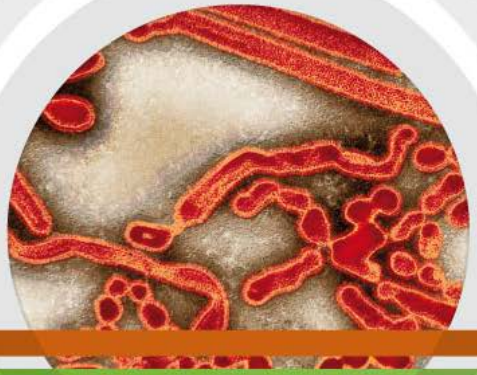


SURVEILLANCE REPORT

Influenza virus characterisation

Summary Europe, November 2017

Summary

This is the first report for the 2017–18 influenza season. As of week 48/2017 nearly 3 000 influenza detections have been reported across the WHO European Region. Co-circulating type A viruses are prevalent over type B, with A(H3N2) less prevalent than A(H1N1)pdm09 viruses and B/Yamagata more prevalent than B/Victoria viruses.

Only two EU/EEA countries have shared influenza positive specimens with the London WHO CC since week 40/2017. Of the 31 specimens received, 23 have collection dates after 31 August 2017 which fall within the time period (1 September 2017 to 31 January 2018) to be considered for the February 2018 WHO Vaccine Consultation Meeting (VCM).

The four A(H1N1)pdm09 viruses characterised antigenically showed good reactivity with antiserum raised against the 2017–18 vaccine virus, A/Michigan/45/2015. While genetic analysis of three viruses is pending, one virus - and others from the European region with collection dates after 31 August 2017 deposited in GISAID - have all fallen in subclade 6B.1, defined by HA1 amino acid substitutions S162N and I216T, many with additional substitutions of S74R, S164T and I295V.

None of the 13 A(H3N2) viruses recovered to date had sufficient HA titre to allow antigenic characterisation by HI assay in the presence of oseltamivir. While genetic analysis of these viruses is pending others - from the European region with collection dates after 31 August 2017 deposited in GISAID - fall within the 3C.2a genetic clade, with a minority falling in the 3C.2a1 genetic subclade.

The two B/Victoria-lineage viruses tested, both from Norway, have collection dates in June 2017 and both were antigenically distinct from tissue culture-propagated surrogates of B/Brisbane/60/2008. Phylogenetic analyses showed both viruses to carry an HA1 double amino acid deletion, falling within a subcluster of genetic clade 1A viruses with recently circulating viruses from Canada, Trinidad and the USA.

Of the five B/Yamagata viruses characterised antigenically, four reacted well with post-infection ferret antiserum raised against egg-propagated B/Phuket/3073/2013, the recommended vaccine virus for use in quadrivalent vaccines for 2017–18 and for trivalent vaccines in the southern hemisphere 2018 season. The two characterised viruses, like others recently circulating in the European region and reported to GISAID, fall within genetic clade 3.

This report was prepared by Rod Daniels, Vicki Gregory, Burcu Ermetal, Aine Rattigan and John McCauley for the European Centre for Disease Prevention and Control (ECDC) under an ECDC framework contract.

Suggested citation: European Centre for Disease Prevention and Control. Influenza virus characterisation, summary Europe, November 2017. Stockholm: ECDC; 2017.

Table 1 shows a summary of influenza virus detections in the WHO European Region reported to TESSy since the start of the 2017–18 season (weeks 40–48/2017). There have been nearly 3 000 detections, with type A viruses prevailing over type B at a ratio of 1.7:1. Of the type A viruses subtyped (n = 796) and the type B viruses ascribed to lineage (n = 129), A(H3N2) had prevailed over A(H1N1)pdm09 and B/Yamagata over B/Victoria by ratios of 3.4:1 and 15.1:1, respectively. While relatively few influenza detections have been reported for weeks 40–48/2017, type A viruses have been predominant over type B by a significantly smaller margin compared to the 2016–17 season (1.7:1 from 6.5:1), with A(H1N1)pdm09 viruses being more prevalent this season, and significant numbers of influenza type B viruses having been detected early in the season, with the dominance of B/Yamagata over B/Victoria increasing from 2.7:1 to 15.1:1.

Since week 40/2017, two shipments of specimens have been received at the Crick Worldwide Influenza Centre (WIC), from two National Influenza Centres in the EU/EEA. These packages contained 31 specimens, a mix of clinical samples and virus isolates, with specimen collection after May 2017 (Table 2). The majority (74%) were type A viruses, and A(H3N2) outnumbered A(H1N1)pdm09 at a ratio of 2.1:1. Of the eight type B specimens received (26% of the specimens), three were B/Victoria-lineage (collected in the course of the 2016–17 season) and five were B/Yamagata-lineage. The antigenic and genetic properties of influenza viruses, characterised since the September 2017 report[1], are presented and discussed in this surveillance report.

Table 1. Influenza virus detections in the WHO European Region from the start of reporting for the 2017–18 season (weeks 40–48/2017)

	Cumulative number of detections			Totals*		Totals for 2016-2017 season*		
Virus type/subtype/lineage	**Sentinel sources**	**Non-sentinel sources**	**Totals**	**%**	**Ratios**	**Number**	**%**	**Ratios**
Influenza A	137	1659	1796	62.8	1.7:1	126 614	86.6	6.5:1
A(H1N1)pdm09	57	124	181	22.7		591	1.1	
A(H3N2)	52	563	615	77.3	3.4:1	53 101	98.9	89.8:1
A not subtyped	28	972	1 000			72 922		
Influenza B	181	884	1 605	37.2		19 570	13.4	
Victoria lineage	2	6	8	6.2		749	27.1	
Yamagata lineage	51	70	121	93.8	15.1:1	2 016	72.9	2.7:1
Lineage not ascribed	128	808	936			16 805		
Total detections (total tested)	318 (7 505)	2 543 (105 478)	2 861 (112 983)			146 184 (686 477)		

** 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.*

[1] European Centre for Disease Prevention and Control. Influenza virus characterisation, summary Europe, September 2017. Stockholm: ECDC; 2017. Available from: https://ecdc.europa.eu/sites/portal/files/documents/ERLI-Net-report-Sep-2017.pdf

Table 2. Summary of clinical samples and virus isolates contained in packages received from EU/EEA Member States since week 40/2017

MONTH*	TOTAL RECEIVED	A		H1N1pdm09		H3N2			B		B Victoria lineage		B Yamagata lineage	
Country		Number received	Number propagated	Number received	Number propagated[1]	Number received	Number propagated[2]		Number received	Number propagated	Number received	Number propagated[1]	Number received	Number propagated[1]
2017 JUNE														
Norway	3										2	2	1	1
2017 JULY														
Norway	1												1	1
2017 AUGUST														
Norway	4			3	2						1	0		
2017 SEPTEMBER														
Norway	2			1	in process								1	1
2017 OCTOBER														
Norway	19			3	2	14	0	14					2	2
2017 NOVEMBER														
Austria	1	1	in process											
Norway	1					1	0	1						
	31	1	0	7	4	15	0	15	0	0	3	2	5	5
		3.2%		22.6%		48.4%			0.0%		9.7%		16.1%	
2 Countries		74.2%							25.8%					

** Month indicates the months in which the clinical specimens were collected*
1. Propagated to sufficient titre to perform HI assay
2. Propagated to sufficient titre to perform HI assay in the presence of 20nM oseltamivir; numbers in red indicate viruses recovered but with insufficient HA titre to permit HI assay.

Influenza A(H1N1)pdm09 virus analyses

Results of haemagglutination inhibition (HI) analyses of viruses performed since the September 2017 report are shown in Table 3. All four A(H1N1)pdm09 viruses from Norway antigenically characterised were similar to the vaccine virus for the present northern hemisphere 2017–18 influenza season, A/Michigan/45/2015 [1], with all viruses being recognised at titres within two-fold of the titre for the homologous virus antiserum. The antiserum raised against A/California/7/2009, the vaccine virus recommended for use for the northern hemisphere 2016–17 influenza season, also recognised all of the test viruses at titres within two-fold of the homologous titre of the antiserum. All four test viruses were recognised by the antiserum panel at titres within four-fold of the antisera titres with their respective homologous viruses, apart from A/Norway/3351/2017, which showed eight-fold reduction with antiserum raised against A/Lviv/N6/2009. Furthermore, over 85% of the individual titres of the test viruses were within two-fold of the titres of the antisera with their homologous viruses.

Genetic analyses of the four test viruses are in process but the HA sequences of A(H1N1)pdm09 viruses from European countries (as defined in GISAID) with collection dates after 31 August 2017 all fall within subclade 6B.1 (Figure 1), as was observed for all EU/EEA A(H1N1)pdm09 viruses characterised throughout the 2016–17 season. The majority of HA genes of recently circulating viruses from EU/EAA countries cluster in a genetic subgroup defined by HA1 amino acid substitutions of S74R, S164T and I295V.

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										
		Passage history			A/Mich 45/15 Egg	A/Cal 7/09 Egg	A/Bayern 69/09 MDCK	A/Lviv N6/09 MDCK	A/Astrak 1/11 MDCK	A/St. P 27/11 Egg	A/St. P 100/11 Egg	A/HK 5659/12 MDCK	A/Sth Afr 3626/13 Egg	A/Slov 2903/2015 Egg	A/Israel Q-504/15 MDCK
		Ferret number			NIB F42/16[*1]	F06/16[*1]	F09/15[*1]	F14/13[*1]	F22/13[*1]	F26/14[*1]	F24/11[*1]	F30/12[*1]	F03/14[*1]	F02/16[*2]	F08/16[*2]
		Genetic group			6B.1				5	6	7	6A	6B	6B.1	6B.2
REFERENCE VIRUSES															
A/Michigan/45/2015		6B.1	2015-09-07	E3/E3	640	640	320	320	640	640	1280	640	640	1280	640
A/California/7/2009	clone 38-32		2009-04-09	E3/E3	640	640	640	640	1280	640	2560	1280	1280	2560	1280
A/Bayern/69/2009	G155E		2009-07-01	MDCK5/MDCK1	<	<	160	160	40	<	40	<	40	40	<
A/Lviv/N6/2009	G155E, D222G		2009-10-27	MDCK4/SIAT1/MDCK3	40	40	640	640	80	80	80	80	80	160	40
A/Astrakhan/1/2011		5	2011-02-28	MDCK1/MDCK5	320	320	320	160	640	320	1280	320	320	640	320
A/St. Petersburg/27/2011		6	2011-02-14	E1/E4	640	640	320	320	640	640	1280	320	640	1280	640
A/St. Petersburg/100/2011		7	2011-03-14	E1/E4	320	320	160	160	320	160	1280	320	320	640	320
A/Hong Kong/5659/2012		6A	2012-05-21	MDCK4/MDCK2	160	320	160	80	320	160	640	320	320	640	320
A/South Africa/3626/2013		6B	2013-06-06	E1/E3	640	320	640	640	640	640	1280	640	1280	1280	640
A/Slovenia/2903/2015	clone 37	6B.1	2015-10-26	E4/E2	320	640	320	160	640	320	1280	640	640	1280	640
A/Israel/Q-504/2015		6B.2	2015-12-15	C1/MDCK2	320	320	320	160	640	320	1280	640	640	1280	640
TEST VIRUSES															
A/Norway/3113/2017		6B.1	2017-08-08	MDCKx/MDCK1	640	640	80	320	640	640	2560	640	640	2560	1280
A/Norway/3133/2017			2017-08-21	MDCK1	1280	640	160	320	640	320	1280	640	640	1280	1280
A/Norway/3333/2017			2017-10-20	MDCK1	640	640	160	160	320	160	1280	640	640	1280	1280
A/Norway/3351/2017			2017-10-24	MDCK1	320	320	160	80	320	160	1280	320	320	640	640

Vaccine

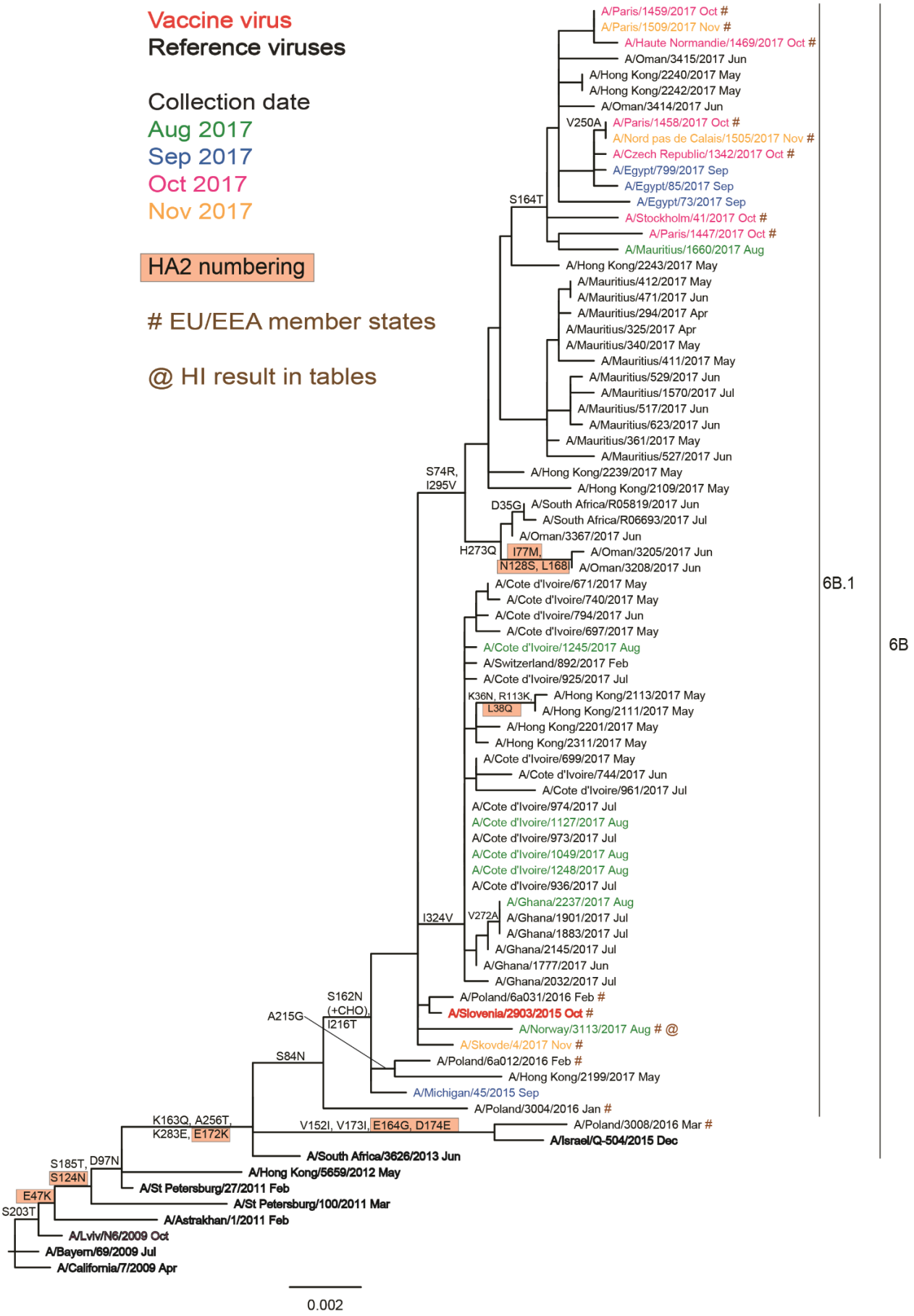
* Superscripts refer to antiserum properties (< relates to the lowest dilution of antiserum used)

1 < = <40; 2 < = <80

[$] Virus clone indicated and significant HA1 amino acid substitutions

Sequences in phylogenetic trees

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



Influenza A(H3N2) virus analyses

As described in many previous reports[2] influenza A(H3N2) viruses have continued to be difficult to characterise antigenically by HI assay due to variable agglutination of red blood cells (RBCs) from guinea pigs, turkeys and humans, often with the loss of ability to agglutinate any of these RBCs. This problem was first highlighted in the November 2014 report[3] and is particularly relevant for most viruses that fall in genetic subclade 3C.2a.

A number of the 15 A(H3N2) virus specimens received so far for the 2017–18 season are in the process of virus isolation and genetic analysis. However, of those successfully isolated to date as shown by positive neuraminidase activity, none could be analysed by HI due to insufficient HA activity in the presence of 20nM oseltamivir.

Phylogenetic analysis of the HA genes of representative A(H3N2) viruses from Europe with recent collection dates after 31 August 2017, as available in GISAID, is shown in Figure 2. Viruses in subclades 3C.2a and 3C.3a have been in circulation since the 2013–14 northern hemisphere influenza season, with subclade 3C.2a viruses predominant since the 2014–15 influenza season and continuing to be predominant in recent months (Figure 2). Clusters of viruses have emerged in both subclades and one of these clusters has been designated 3C.2a1. Amino acid substitutions that define these subdivisions and subclades are:

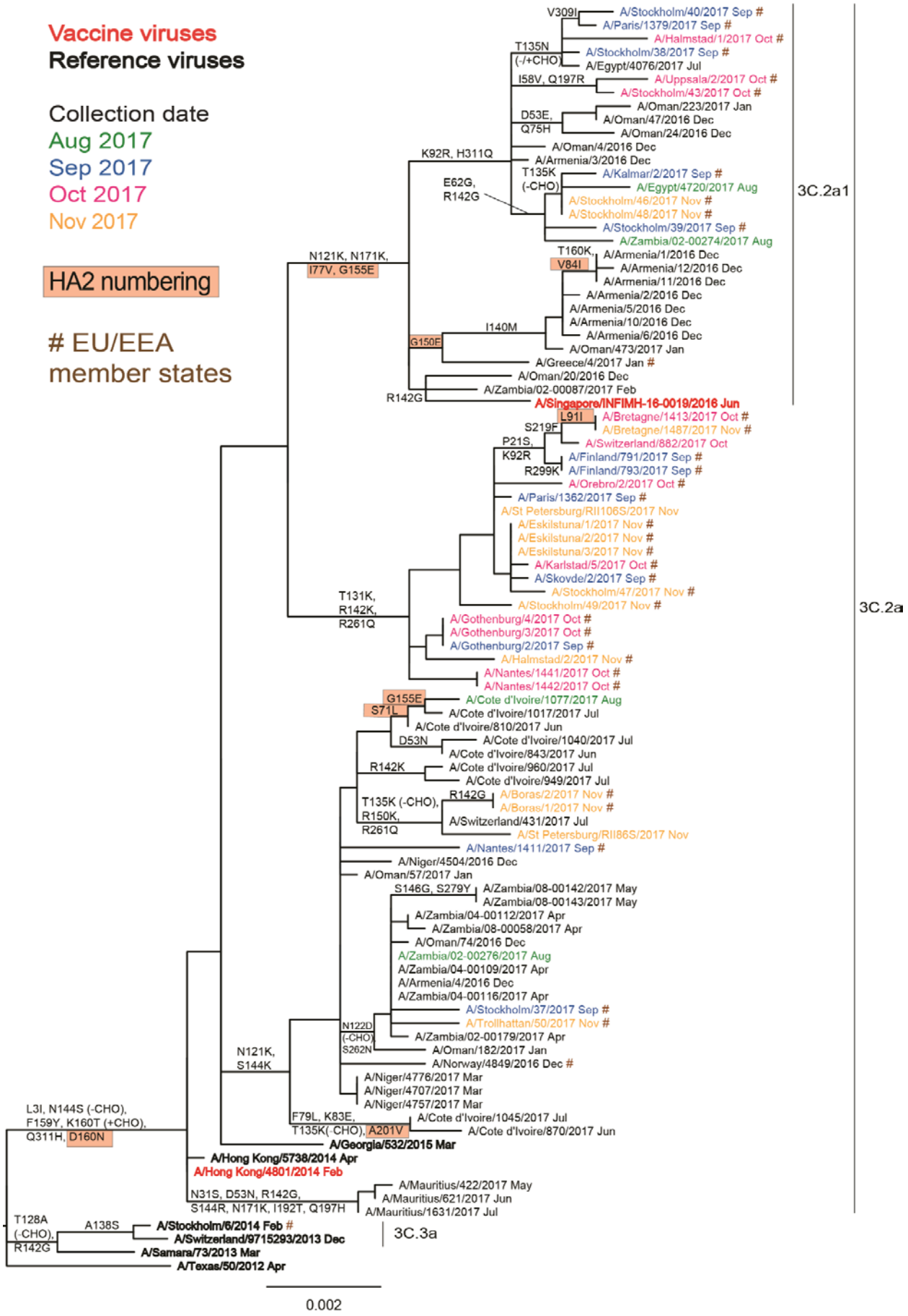
- 3C.2a: N145S in HA1, and D160N in HA2, which defined clade 3C.2, plus L3I, N144S (resulting in the loss of a potential glycosylation site), F159Y, K160T (in the majority of viruses, resulting in the gain of a potential glycosylation site), N225D and Q311H in HA1 - e.g. A/Hong Kong/4801/2014;
- 3C.2a1: those in 3C.2a, plus N171K in HA1 and I77V and G155E in HA2, e.g. A/Bolzano/7/2016 and A/Iasi/206625/2017, often with N121K in HA1 - e.g. A/Scotland/63440583/2016 and A/Bulgaria/471/2017;
- 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/Switzerland/9715293/2013.

Currently circulating viruses fall into genetic groups within both subclades 3C.2a and 3C.2a1, with the majority of recently circulating viruses in EU/EEA countries falling in subclade 3C.2a. The location of A/Singapore/INFIMH-16-0019/2016 (3C.2a1), the A(H3N2) virus recommended for inclusion in vaccines for the southern hemisphere 2018 season [2], is indicated in Figure 2.

[2] For example, the September 2013 report: European Centre for Disease Prevention and Control. Influenza virus characterisation, summary Europe, September 2013. Stockholm: ECDC; 2013. Available from: https://ecdc.europa.eu/sites/portal/files/media/en/publications/Publications/influenza-virus-characterisation-sep-2013.pdf

[3] European Centre for Disease Prevention and Control. Influenza virus characterisation, summary Europe, November 2014. Stockholm: ECDC; 2014. Available from: http://www.ecdc.europa.eu/en/publications/Publications/ERLI-Net report November 2014.pdf

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



Influenza B virus analyses

Norway have provided eight influenza type B-positive specimens with collection dates after May 2017: three B/Victoria-lineage and five B/Yamagata-lineage (Table 2).

Influenza B – Victoria lineage

The two Norwegian viruses recovered had collection dates in June 2017 and both were clade 1A viruses carrying the double amino acid deletion in HA1, Δ162-163. HI results are shown in Table 4. Both test viruses showed poor reactivity with all antisera generated against B/Victoria lineage viruses that did not carry the double amino acid deletion, but good reactivity (titres within two-fold of the homologous titre) with antiserum raised against the Δ162-163 reference virus cell culture-propagated B/Norway/2409/2017.

Few (11) HA gene sequences of B/Victoria lineage viruses with collection dates after 31 August 2017 have been deposited in GISAID, none of which was from EU/EEA countries. These recently circulating viruses, like those earlier viruses from Europe and elsewhere, continue to have HA genes that fall in the B/Brisbane/60/2008 clade (clade 1A; Figure 3). The great majority of viruses, with collection dates since October 2015, fall in a major subcluster defined by amino acid substitutions **I117V, N129D** and **V146I** within clade 1A. Two new groups have emerged with deletions in the HA gene. For one group the HA gene encodes an HA with deletion of residues 162 and 163 of HA1 (exemplified by B/Norway/2409/2017; △162-163). Recently circulating examples of this gene have been detected in Canada, Trinidad and the USA. Meanwhile, the other group encodes an HA with deletion of residues 162, 163 and 164 of HA1 (exemplified by B/Hong Kong/269/2017; △162-164). The △162-163 viruses have additional substitutions **D129G, I180V** in **HA1** and **R151K** in **HA2** and the △162-164 viruses from Hong Kong have additional substitutions **I180T** and **K209N** in **HA1**.

Influenza B – Yamagata lineage

HI results for five B/Yamagata-lineage test viruses analysed since the September 2017 report are shown in Table 5. The two viruses analysed genetically to date belong to genetic clade 3, the B/Wisconsin/1/2010-B/Phuket/3073/2013 clade.

The antiserum raised against egg-propagated B/Phuket/3073/2013, recommended recently for inclusion in trivalent vaccines for the southern hemisphere 2018 season [2], recognised all five test viruses at titres within four-fold of the antiserum titre with the homologous virus and four within two-fold. An antiserum raised against the cell culture-propagated cultivar of B/Phuket/3073/2013 similarly recognised three of five test viruses at titres within four-fold of the homologous titre of the antiserum. An antiserum raised against a former vaccine virus, egg-propagated B/Wisconsin/1/2010 with a homologous titre of 160, recognised all of the test viruses at titres within four-fold of the homologous titre of the antiserum, and four within two-fold of the homologous titre. The antiserum raised against egg-propagated B/Stockholm/12/2011 also recognised all of the test viruses at titres within four-fold of the homologous titre of the antiserum, but only one within two-fold, and the antiserum raised against egg-propagated B/Hong Kong/3417/2014 recognised all five viruses at titres within two-fold of the homologous titre of the antiserum.

Antisera raised against both egg- and cell-propagated clade 2 viruses, recognised none of the test viruses well, all being recognised at titres reduced at least eight-fold compared to the respective homologous titres of the antisera.

Figure 4 shows a phylogenetic analysis of the HA genes of representative B/Yamagata-lineage viruses. Worldwide, the vast majority of HA genes from viruses collected in 2017 have fallen in clade 3, the B/Wisconsin/1/2010–B/Phuket/3073/2013 clade. The vast majority of viruses, including those with collection dates after 31 August from Europe as deposited in GISAID, fall in a subgroup defined by **HA1 L172Q** and **M251V** amino acid substitutions.

Table 4. Antigenic analysis of influenza B/Victoria-lineage viruses by HI

Viruses	Other information	Passage history	Collection date	Passage history	Haemagglutination inhibition titre: Post-infection ferret antisera										
					B/Bris 60/08 Egg	B/Mal 2506/04 Egg	B/Bris 60/08 Egg	B/Malta 636714/11 Egg	B/Jhb 3964/12 Egg	B/For V2367/12 MDCK	B/Sth Aus 81/12 Egg	B/HK 514/09 MDCK	B/Ireland 3154/16 MDCK	B/Nord-West 1/16 MDCK	B/Nor 2409/17 MDCK
		Ferret number			Sh 539, 540, 543, 544, 570, 571, 574[*1,3]	F41/14[*2]	NIB F52/16[*2]	F29/13[*2]	F04/16[*4]	F09/16[*2]	F41/13[*2]	F09/13[*2]	F15/16[*2]	F16/16[*2]	F26/17[*2]
		Genetic group			1A		1A	1A	1A	1A	1A	1B	1A	1A	1A(Δ2)
REFERENCE VIRUSES															
B/Malaysia/2506/2004			2004-12-06	E3/E6	2560	320	160	80	40	80	160	10	<	<	20
B/Brisbane/60/2008		1A	2008-08-04	E4/E4	2560	160	640	320	160	320	640	80	40	40	20
B/Malta/636714/2011		1A	2011-03-07	E4/E1	1280	80	640	320	160	160	320	40	40	40	10
B/Johannesburg/3964/2012		1A	2012-08-03	E1/E2	5120	640	1280	1280	1280	1280	1280	320	320	320	80
B/Formosa/V2367/2012		1A	2012-08-06	MDCK1/MDCK3	5120	40	640	320	160	320	640	80	80	80	20
B/South Australia/81/2012		1A	2012-11-28	E4/E2	2560	160	640	320	160	320	640	40	40	40	20
B/Hong Kong/514/2009		1B	2009-10-11	MDCK1/MDCK2	2560	10	40	40	40	320	80	80	80	160	20
B/Ireland/3154/2016		1A	2016-01-14	MDCK1/MDCK4	2560	<	40	20	40		80	80	80	160	20
B/Nordrhein-Westfalen/1/2016		1A	2016-01-04	C2/MDCK2	1280	<	40	20	40	160	40	80	80	80	20
B/Norway/2409/2017		1A(Δ2)	2017-04-27	MDCK1/MDCK2	80	<	<	<	<	10	<	<	<	<	320
TEST VIRUSES															
B/Norway/2957/2017		1A(Δ2)	2017-06-07	MDCK1	80	<	<	<	<	<	<	<	<	<	320
B/Norway/2977/2017		1A(Δ2)	2017-06-21	MDCK1	80	<	<	<	<	<	<	<	<	<	160
							Vaccine								

* Superscripts refer to antiserum properties (< relates to the lowest dilution of antiserum used):
[1] < = <40; [2] < = <10; [3] hyperimmune sheep serum; [4] < = <20

Sequences in phylogenetic trees

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

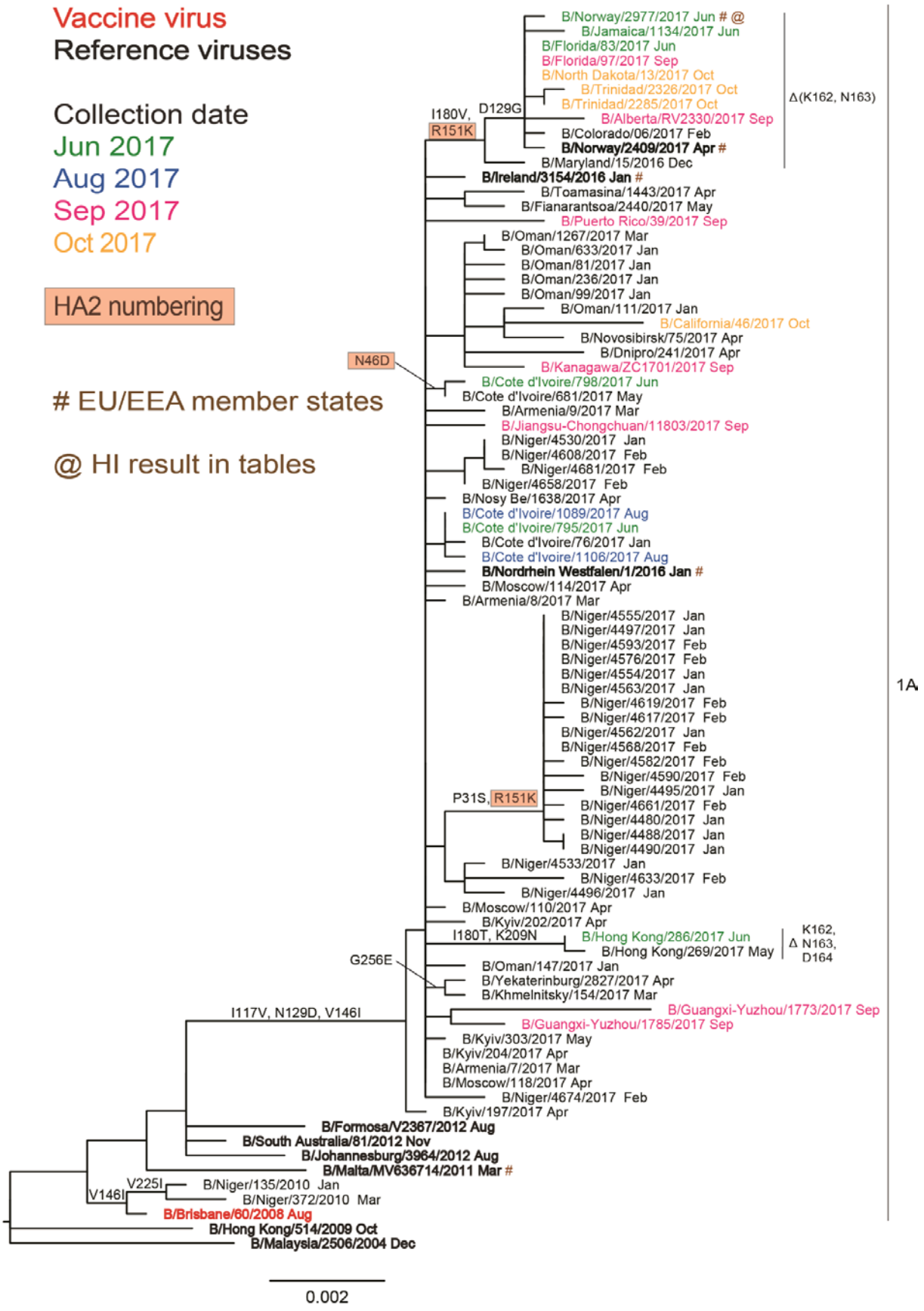


Table 5. Antigenic analysis of influenza B/Yamagata-lineage viruses by HI

Viruses	Other information	Passage history / Ferret number / Genetic Group	Collection date	Passage history	Haemagglutination inhibition titre										
						Post-infection ferret antisera									
					B/Phuket 3073/13	B/Fl 4/06	B/Bris 3/07	B/Estonia 55669/11	B/Mass 02/12	B/Mass 02/12	B/Wis 1/10	B/Stock 12/11	B/Phuket 3073/13	B/Phuket 3073/13	B/HK 3417/14
		Passage history			Egg	Egg	Egg	MDCK	MDCK	Egg	Egg	Egg	MDCK	Egg	Egg
		Ferret number			SH614[*1,3]	F17/13[*1]	F38/14[*2]	F27/13[*2]	F05/15[*2]	F16/14[*1]	F36/15[*2]	F06/15[*2]	F27/15[*4]	NIB F51/16*[2]	St Judes F715/14[*2,4]
		Genetic Group			3	1	2	2	2	2	3	3	3	3	3
REFERENCE VIRUSES															
B/Florida/4/2006		1	2006-12-15	E7/E1	1280	640	640	80	80	1280	160	160	40	640	160
B/Brisbane/3/2007		2	2007-09-03	E2/E2	2560	1280	1280	160	160	1280	160	320	40	1280	320
B/Estonia/55669/2011		2	2011-03-14	MDCK2/MDCK3	1280	40	40	320	80	80	40	20	40	40	80
B/Massachusetts/02/2012		2	2012-03-13	MDCK1/C2/MDCK4	5120	320	320	640	640	640	320	160	160	640	320
B/Massachusetts/02/2012		2	2012-03-13	E3/E4	1280	640	640	80	80	1280	160	160	20	640	160
B/Wisconsin/1/2010		3	2010-02-20	E3/E2	2560	160	160	20	10	320	160	80	40	640	160
B/Stockholm/12/2011		3	2011-03-28	EX/E2	2560	160	80	20	<	160	80	160	40	320	80
B/Phuket/3073/2013		3	2013-11-21	MDCK2/MDCK2	5120	160	160	160	160	320	320	160	320	640	160
B/Phuket/3073/2013		3	2013-11-21	E4/E3	1280	80	80	10	<	160	80	80	20	320	80
B/Hong Kong/3417/2014		3	2014-06-04	E4/E3	1280	80	40	10	<	80	80	40	20	160	160
TEST VIRUSES															
B/Norway/2924/2017			2017-06-08	MDCK1	2560	80	40	40	20	80	80	40	40	160	80
B/Norway/3098/2017			2017-07-28	MDCK1/MDCK1	2560	80	40	40	40	80	80	80	80	160	80
B/Norway/3157/2017		3	2017-09-01	MDCK1	2560	40	40	40	20	80	40	40	40	80	80
B/Norway/3244/2017		3	2017-10-02	MDCK1	2560	80	40	20	40	80	80	40	80	160	160
B/Norway/3387/2017			2017-10-30	MDCK1	2560	80	40	40	40	80	80	40	80	320	160

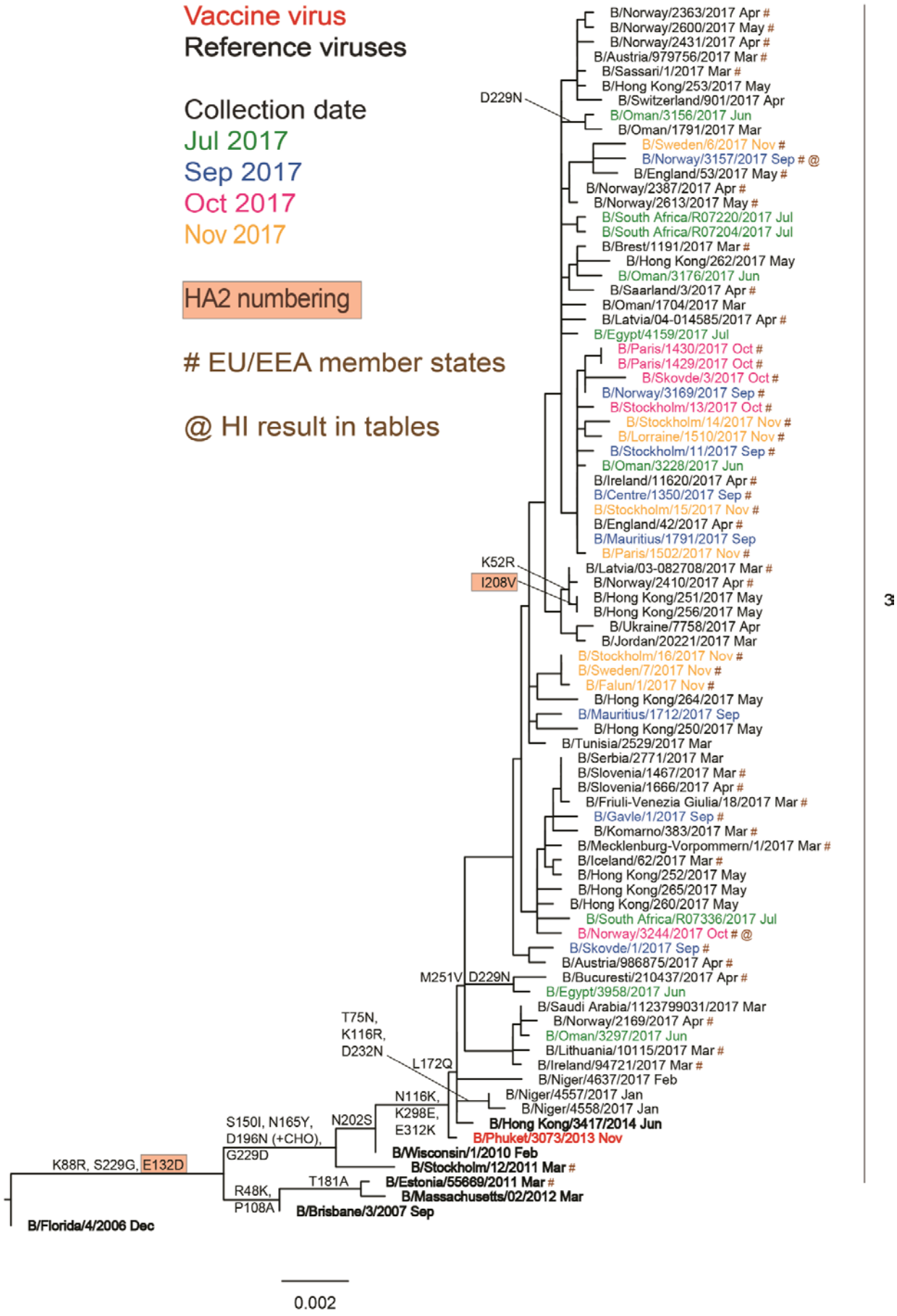
* Superscripts refer to antiserum properties (< relates to the lowest dilution of antiserum used):
1 < = <40; 2 < = <10; 3 hyperimmune sheep serum; 4 RDE serum pre-adsorbed with TRBC
ND = Not Done

Vaccine[#]

[#] B/Yamagata-lineage virus recommended for use in quadravalent vaccines

Sequences in phylogenetic trees

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



Summary of genetic data submitted to TESSy

For the 2017–18 season, weeks 40–48/2017, 122 viruses have been characterised genetically:

- A total of 16 were defined as A(H1N1)pdm09 subclade 6B.1 as represented by A/Michigan/45/2015;
- In all, 45 were A(H3N2) subclade 3C.2a represented by A/Hong Kong/4801/2014 and 29 were subclade 3C.2a1 represented by A/Singapore/INFIMH-16-0019/2016;
- Three were B/Victoria-lineage clade 1A represented by B/Brisbane/60/2008;
- A total of 26 were B/Yamagata-lineage clade 3 represented by B/Phuket/3073/2013, with three that were not attributed to a clade.

Antiviral susceptibility

Phenotypic testing for susceptibility to oseltamivir and zanamivir has been conducted on 24 viruses from Norway contained in the package received after week 40/2017 at the WIC: 4 A(H1N1)pdm09, 13 A(H3N2), 2 B/Victoria-lineage and 5 B/Yamagata-lineage viruses. All showed normal inhibition by the two antivirals.

For weeks 40–48/2017 of the 2017–18 influenza season, countries reported on the antiviral susceptibility of 11 A(H1N1)pdm09 viruses, 33 A(H3N2) viruses and 11 influenza type B viruses from sentinel and non-sentinel sources to TESSy. All but one showed no molecular or phenotypic evidence of reduced inhibition (RI) by neuraminidase inhibitors (oseltamivir and zanamivir); an A(H3N2) isolate showed RI by both oseltamivir and zanamivir.

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 WHO of three cases of human infection with influenza A(H7N9). A description of the characteristics of H7N9 viruses can be found on WHO's website [4]. Increased numbers of cases were reported over the course of the following seasons and cases have also been reported in 2017, during the fifth and largest wave to date. This wave has included the emergence of Highly Pathogenic Avian Influenza (HPAI) strains that have caused human cases [5]. A revised Rapid Risk Assessment [6] for these A(H7N9) viruses was published by ECDC on 11 February 2015 and most recently updated on 3 July 2017 [7] and on 16 October 2017 in a joint EFSA/ECDC report [8]. WHO posted an analysis of recent information on A(H7N9) viruses on 10 February 2017 [9] and a summary and assessment of influenza viruses at the human-animal interface on 30 October 2017 [10], with the latest cases being reported on 26 October 2017 [5].

Influenza A(H5) virus

The most recent monthly risk assessment of influenza at the human-animal interface was published by WHO on 30 October 2017 [9]. ECDC published an updated rapid risk assessment on the situation in Egypt on 13 March 2015 [11] and an epidemiological update on 10 April 2015 and 16 October 2017 [12]. On 18 November 2016, ECDC published a rapid risk assessment related to outbreaks of highly pathogenic avian influenza A(H5N8) viruses in Europe and on 16 October 2017 a joint EFSA/ECDC report [8,13].

WHO Collaborating Centre reports

A description of results generated by the WHO Collaborating Centre for Reference and Research on Influenza at the Crick Worldwide Influenza Centre (Francis Crick Institute) and used at the WHO vaccine composition meetings held at WHO Geneva 27 February–1 March 2017 and The Peter Doherty Institute, University of Melbourne 25–27 September 2017 can be found at:

https://www.crick.ac.uk/media/358671/crick_nh_vcm_report_feb_2017_v2.pdf
and
https://www.crick.ac.uk/media/393884/crick_sh2017_vcm_report_to_post.pdf

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 National Influenza Centres in EU/EEA countries are marked (#). Sequences for some viruses from non-EU/EEA countries were recovered from GISAID. We gratefully acknowledge the authors, the originating and submitting laboratories of the sequences from GISAID's 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), and all the laboratories that submitted sequences directly to the London WHO Collaborating Centre.

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