



SURVEILLANCE REPORT

Influenza virus characterisation

Summary Europe, November 2018

Summary

This is the second report for the 2018–19 influenza season. As of week 48/2018, only 2 061 influenza detections across the WHO European Region had been reported. Detections were made up of 90.4% type A viruses, with A(H1N1)pdm09 prevailing over A(H3N2), and 9.6% type B viruses, with 10 (90.9%) of 11 ascribed to a lineage being B/Yamagata.

One EEA country, Norway, has shared influenza-positive specimens with the London WHO CC, the Francis Crick Worldwide Influenza Centre (WIC), since week 40/2018. Hence, the main focus of this report is phylogenetic analyses of HA genes of globally collected seasonal influenza viruses with recent collection dates (1 June 2018 or later) that international laboratories had submitted to the GISAID EpiFlu database by 10 December 2018.

HA genes of A(H1N1)pdm09 viruses continue to fall within subclade 6B.1, defined by HA1 amino acid substitutions S84N, S162N and I216T, represented by the vaccine virus A/Michigan/45/2015, with all recently circulating viruses having additional substitutions of S74R, S164T and I295V. The six characterised viruses from Norway all fell in subclade 6B.1 and were antigenically similar to the vaccine virus.

The great majority of recently circulating A(H3N2) viruses have HA genes falling in subclade 3C.2a2 and subgroup 3C.2a1b, as seen for the seven viruses characterised by the WIC (six 3C.2a1b and one 3C.2a2). Due to a lack of HA activity, the seven viruses could not be characterised antigenically by HI assay; however, based on previous HI analyses performed with post-infection ferret antisera raised against similar viruses, those raised against subgroup 3C.2a1b viruses induced responses that gave cross-subclade reactivity while those raised against subclade 3c.2a2 viruses gave more subclade-specific reactivity.

All 80 B/Victoria-lineage viruses with collection dates from 1 June 2018 had HA genes that encoded HA1 amino acid substitutions of I117V, N129D and V146I and fell within a subclade of clade 1A (the B/Brisbane/60/2008 clade). However, 16 and 49 of these, respectively, fell in groups defined by deletions of three (Δ 162-164) or two (Δ 162-163) amino acids in HA1. Previous HI analyses with panels of post-infection ferret antisera have shown these three groups of viruses to be antigenically distinguishable and a Δ 162-163 virus, B/Colorado/06/2017, has been recommended for use in trivalent vaccines for the current northern and upcoming southern hemisphere influenza seasons.

All 73 B/Yamagata-lineage viruses with collection dates from 1 August 2018 had HA genes that fell within the B/Phuket/3073/2013 vaccine virus clade (clade 3) and encoded HA1 amino acid substitutions of L172Q and M251V, with some having additional substitutions. Previous HI analyses with post-infection ferret antisera raised against B/Phuket/3073/2013 have shown such viruses to be antigenically similar to the vaccine virus which has been recommended for use in quadrivalent vaccines for the current northern and upcoming southern hemisphere influenza seasons.

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Table 1 shows a summary of influenza virus detections in the WHO European Region reported to ECDC's TESSy database since the start of the 2018–19 season (weeks 40–48/2018). As is usual for this time of the year, a low number (2 061) of detections have been reported, with type A (90.4%) predominating over type B (9.6%) viruses, which is a common pattern, unlike the 2017–18 season when type B predominated over type A at the start of the season and throughout most of it. Of the type A viruses subtyped ($n = 817$) and the type B viruses ascribed to a lineage ($n = 11$), A(H1N1)pdm09 ($n = 517$) are prevailing over A(H3N2) ($n = 300$) viruses, and 10 of 11 B viruses have been B/Yamagata-lineage; these relative proportions are comparable to those summarised in the October 2018 characterisation report¹. Overall, the ratio of type A to type B detections is significantly increased compared with the 2017–18 season (0.8:1 to 9.4:1), and of the influenza A viruses that have been subtyped, an increase in the proportion of A(H1N1)pdm09 has been seen (63.3% in 2018–19 compared with 50.6% in 2017–18).

Since week 40/2018, a single shipment of specimens has been received at the Crick Worldwide Influenza Centre (WIC) from an EU/EEA country (Norway). Consequently, limited antigenic characterisation data has been generated for viruses in EU/EEA countries and this report contains haemagglutinin (HA) phylogenetic analyses for seasonal influenza viruses, with the most recent collection dates, that have been deposited in the GISAID EpiFlu database (as of 10 December 2018). These phylogenetic analyses serve to illustrate the recent global situation as an indicator of what may emerge in the WHO European Region.

Table 1. Influenza virus detections in the WHO European Region from the start of reporting for the 2018–19 season (weeks 40–48/2018)^a

Virus type/subtype/lineage	Cumulative number of detections			Totals*		Totals for 2017-18 season*		
	Sentinel sources	Non-sentinel sources	Totals	%	Ratios	Number	%	Ratios
Influenza A	147	1 716	1863	90.4	9.4:1	106 003	44.1	0.8:1
A(H1N1)pdm09	79	438	517	63.3		23 121	50.6	
A(H3N2)	62	238	300	36.7	0.6:1	22 568	49.4	1:1
A not subtyped	6	1 040	1 046			60 314		
Influenza B	12	186	198	9.6		134 618	55.9	
Victoria lineage	1	0	1	9.1		301	1.9	
Yamagata lineage	4	6	10	90.9	10:1	15 701	98.1	52.2:1
Lineage not ascribed	7	180	187			118 616		
Total detections (total tested)	159 (6 128)	1 902 (112 647)	2 061 (118 775)			240 621 (903 182)		

* Percentages are shown for total detections (types A & B [in bold type], and for viruses ascribed to influenza A subtype and influenza B lineage). Ratios are given for type A:B [in bold type], A(H3N2):A(H1N1)pdm09 and Yamagata:Victoria lineages.

Table 2. Summary of clinical samples and virus isolates, contained in packages received from EU/EEA Member States since week 40/2018

MONTH Country	Total Number received	A		H1N1pdm09		H3N2		B		B Victoria lineage		B Yamagata lineage		
		Number received	Number propagated ¹	Number received	Number propagated ¹	Number received	Number propagated ²	Number received	Number propagated ¹	Number received	Number propagated ¹	Number received	Number propagated ¹	
2018														
SEPTEMBER														
OCTOBER														
Norway	26			11	in process	12	0	7				3	in process	
NOVEMBER														
Norway	2					2	0	1						
	28	0	0	11	0	14	0	8	0	0	0	0	3	0
1 Country				39.3%		50.0%					0.0%		10.7%	
				89.3%							10.7%			

1. Propagated to sufficient titre to perform HI assay (the totalled number does not include any from batches that are in process)

2. Propagated to sufficient titre to perform HI assay in the presence of 20nM oseltamivir (the totalled number does not include any from batches that are in process)

Numbers in red indicate viruses recovered but with insufficient HA titre to permit HI assay

* As of 2018-12-10

¹ European Centre for Disease Prevention and Control. Influenza virus characterisation, summary Europe, October 2018. Stockholm: ECDC; 2018. Available from: <https://ecdc.europa.eu/sites/portal/files/documents/ECDCFlu-characterisation-rep-Oct-2018.pdf>

Influenza A(H1N1)pdm09 virus analyses

Results of haemagglutination inhibition (HI) analyses of viruses performed since the October 2018 report are shown in Table 3. All six A(H1N1)pdm09 test viruses antigenically characterised were similar to the vaccine virus for the northern hemisphere 2018–19 influenza season, A/Michigan/45/2015 [1], being recognised at titres within twofold of the titre of the antiserum for the homologous virus. Five of the test viruses showed good reactivity with eight other antisera in the panel, all being recognised at titres within twofold of the respective homologous titres. The antiserum raised against cell culture-propagated A/Lviv/N6/2009 (which has HA1 amino acid substitutions of G155E and D222G) recognised the test viruses less well, with three of six viruses being recognised at titres that were at least eightfold reduced compared with the homologous titre. A/Norway/3283/2018 showed the highest fold reductions, compared with homologous titres, with all antisera in the panel and carried HA1 amino acid polymorphism at HA1 position 190 (S190S/R).

All viruses with collection dates from 1 October 2018, for which HA gene sequences have been submitted to the GISAID EpiFlu database, fall within clade 6B.1, the A/Michigan/45/2015 vaccine virus clade [1], but carry additional amino acid substitutions of S74R, S164T and I295V (Figure 1). A number of genetic subgroups defined by specific amino acid substitutions have emerged, but the great majority of viruses in the various subgroups have remained antigenically similar to A/Michigan/45/2015 as shown in the September 2018² and earlier characterisation reports.

Of the 66 viruses from EU/EEA countries with collection dates in October and November 2018 (four from France, two from the Netherlands, five from Norway, 12 from Sweden, and 43 from Wales) featured in Figure 1:

- 23 fall in a subgroup defined by **L161I** in **HA1** with **I77M**, **N128T** and **D174E** in **HA2**;
- one within a subgroup defined by **I286V** in **HA1** with **I45V** in **HA2**;
- one within a subgroup defined by **S183P** and **L233I** in **HA1** and **V193A** in **HA2**;
- one within a subgroup defined by **N129D**, **A141E** and **S183P** in **HA1**;
- seven within a subgroup defined by **S183P** and **K302T** in **HA1** and **I77M**, **N196S** and **E179D** in **HA2**;
- one within a subgroup defined by **R45G**, **H126Y**, **S183P**, **P282A** and **I298V** in **HA1**;
- four within a subgroup defined by **T120A** and **S183P** in **HA1**;
- one within a subgroup defined by **S183P**, **E235D**, **N260D** in **HA1** and **V193A** in **HA2**; and
- 27 within a subgroup defined by **N129D**, **S183P**, **T185I**, **N260D** in **HA1**, with 18 from Wales also carrying **V152I** in **HA2**.

Subgroups 1 and 9 are predominantly populated by viruses from Wales. Sequences of recently circulating viruses from countries outside of the European Region predominantly fall outside of subgroups 1 and 9.

² European Centre for Disease Prevention and Control. Influenza virus characterisation, summary Europe, September 2018. Stockholm: ECDC; 2018. Available from: <https://ecdc.europa.eu/sites/portal/files/documents/ECDC-Flu-Characterisation-Report-Sep-2018.pdf>

Table 3. Antigenic analysis of A(H1N1)pdm09 viruses by HI

Viruses	Other information	Collection date	Passage history	Haemagglutination inhibition titre											
				Post-infection ferret antisera											
				A/Cal 7/09 Egg F07/16 ⁻¹ F07/16 ⁻¹ 6B.1	A/Bayern 69/09 MDCK F09/15 ⁻¹	A/Lviv N6/09 MDCK F14/13 ⁻¹ F14/13 ⁻¹	A/Astrak 1/11 MDCK F22/13 ⁻¹ F22/13 ⁻¹	A/HK 5659/12 MDCK F17/15 ⁻¹ F17/15 ⁻¹	A/Slov 2903/2015 Egg F02/16 ⁻¹ F02/16 ⁻¹	A/Paris 1447/17 MDCK F03/18 ⁻² F03/18 ⁻²	A/Swit 2656/17 Egg F20/18 ⁻¹ F20/18 ⁻¹	A/Swit 3330/17 Egg F23/18 ⁻¹ F23/18 ⁻¹	6B.1	6A	5
Passage history	6B.1	6B.1	6B.1	6B.1	6B.1	6B.1	6B.1	6B.1	6B.1	6B.1	6B.1				
REFERENCE VIRUSES															
A/Michigan/45/2015		2015-09-07	E3/E3	1280	320	320	640	640	640	1280	2560	1280	640	640	
A/California/7/2009	clone 38-32	2009-04-09	E3/E3	1280	320	320	320	640	640	1280	1280	1280	640	320	
A/Bayern/69/2009	G155E	2009-07-01	MDCK5/MDCK1	80	320	320	40	40	40	80	320	80	80	80	
A/Lviv/N6/2009	G155E, D222G	2009-10-27	MDCK4/SIAT1/MDCK3	160	1280	1280	40	160	160	320	640	320	160	160	
A/Astrakhan/1/2011		2011-02-28	MDCK1/MDCK5	1280	320	320	320	640	640	1280	1280	640	640	320	
A/Hong Kong/5659/2012		2012-05-21	MDCK4/MDCK2	1280	320	320	320	320	640	640	1280	1280	320	320	
A/Slovenia/2903/2015	clone 37	2015-10-26	E4/E2	1280	320	320	320	640	640	1280	2560	1280	640	320	
A/Paris/1447/2017		2017-10-20	MDCK1/MDCK3	640	320	160	320	320	320	1280	1280	1280	640	320	
A/Switzerland/2656/2017		2017-12-21	E5/E2	1280	640	320	640	640	640	1280	2560	1280	640	640	
A/Switzerland/3330/2017	clone 35	2017-12-20	E6/E2	640	320	160	320	640	640	1280	1280	1280	640	640	
TEST VIRUSES															
A/Norway/3282/2018		2018-10-08	MDCK1/MDCK1	1280	320	160	320	640	640	1280	2560	1280	640	320	
A/Norway/3283/2018		2018-10-12	MDCK1/MDCK1	320	160	80	80	160	160	640	640	640	320	160	
A/Norway/3316/2018		2018-10-19	MDCK1	640	320	160	320	320	320	640	1280	1280	640	320	
A/Norway/3415/2018		2018-10-30	MDCK1	640	320	320	160	320	320	640	1280	1280	640	320	
A/Norway/3433/2018		2018-10-30	MDCK1	1280	320	320	320	640	640	1280	2560	1280	640	640	
A/Norway/3401/2018		2018-10-31	MDCK1	640	320	320	320	640	640	1280	2560	1280	640	320	
			Vaccine												

* Superscripts refer to antiserum properties (< relates to the lowest dilution of antiserum used)
 1 <= <40; 2 <= <80

Sequences in phylogenetic trees

Figure 1. Phylogenetic comparison of influenza A(H1N1)pdm09 HA genes

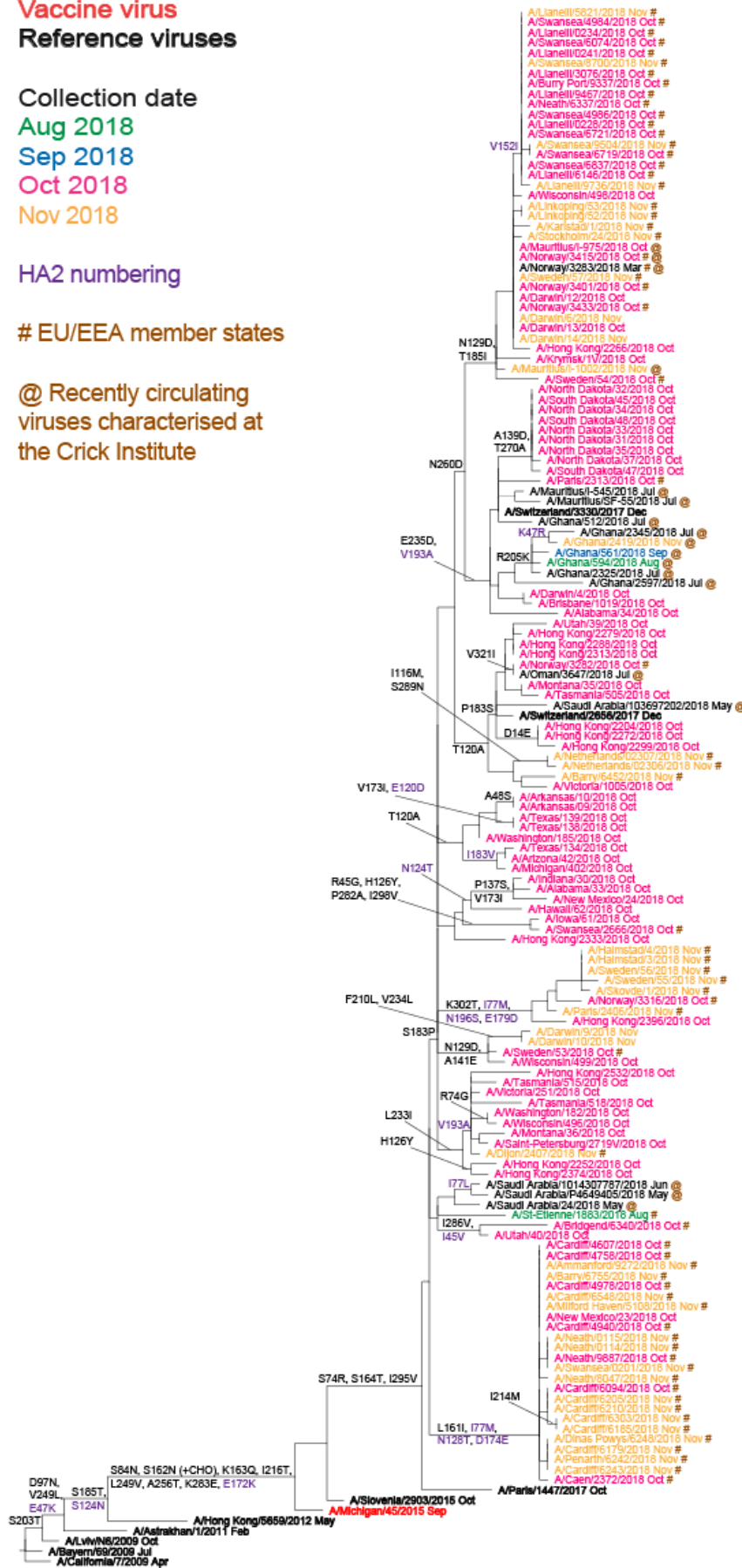
Vaccine virus
Reference viruses

Collection date
Aug 2018
Sep 2018
Oct 2018
Nov 2018

HA2 numbering

EU/EEA member states

@ Recently circulating viruses characterised at the Crick Institute



6B.1

Influenza A(H3N2) virus analyses

None of the eight viruses from Norway with recent collection dates retained sufficient haemagglutinating (HA) activity to permit antigenic analysis by HI assay (Table 2).

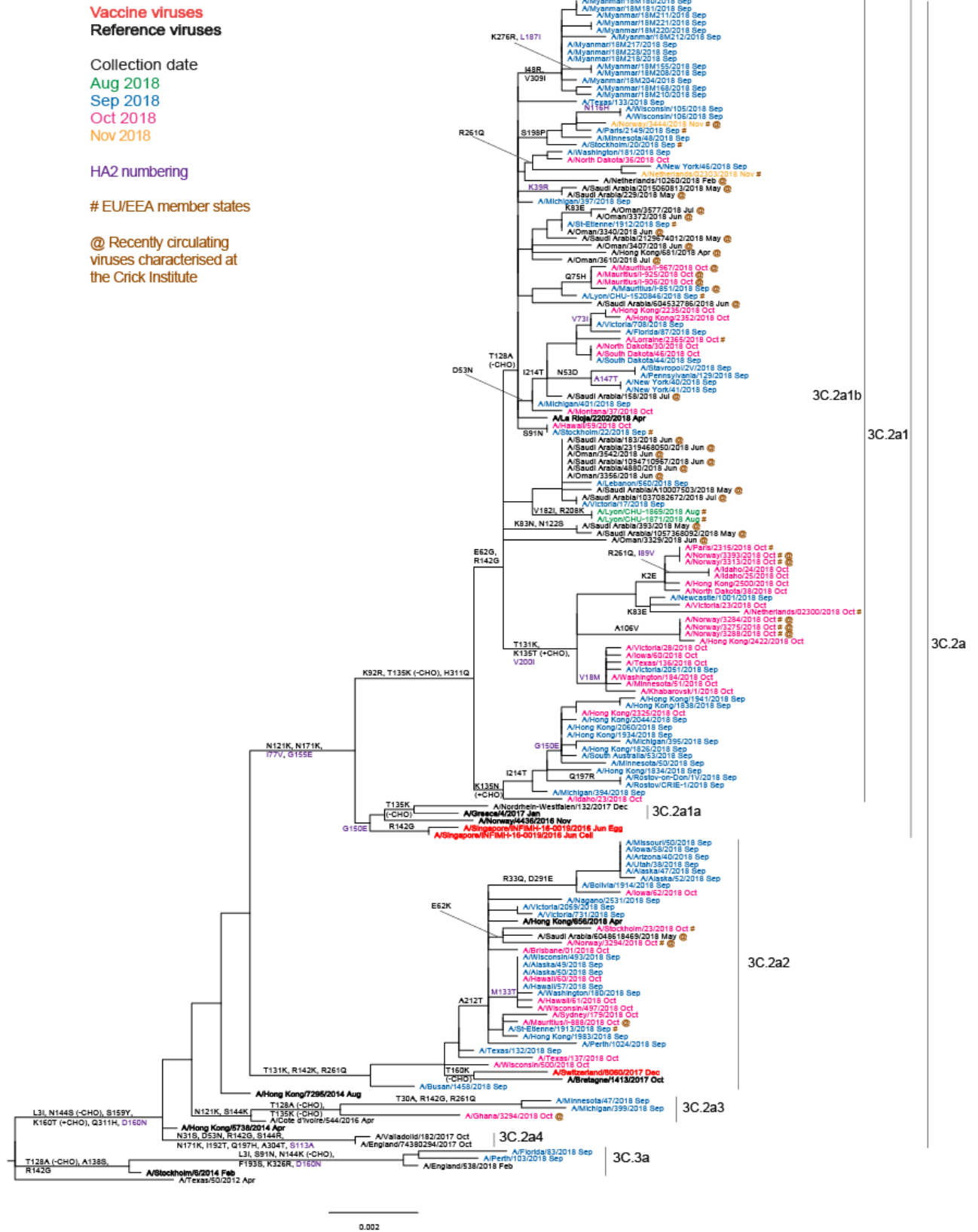
Phylogenetic analysis of HA genes of A(H3N2) viruses with collection dates from 1 September 2018, available in the GISAID EpiFlu database, is shown in Figure 2. Viruses in clades 3C.2a and 3C.3a have been in circulation since the 2013–14 northern hemisphere influenza season, with clade 3C.2a viruses predominating since the 2014–15 influenza season and continuing to predominate in recent months (Figure 2) but the HA gene sequences continue to diverge. Notably, clade 3C.3a viruses have evolved to carry **HA1** amino acid substitutions of **L3I**, **S91N**, **N144K** (loss of a N-linked glycosylation motif at residues 144–146), **F193S** and **K326R**, compared to A/Stockholm/6/2014 (Figure 2), and new genetic groups have emerged among the clade 3C.2a viruses, designated as subclades/subgroups. Amino acid substitutions that define these subclades/subgroups are:

- Clade 3C.2a: **L3I**, **N144S** (resulting in the loss of a potential glycosylation site), **F159Y**, **K160T** (in the majority of viruses, resulting in the gain of a potential glycosylation site) and **Q311H** in **HA1**, and **D160N** in **HA2**, e.g. A/Hong Kong/7295/2014 a cell culture-propagated surrogate for A/Hong Kong/4801/2014 (a former vaccine virus)
- Subclade 3C.2a1: Those in clade 3C.2a plus: **N171K** in **HA1** and **I77V** and **G155E** in **HA2**, most also carry **N121K** in **HA1**, e.g. A/Singapore/INFIMH-16-0019/2016 (2018–19 northern hemisphere vaccine virus)
- Subgroup 3C.2a1a: Those in subclade 3C.2a1 plus **T135K** in **HA1**, resulting in the loss of a potential glycosylation site, and also **G150E** in **HA2**, e.g. A/Greece/4/2017
- Subgroup 3C.2a1b: Those in subclade 3C.2a1 plus **K92R** and **H311Q** in **HA1**, e.g. A/La Rioja/2202/2018, with many viruses in this subgroup carrying additional HA1 amino acid substitutions
- Subclade 3C.2a2: Those in clade 3C.2a plus **T131K**, **R142K** and **R261Q** in **HA1**, e.g. A/Switzerland/8060/2017 (2019 southern hemisphere vaccine virus)
- Subclade 3C.2a3: Those in clade 3C.2a plus **N121K** and **S144K** in **HA1**, e.g. A/Cote d'Ivoire/544/2016
- Subclade 3C.2a4: Those in clade 3C.2a plus **N31S**, **D53N**, **R142G**, **S144R**, **N171K**, **I192T**, **Q197H** and **A304T** in **HA1** and **S113A** in **HA2**, e.g. A/Valladolid/182/2017
- Clade 3C.3a: **T128A** (resulting in the loss of a potential glycosylation site), **R142G** and **N145S** in **HA1** which defined clade 3C.3 plus **A138S**, **F159S** and **N225D** in **HA1**, many with **K326R**, e.g. A/England/538/2018.

Globally, the great majority of viruses with collection dates from 1 September 2018 have HA genes that continue to fall into genetic groups within clade 3C.2a, notably the 3C.2a2 subclade and the 3C.2a1b subgroup, with a small number of viruses falling in clade 3C.3a (Figure 2). Viruses in subgroup 3C.2a1b have been more numerous (10 from EU/EEA countries) than those in subclade 3C.2a2 (two from EU/EEA countries) for the period October to November 2018; notably, seven of the subgroup 3C.2a1b viruses (five from Norway, one from France and one from the Netherlands) have fallen in a recently emerged cluster defined by substitutions **T131K** and **K135T** (a reversion resulting in re-establishment of the **133-135** glycosylation sequon) in **HA1** with **V200I** in **HA2**. Antigenic characterisation of viruses in this cluster is currently unavailable.

The location of A/Singapore/INFIMH-16-0019/2016 (3C.2a1), the A(H3N2) virus recommended for inclusion in vaccines for the northern hemisphere 2018–2019 influenza seasons [1], is indicated in Figure 2, as is A/Switzerland/8060/2017 (3C.2a2), the A(H3N2) virus recommended for inclusion in vaccines for the southern hemisphere 2019 [2].

Figure 2. Phylogenetic comparison of influenza A(H3N2) HA genes



Influenza B virus analyses

Influenza B – Victoria lineage

No B/Victoria-lineage HA gene sequences from EU/EEA countries or the wider WHO European Region, for viruses with collection dates from 1 June 2018, have been deposited in the GISAID EpiFlu database, and only small numbers have been deposited from elsewhere (Figure 3). However, the 44 viruses of this lineage with collection dates from 1 July 2018 continue to have HA genes that fall in the B/Brisbane/60/2008 clade (clade 1A; Figure 3), with all falling in a subcluster defined by **HA1** amino acid substitutions **I117V**, **N129D** and **V146I** within clade 1A. Two new groups within this cluster have deletions in the HA gene. A major group seen in Europe, the Americas, Asia, Oceania and South Africa have HA genes encoding an HA with deletion of residues **K162** and **N163** of **HA1** (1A(Δ 2) in Figure 3). These viruses have additional substitutions of **D129G** and **I180V** in **HA1**, and **R151K** in **HA2**. This group of viruses is more prevalent than the subcluster viruses that show no deletions. Less common are viruses with HA genes encoding a deletion of three **HA1** amino acids, **K162**, **N163** and **D164** (1A(Δ 3) in Figure 3), which have been detected primarily in the Far East and Africa (with no detections in the WHO European Region at the time of this report), many of which carry additional substitutions of **I180T** and **K209N** in **HA1**. Other members of the 1A(Δ 3) group carry the **HA1** substitution **K136E**, often with additional HA1 substitutions of **K52N** and **E198G** (within the **197-199** glycosylation site), notably for a batch of viruses from Cote d'Ivoire which have been characterised by WHO CC London. The September 2018 characterisation report – and previous ones – noted that the clade 1A viruses without deletions, the 1A(Δ 2) and 1A(Δ 3) groups, and the 1A(Δ 3) viruses with the **HA1 K136E** substitution are antigenically distinct from one another. Following the emergence and spread of viruses in the 1A(Δ 2) group a representative, B/Colorado/06/2017, has been recommended for use in trivalent influenza vaccines for both the 2018–19 northern hemisphere [1] and 2019 southern hemisphere [2] seasons.

Influenza B – Yamagata lineage

Of the B/Yamagata-lineage HA gene sequences in the GISAID EpiFlu database, from viruses with collection dates from 1 June 2018, only three are from EU/EEA countries; one each from France, Norway and Sweden (Figure 4). The HA gene sequences from recently circulating viruses fall in genetic clade 3 (the B/Wisconsin/1/2010–B/Phuket/3073/2013 clade), as has been the case, worldwide, for all HA genes from viruses collected since 1 August 2017. Compared with the vaccine virus, B/Phuket/3073/2013, all recently circulating viruses have fallen in a subgroup defined by **HA1 L172Q** and **M251V** amino acid substitutions. Some subclustering of sequences, defined by specific amino acid substitutions (e.g. **HA1 N217D** or **D229N** or **D232N** [introducing a potential N-linked glycosylation site]), can be seen amongst the most recently circulating viruses characterised (Figure 4). It has been noted in the September 2018 (and earlier) characterisation reports that none of these amino acid substitutions have any obvious antigenic effects based on haemagglutination inhibition (HI) assays using post-infection ferret antisera raised against egg-propagated B/Phuket/3073/2013, which has been recommended for inclusion in quadrivalent vaccines for the 2018–19 [1] northern hemisphere and the 2019 [2] southern hemisphere seasons.

Figure 3. Phylogenetic comparison of influenza B/Victoria-lineage HA genes

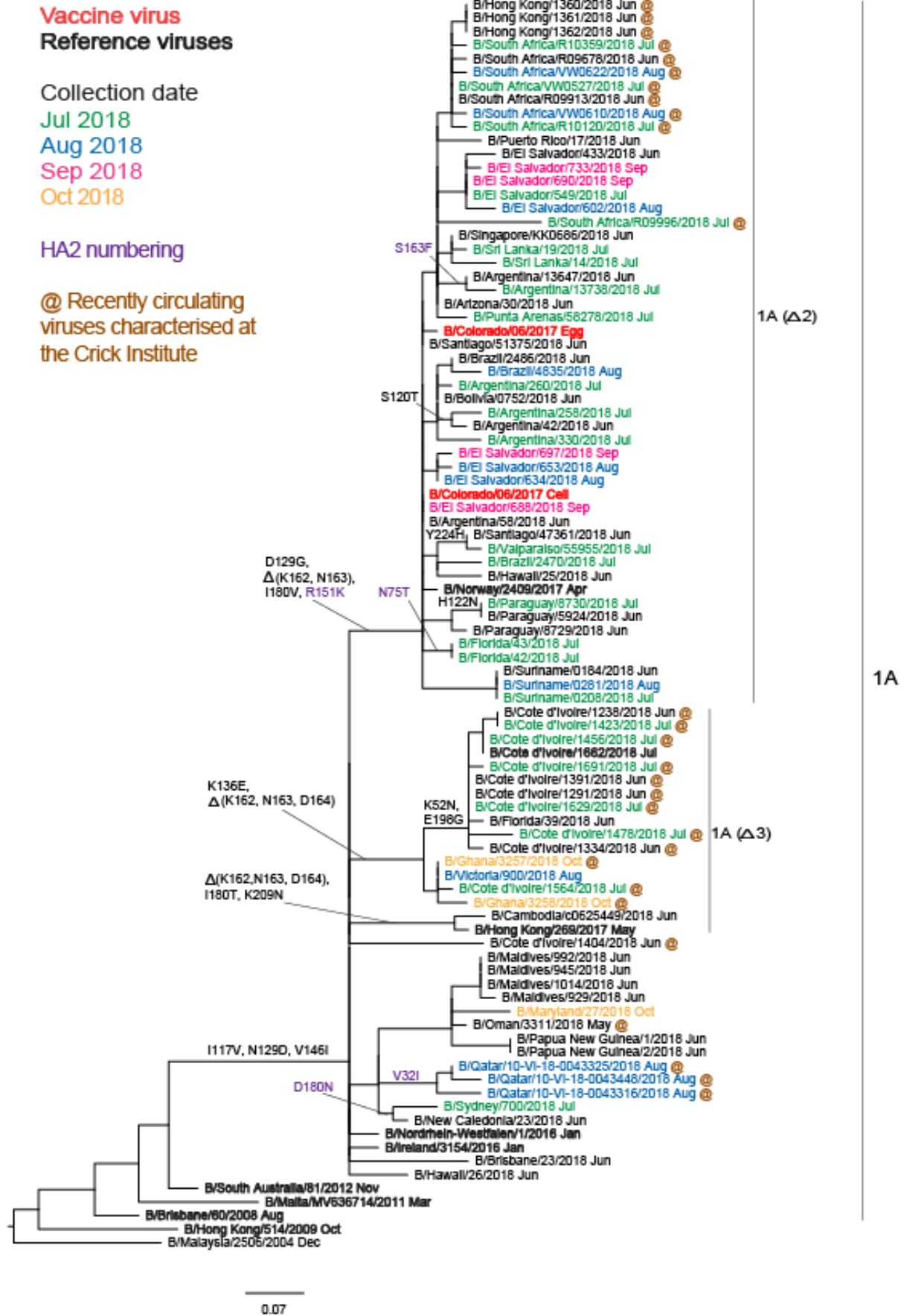
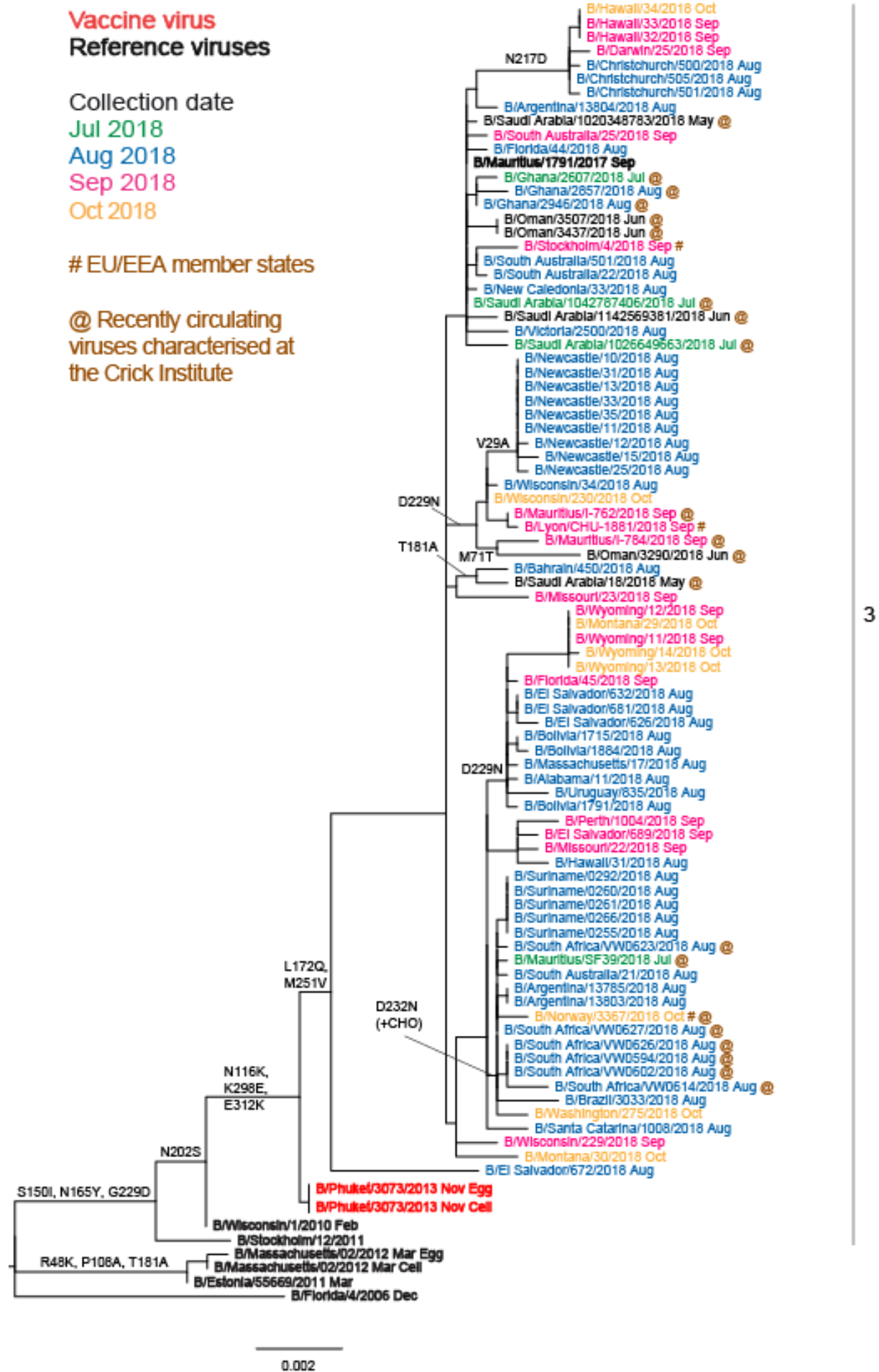


Figure 4. Phylogenetic comparison of influenza B/Yamagata-lineage HA genes



Summaries of data submitted to TESSy

Genetic characterisation

For the 2018–19 season, as of week 48/2018, 65 viruses had been characterised genetically and ascribed to a genetic clade:

- 48 A(H1N1)pdm09 were subclade 6B.1, represented by the vaccine virus A/Michigan/45/2015
- 16 were A(H3N2) viruses, with 15 being subclade 3C.2a1 represented by A/Singapore/INFIMH-16-0019/2016, and one was attributed to a subgroup not listed in current TESSy reporting categories
- One was B/Yamagata-lineage clade 3, represented by the vaccine virus B/Phuket/3073/2013.

Antiviral susceptibility

For the 2018–19 season, as of week 48/2018, 53 A(H1N1)pdm09 and four A(H3N2) viruses have been tested for susceptibility to neuraminidase inhibitors. None showed evidence of reduced susceptibility to the inhibitors.

Influenza A(H7N9) virus

On 1 April 2013, the World Health Organization (WHO) Global Alert and Response [3] reported that the China Health and Family Planning Commission notified the WHO of three cases of human infection with influenza A(H7N9). A description of the characteristics of H7N9 viruses can be found on the WHO website [4]. Increased numbers of cases were reported over the course of the following seasons; cases were reported in 2017, including the fifth (2016–17) and largest wave to date, which included the emergence of highly pathogenic avian influenza (HPAI) strains that have caused some zoonoses, though few human cases were reported during the 2017–18 season [5]. WHO posted an analysis of information on A(H7N9) viruses on 10 February 2017 [6]; a summary and assessment of influenza viruses at the human-animal interface on 1 November 2018 indicates that A(H7N9) avian influenza viruses continue to be detected by agricultural authorities in China but at lower levels than before [7], with the latest human case having occurred early in February 2018 [8]. The latest overview of avian influenza by ECDC in collaboration with the European Food Safety Authority and the EU Reference Laboratory for Avian Influenza was published on 27 September 2018 and can be found on the ECDC website [9].

Influenza A(H5) virus

The most recent monthly risk assessment of influenza at the human–animal interface was published by WHO on 1 November 2018, indicating that various A(H5Nx) subtypes continue to be detected in birds in Africa, Europe and Asia: notably A(H5N6) viruses, though these viruses differ from A(H5N6) viruses that previously infected humans in China; two new cases of A(H5N6) infection were detected, one each in September and October 2018 [7]. By 1 November 2018, no cases of human infection by A(H5N1) viruses had been reported to WHO in 2018 [10]. On 18 November 2016, ECDC published a rapid risk assessment related to outbreaks of highly pathogenic avian influenza H5N8 viruses in Europe [11]. As described above, the EU Reference Laboratory for Avian Influenza, in collaboration with ECDC and the European Food Standards Agency, published the latest overview of avian influenza on 27 September 2018 [9].

WHO CC reports

A description of results generated by the London WHO CC at the WIC and used at WHO vaccine composition meetings held at 1) WHO Geneva, 19–21 February 2018, and 2) CDC Atlanta, 24–26 September 2018, can be found at:

https://www.crick.ac.uk/sites/default/files/2018-07/crick_feb2018_report_for_the_web.pdf [accessed 10 Dec 2018]

and

https://www.crick.ac.uk/sites/default/files/2018-10/September%202018%20interim%20report_opt.pdf [accessed 10 Dec 2018].

Note on the figures

The phylogenetic trees were constructed using [RAxML](#), drawn using [FigTree](#) and annotated using Adobe Illustrator. The bars indicate the proportion of nucleotide changes between sequences. Reference strains are viruses to which post-infection ferret antisera have been raised. The colours indicate the month of sample collection. Isolates from WHO NICs in EU/EEA countries are marked (#). Sequences for most viruses from non-EU/EEA countries were recovered from the GISAID EpiFlu database; those that were generated by WHO CC London are indicated (@). We gratefully acknowledge the authors, originating and submitting laboratories of the sequences from the GISAID EpiFlu database which were downloaded for use in the preparation of this report (all submitters of data may be contacted directly via the [GISAID website](#)), along with all laboratories who submitted sequences directly to WHO CC London.

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