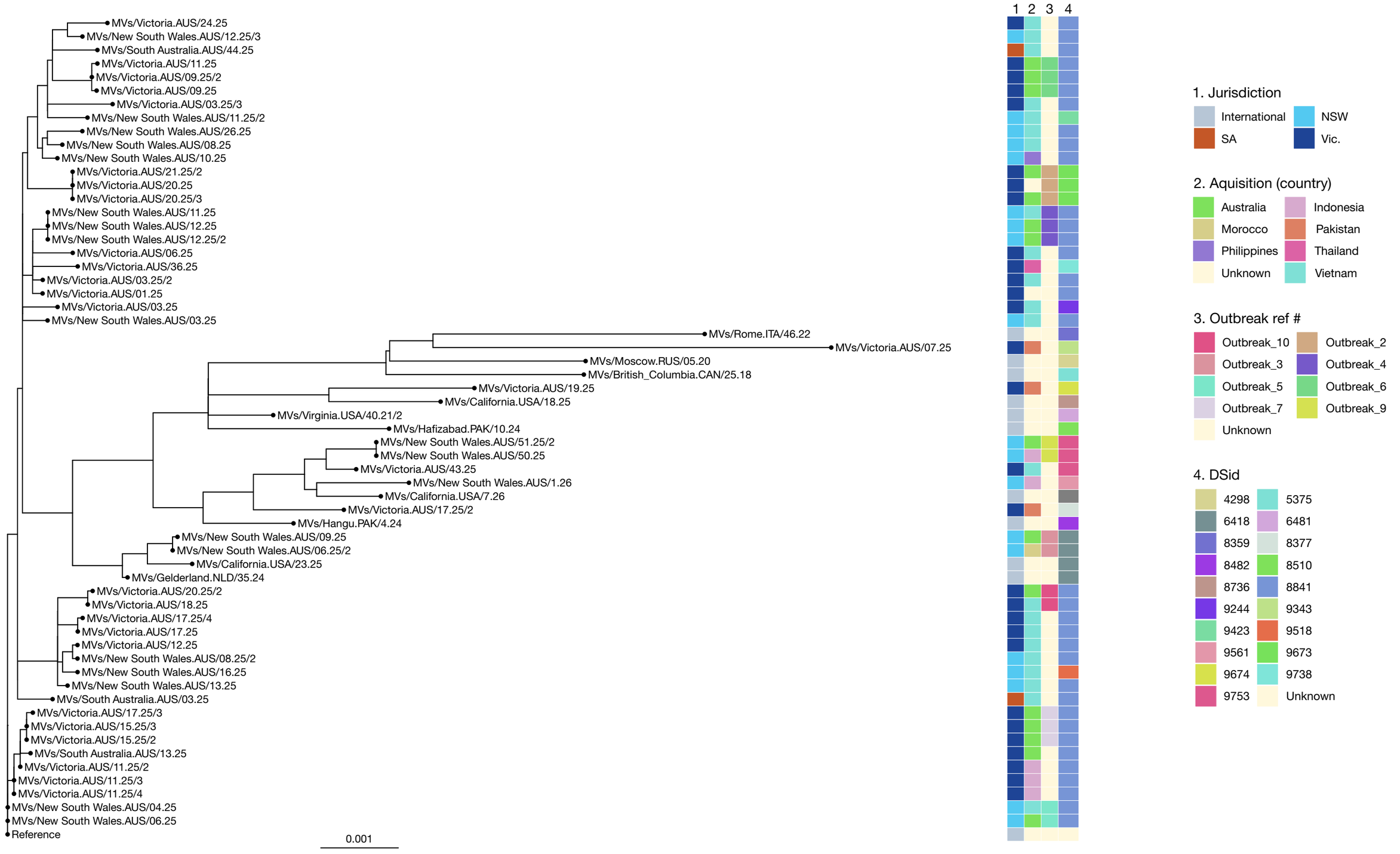
WGS results provided greater resolution than did legacy Sanger-based sequencing for distinguishing between genotype B3 sequences assigned to the same N450-DSid, as evidenced by the substantial number of SNP differences observed. Genotypic and genomic analysis of measles genotype B3 viruses showed no evidence of sustained local transmission in Australia in 2025 (Figures 1, 2, 4 and 5). Furthermore, no Australian N450 sequences represented unique lineages absent from MeaNS2; all viruses matched (or were closely related to) known internationally circulating strains, supporting an import-related origin.

Figure 4: Phylogenetic tree of measles virus genotype B3 based on N450 sequences,[a] Australia, 2025



a Australian sequences from 2025 are shown alongside reference World Health Organization and named strains obtained from the MeaNS2 database for context. Tips are labelled by strain name (location/country and epidemiological week/year). Clusters corresponding to epidemiologically defined outbreaks and distinct sequence identifiers are indicated, with colouring denoting jurisdiction [New South Wales (NSW), Queensland (Qld), South Australia (SA), Victoria (Vic.) and Western Australia (WA)], country of acquisition, outbreak reference number, and distinct sequence identifier (DSid). Branch lengths are proportional to nucleotide substitutions per site, as indicated by the scale bar. Reference sequences were selected as described in the Methods.

Figure 5: Phylogenetic tree of measles virus genotype B3 genomes generated by whole genome sequencing,[a] Australia, 2025



a Australian sequences from 2025 are shown alongside reference World Health Organization and named strains obtained from the MeaNS2 database for context. Tips are labelled by strain name (location/country and epidemiological week/year). Clusters corresponding to epidemiologically defined outbreaks and distinct sequence identifiers (DSids) are indicated, with colouring denoting jurisdiction [New South Wales (NSW), South Australia (SA) and Victoria (Vic.)], outbreak reference number, and country of acquisition. Genomes generated from confirmed measles cases detected in Western Australia and Queensland were not provided for inclusion in this report. Branch lengths are proportional to nucleotide substitutions per site, as indicated by the scale bar. Reference sequences were selected as described in the Methods.

Measles genotype D8 in Australia

Measles genotype D8 was identified in 90 of 163 N450 sequenced confirmed cases (55%). An additional four cases, for which genotyping was unsuccessful, were epidemiologically linked to D8 outbreaks (data not shown). In contrast to measles genotype B3, genotype D8 was predominantly detected in the second half of 2025 (Figures 1 and 3).

Among laboratory confirmed and epidemiologically linked measles D8 cases, 34 (36.2%) were acquired overseas, with importations predominantly from Indonesia (85.3%). Single cases were also imported into Australia from New Zealand, South Africa, Thailand and Vietnam. Additionally, no Australian N450 sequences represented unique lineages absent from MeaNS2; all viruses matched (or were closely related to) known internationally circulating strains, consistent with an import-related origin.

Measles genotype D8 diversity in Australia

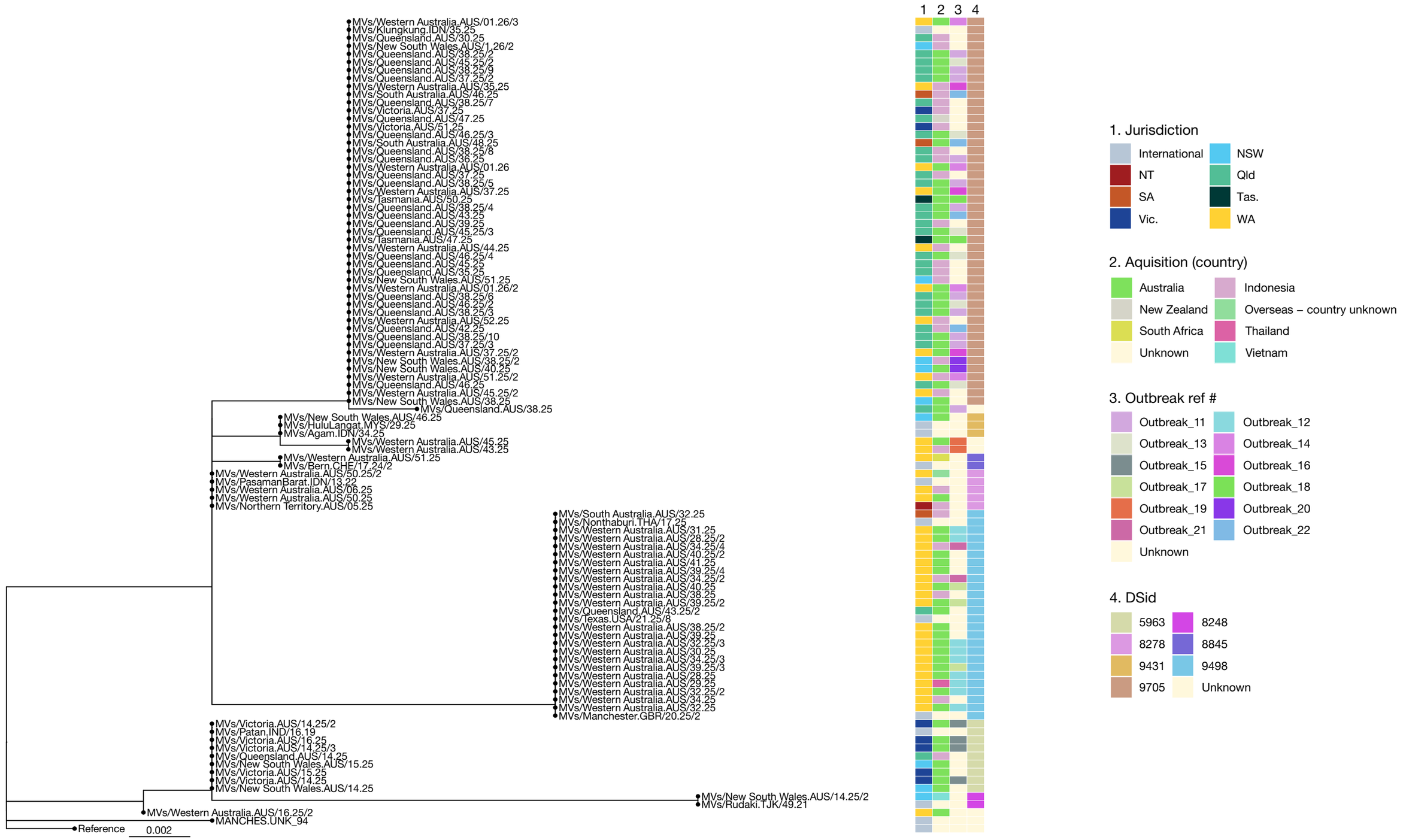
On the basis of N450 Sanger sequencing data, the 90 genotype D8 viruses belonged to seven DSids, with half of all cases belonging to predominant DSid 9705 (Figures 3, 6 and 7). DSid 9705 was primarily associated with importations from neighbouring countries, with Indonesia accounting for 95.5% of imported DSid 9705 cases. WGS revealed substantial diversity among DSid 9705 sequences, with up to 14 SNP differences observed (Table 1). Of the remaining six DSids identified amongst genotype D8 viruses, DSid 9498 accounted for 24.5% of cases, followed by DSid 5963 and DSid 8278, accounting for 8.5% and 4.3%, respectively. Cases classified as belonging to DSids 9498, 5963 and 8278 were predominantly associated with travel to Indonesia. The genomic diversity among sequences belonging to each of these DSids is shown in Table 1.

Analogous to the findings for measles genotype B3, WGS results provided greater resolution than did legacy Sanger-based sequencing for distinguishing between genotype D8 sequences assigned to the same N450-DSid, evidenced by the substantial number of SNP differences. Genotypic and genomic analysis of genotype D8 viruses found no evidence of sustained local transmission in Australia in 2025 (Figures 3, 6 and 7). Additionally, no novel variants were detected; all strains matched (or were closely related to) known internationally circulating strains, consistent with an import-related origin.

Unknown/unsuccessful measles genotype

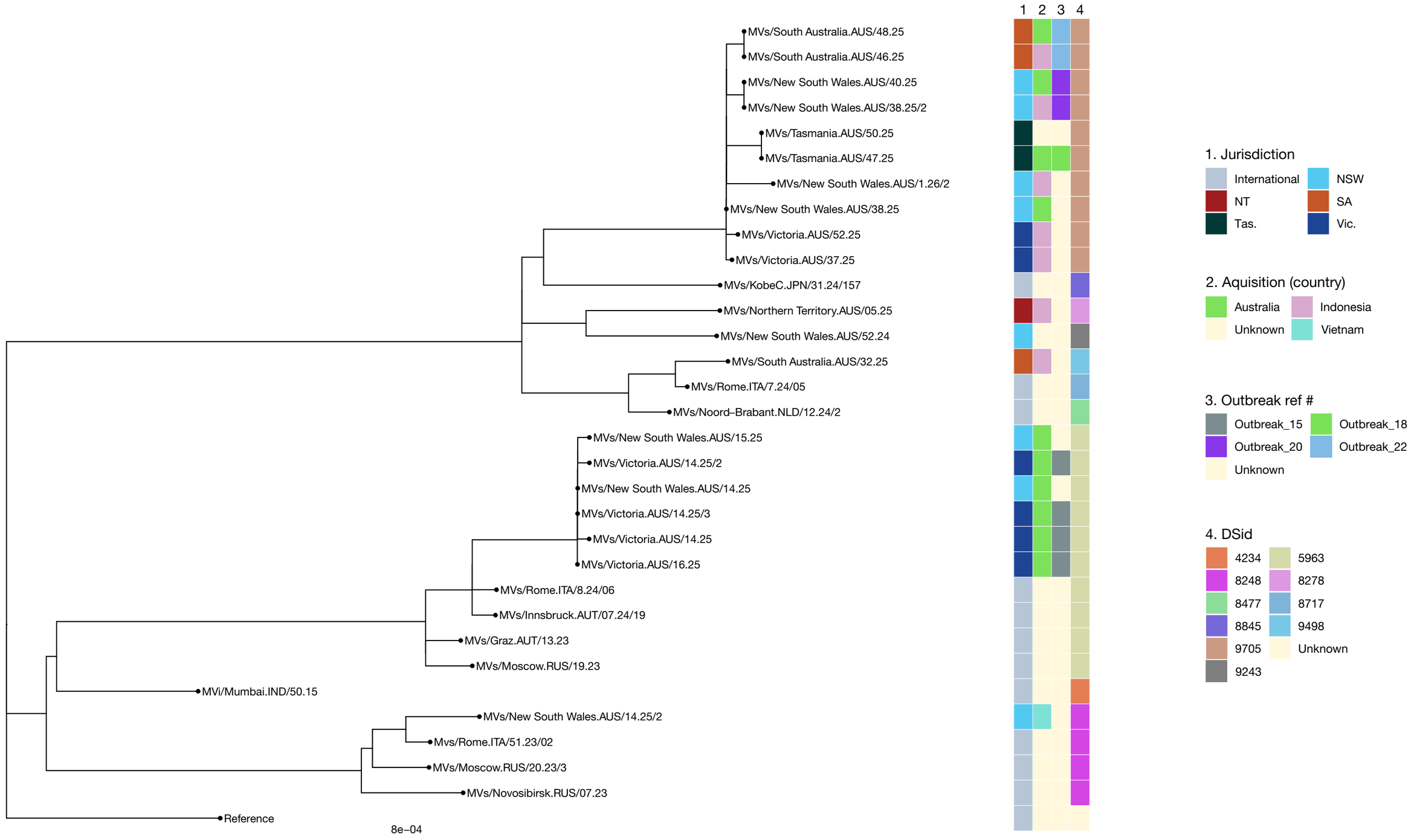
A genotype could not be assigned for 18 measles cases. Apart from one serologically diagnosed case, for which viral load was unknown, 17/18 samples had low viral loads that precluded successful genotyping or lacked sufficient remaining material for further analysis. Of the 17 cases from which low viral load/insufficient volume samples were provided to VIDRL, four were acquired overseas; eight were epidemiologically linked to a known outbreak; and the source of infection was unknown for five cases.

Figure 6: Phylogenetic tree of measles virus genotype D8 based on N450 sequences,[a] Australia, 2025



a Australian sequences from 2025 are shown alongside reference World Health Organization and named strains obtained from the MeaNS2 database for context. Tips are labelled by strain name (location/country and epidemiological week/year). Clusters corresponding to epidemiologically defined outbreaks and distinct sequence identifiers are indicated, with colouring denoting jurisdiction [New South Wales (NSW), Northern Territory (NT), Queensland (Qld), South Australia (SA), Tasmania (Tas.), Victoria (Vic.) and Western Australia (WA)], country of acquisition, outbreak reference number, and distinct sequence identifier (DSid). Branch lengths are proportional to nucleotide substitutions per site, as indicated by the scale bar. Reference sequences were selected as described in the Methods.

Figure 7: Phylogenetic tree of measles virus genotype D8 genomes generated by whole genome sequencing,[a] Australia, 2025



a Australian sequences from 2025 are shown alongside reference World Health Organization and named strains obtained from the MeaNS2 database for context. Tips are labelled by strain name (location/country and epidemiological week/year). Clusters corresponding to epidemiologically defined outbreaks and distinct sequence identifiers (DSids) are indicated, with colouring denoting jurisdiction [New South Wales (NSW), Northern Territory (NT), South Australia (SA), Tasmania (Tas.) and Victoria (Vic.)], outbreak reference number, and country of acquisition. Genomes generated from confirmed measles cases detected in Western Australia and Queensland were not provided for inclusion in this report.

Measles outbreaks

Thirty-one distinct, epidemiologically confirmed measles outbreaks were identified in Australia in 2025 (Figures 4–7). Of the 31 outbreaks, 13 were attributed to genotype B3 and 17 to genotype D8. A genotype was unavailable/unable to be determined for one outbreak, from which only a single sample was referred to VIDRL. A sample or sequence from two or more epidemiologically linked cases was available for 22 of the 31 distinct outbreaks. The two or more sequences obtained from cases linked to these 22 outbreaks (ten B3 and twelve D8 outbreaks) are annotated as outbreak sequences in the heatmaps accompanying Figures 4–7. Whilst the sole sequences obtained from each of the remaining nine outbreaks are included in the phylogenetic trees, they are not annotated as outbreak sequences in the heatmaps accompanying Figures 4–7.

Thirty-six genotype B3 N450 sequences were collectively identified as belonging to 13 distinct outbreaks in 2025. Of the 13 outbreaks, a sample or sequence was submitted to VIDRL from two or more cases for 10 outbreaks. Of the latter 10 genotype B3 outbreaks, sequences from six outbreaks were classified as belonging to DSid 8841. Despite up to 33 SNP differences observed between all the 2025 DSid 8841 sequences by whole genome sequencing (Table 1), sequences obtained from cases belonging to each outbreak differed by no more than two SNPs (where WGS data were available: Table 2). Genomic and epidemiological data were concordant where both were available, highlighting the enhanced resolution and concordance with epidemiological information provided by WGS data relative to conventional DSid designation in the context of measles outbreak investigation.

Table 2: Number of single nucleotide polymorphism (SNP) differences obtained using whole genome sequencing between epidemiologically linked measles genotype B3 and D8 outbreak cases,[a] Australia, 2025

Outbreak reference	Genotype (DSid)[b]	# N450 sequences	# genomes	Min. SNPs	Max. SNPs	Figure
Outbreak 2	B3 (9673)	4	3	0	0	5
Outbreak 3	B3 (6418)	3	2	0	1	5
Outbreak 4	B3 (8841)	3	3	0	0	5
Outbreak 5	B3 (8841)	3	2	0	0	5
Outbreak 6	B3 (8841)	3	3	0	2	5
Outbreak 7	B3 (8841)	3	3	0	1	5
Outbreak 10	B3 (8841)	2	2	0	1	5
Outbreak 15	D8 (5963)	4	4	0	4	7
Outbreak 20	D8 (9705)	2	2	0	0	7
Outbreak 22	D8 (9705)	4	2	0	0	7

a Only outbreaks from which two or more genomes were available from linked cases for comparison are shown.

b DSid: distinct sequence identifier.

Of the 49 genotype D8 N450 sequences collectively identified as belonging to 17 distinct outbreaks in 2025, a sample or sequence was available from two or more cases for 12 D8 outbreaks. Sequences from six outbreaks were classified as belonging to DSid 9705. Despite up to 14 SNP differences observed between all the 2025 DSid 9705 sequences by WGS (Table 1), zero SNPs were identified between DSid 9705 sequences obtained from cases belonging to epidemiologically distinct Outbreaks 20 and 22 (Table 2). Genomic and epidemiological data were concordant where both were available. Indeed, for both genotypes B3 and D8, epidemiologically linked outbreak cases were interspersed within broad phylogenetic clusters consisting of unrelated samples sharing the same N450 DSid (Figures 4 and 6) compared to tight, epidemiologically concordant phylogenomic clusters following WGS (Figures 5 and 7). Conversely, as distinct DSids are assigned to measles virus N450 sequences on as little as a single SNP difference, the limited discriminatory power of distinct DSids assigned using N450 region compared to analysis of complete genomes is evidenced by the dispersion of DSids throughout the heatmaps in Figures 5 and 7.

The WGS results presented in this report quantitatively demonstrated greater discriminatory resolution than N450-based Sanger sequencing. Genomes obtained from cases belonging to epidemiologically distinct outbreaks that were assigned the same N450 DSid differed by as many as 33 SNPs for genotype B3 DSid 8841 and 36 SNPs for genotype D8 DSid 5963 (Table 1), whereas genomes obtained from epidemiologically linked outbreak cases differed by 0–4 SNPs (Table 2). Genomic and epidemiological data were concordant for outbreak cases where both data types were available, demonstrating the additional resolution provided by WGS for distinguishing epidemiologically linked from unrelated cases that cannot be reliably differentiated using N450 DSid assignment alone. The data presented here highlight the potential public health utility of WGS in confirming epidemiological links between cases, as well as in differentiating local transmission from importation events for cases of unknown origin, both within and between jurisdictions, when combined with epidemiological data. Whilst SNP-based thresholds to determine genomic relatedness between measles cases are yet to be established, either within Australia or internationally, thresholds based on genomic epidemiological evidence are being prospectively trialled at VIDRL. Routine WGS of retrospective and prospective measles cases will be essential to develop robust, SNP-based thresholds to accurately inform the public health management of measles in Australia.

Rubella molecular epidemiology, Australia, 2025

The prevalence of rubella virus infection in Australia remains extremely low. At VIDRL, 157 samples were tested for rubella virus RNA by real-time PCR, with no detections identified. However, two rubella cases were reported to the National Notifiable Diseases Surveillance System in 2025. Genotyping data were unavailable for these cases, as rubella virus was detected in vitreous fluid specimens and no residual sample remained for referral and genotyping. Detection of rubella virus in ocular specimens is more consistent with chronic or persistent infection than with acute rubella infection. Consequently, no Australian rubella virus sequences from cases notified in 2025 were available for submission to or comparison with the WHO GMRLN RubeNS2 database.[3] Despite the absence of genotyping information, these findings underscore the continued rarity of rubella in Australia.

Limitations

There were notable limitations to the analysis presented in this report. Most importantly, whole genome sequences from confirmed measles cases detected in Queensland and Western Australia were not available for inclusion in the national genomic analysis. These jurisdictions accounted for 53.6% of notified measles cases in Australia in 2025, limiting the completeness of national phylogenomic analyses and the extent to which genomic findings can be generalised to all Australian cases. In addition, SNP-based thresholds for defining genomic relatedness between measles cases have not yet been established nationally or internationally; therefore, genomic distances were interpreted in an epidemiological context rather than using a predefined SNP threshold.

Conclusions

The findings presented in this report confirm that outbreak-associated measles cases were either imported, directly linked to importations, or associated with import-related sequences. Despite the increase in measles case notifications through the year, the available genomic evidence revealed the presence of discrete, distinct outbreak events, supporting the absence of endemic measles transmission in Australia in 2025.

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