Recommended composition of influenza virus vaccines for use in the 2020 southern hemisphere influenza season

September 2019*

WHO convenes technical consultations[1] in February and September each year to recommend viruses for inclusion in influenza vaccines[2] for the northern and southern hemisphere influenza seasons, respectively. This recommendation relates to the influenza vaccines for use in the forthcoming southern hemisphere 2020 influenza season. A recommendation will be made in February 2020 relating to vaccines that will be used for the northern hemisphere 2020-2021 influenza season. For countries in tropical and subtropical regions, WHO guidance for choosing between the northern and southern hemisphere formulations is available on the WHO Global Influenza Programme website[3].

Seasonal influenza activity

Between February and September 2019, influenza activity was reported globally, with influenza A(H1N1)pdm09, A(H3N2) and both lineages of influenza B viruses co-circulating.

In the temperate zone of the northern hemisphere, influenza activity declined from February to April and remains at inter-seasonal levels in most countries. Influenza A(H1N1)pdm09 was the predominant type A virus in most countries in northern and eastern Europe, northern Africa and Asia. Influenza A(H3N2) was the predominant type A virus in south-western Europe, North America and some countries in Asia. Influenza B viruses, of both B/Victoria/2/87 and B/Yamagata/16/88 lineages, circulated in considerably lower proportions than influenza A viruses in most countries. In China influenza A(H1N1)pdm09 viruses predominated followed by influenza B and A(H3N2) viruses.

Influenza activity in the tropical and subtropical countries of Africa was generally low with predominance of influenza A(H3N2) viruses followed by influenza B (mostly B/Victoria lineage) and A(H1N1)pdm09 viruses. In tropical countries of Asia, influenza A(H1N1)pdm09 viruses predominated followed by influenza B (mostly B/Victoria lineage) and influenza A(H3N2) viruses. In tropical countries of South America, the Caribbean and Central America, influenza A and influenza B viruses co-circulated. Influenza A(H1N1)pdm09 viruses circulated in higher proportions than A(H3N2) and influenza B/Yamagata lineage viruses circulated in higher proportion than B/Victoria lineage viruses.

In the temperate zone of the southern hemisphere, influenza activity increased from May to July. In the southern cone of South America, influenza A(H1N1)pdm09 viruses was predominant followed by A(H3N2) and influenza B with co-circulation of both lineage viruses. In South Africa, influenza A(H3N2) was predominant with very few detections of A(H1N1)pdm09 and influenza B viruses. Influenza activity was moderate to high in Australia with mainly influenza A(H3N2) and influenza B (mostly B/Victoria/87 lineage). Overall, influenza activity in the southern hemisphere declined since August, though there has been continued circulation of influenza B viruses in Chile.

Influenza A

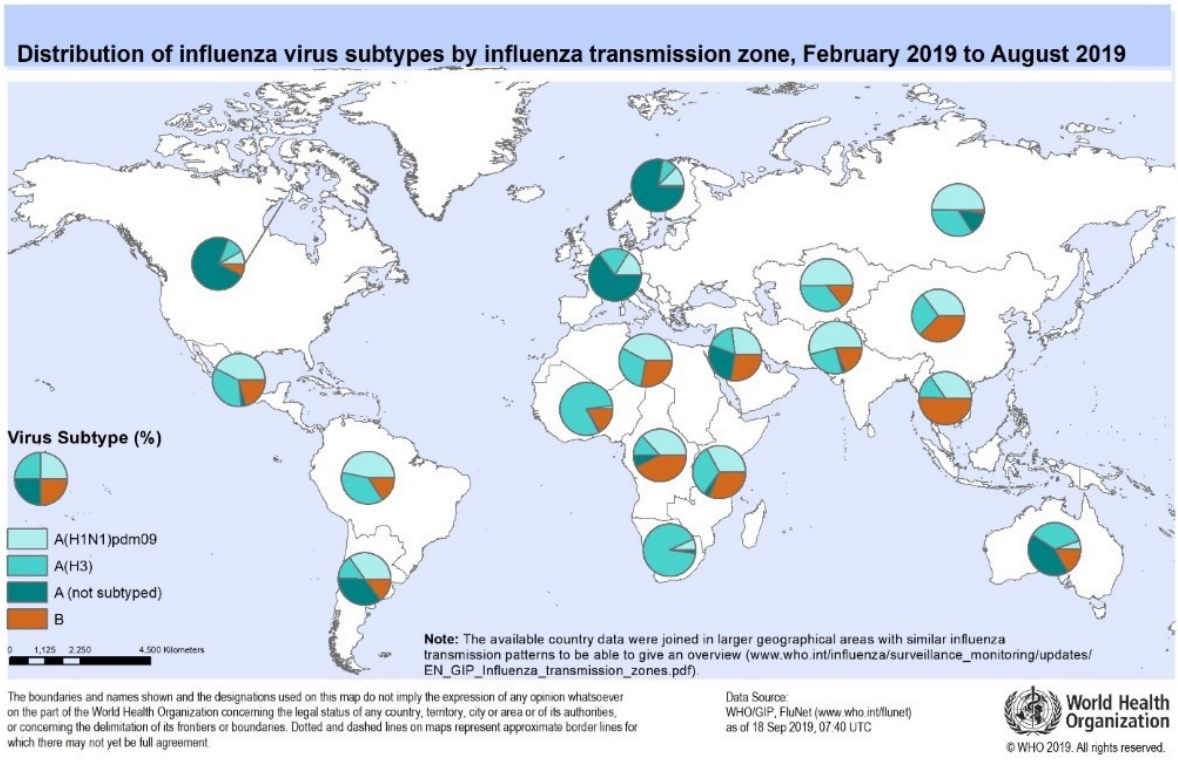
Influenza A viruses were predominant in most countries during this period, including countries in Africa, North America, central America, temperate and tropical South America, Europe, Asia and Oceania. Globally, co-circulation of both A(H1N1)pdm09 and A(H3N2) viruses was evident in all countries, areas and territories. Influenza A(H1N1)pdm09 viruses circulated in higher proportions in

[1] http://www.who.int/influenza/vaccines/virus/en/
[2] Description of the process of influenza vaccine virus selection and development available at: http://www.who.int/gb/pip/pdf_files/Fluvaccvirusselection.pdf
[3] Influenza in the tropics and sub-tropics: http://www.who.int/influenza/vaccines/tropics/en/

most countries in northern, eastern and central Africa, central, south-eastern and western Asia, northern Europe and central America. Influenza A(H3N2) viruses circulated in higher proportions in some countries in Oceania, southern and western Africa, tropical regions of South America including the Caribbean, North America and south-western Europe.

Influenza B

Influenza B viruses circulated at much lower frequency than influenza A viruses in most countries in Europe, southern and western Africa and the Americas. By contrast, influenza B circulated in higher proportions than influenza A in most countries in south east Asia, and there was increased influenza B activity in some countries in the southern cone of South America (especially Chile) from mid-August. Although both influenza B/Victoria and B/Yamagata lineage viruses co-circulated, B/Victoria lineage viruses predominated in most regions of the world. The exception was South America where there was higher circulation of B/Yamagata lineage viruses.



Detailed information by country of the extent and type of seasonal influenza activity worldwide is available on the WHO website: http://www.who.int/influenza/resources/charts/en/.

Zoonotic influenza infections caused by A(H5), A(H7N9), A(H9N2) and A(H1N1)v viruses

From 18 February 2019 to 24 September 2019, one human case of highly pathogenic avian influenza A(H5N6) virus infection was reported by China and one human case of A(H5N1) virus infection was reported by Nepal. Related viruses were present in poultry in these countries. Since December 2003, a total of 885 human cases of avian influenza A(H5) virus infection with 463 deaths have been confirmed in 17 countries. To date there has been no evidence of sustained human-to-human transmission.

During this period, one human case of highly pathogenic avian influenza A(H7N9) virus infection was reported by China. Since March 2013, a total of 1568 cases of A(H7N9) virus infection with 616 deaths have been reported. Both China and Oman each reported a single human case of avian influenza

A(H9N2) virus infection during this period. Additionally, one case of A(H1N1)v virus infection was reported by the United States.

Antigenic and genetic characteristics of recent seasonal influenza viruses, serology and antiviral susceptibility

Influenza A(H1N1)pdm09 viruses

Nearly all A(H1N1)pdm09 viruses had haemagglutinin (HA) gene sequences that belonged to phylogenetic clade 6B.1, represented by A/Michigan/45/2015. The vast majority fell into subclade 6B.1A, with the HA1 amino acid substitutions of S74R, S164T, S183P and I295V as seen in A/Brisbane/02/2018. Several genetic subgroups within 6B.1A circulated, with the majority of viruses sharing amino acid substitutions N129D and T185I. Although almost all recent A(H1N1)pdm09 viruses were well recognised in haemagglutination inhibition (HI) assays by post-infection ferret antisera raised against the 2019 southern hemisphere vaccine virus, egg-propagated A/Michigan/45/2015 and its cell culture-propagated equivalent, it was previously shown that many were recognised less well by post-vaccination human antisera. In HI assays with post-infection ferret antisera circulating viruses were antigenically like the egg-propagated A/Brisbane/02/2018 and cell culture-propagated A/Idaho/07/2018 vaccine viruses of the 2019-2020 northern hemisphere vaccines, except for a small number of viruses with HA1 amino acid substitutions at residues 155 or 156.

Human serology studies used serum panels from children, adults and elderly adults who had received either trivalent or quadrivalent inactivated vaccines with the composition used in the northern hemisphere 2018-2019 or southern hemisphere 2019 seasons. The 2018-2019 northern hemisphere vaccine contained A/Michigan/45/2015 (H1N1)pdm09-like, A/Singapore/INFIMH-16-0019/2016 (H3N2)-like, B/Colorado/06/2017-like viruses in trivalent vaccines, with B/Phuket/3073/2013-like virus included in quadrivalent vaccines. Southern hemisphere serum panels were taken from people who received vaccine containing A/Michigan/45/2015 (H1N1)pdm09-like, A/Switzerland/8060/2017 (H3N2)-like, and B/Phuket/3073/2013-like viruses in trivalent vaccines, and B/Colorado/06/2017-like viruses in quadrivalent vaccines. Geometric mean HI titres against many recent representative cell culture-propagated A(H1N1)pdm09 viruses with the HA1 amino acid substitution of S183P were reduced compared to HI titres to the cell culture-propagated reference virus A/Michigan/45/2015; reductions were more pronounced when measured against the egg-propagated vaccine virus.

Of 4626 influenza A(H1N1)pdm09 viruses tested for neuraminidase inhibitor (NAI) susceptibility, 33 showed reductions in susceptibility to one or more of the inhibitors. Twenty-six viruses from seven countries carried an H275Y amino acid substitution in the neuraminidase (NA), which conferred highly reduced inhibition by oseltamivir and peramivir. Seven viruses carried other amino acid substitutions in the NA, which conferred reduced inhibition to at least one NAI. One thousand two hundred and twenty-eight viruses were tested for susceptibility to the endonuclease inhibitor baloxavir. Two viruses from patients treated with baloxavir in Japan were genotypically and phenotypically confirmed to have reduced susceptibility to this inhibitor.

Influenza A(H3N2) viruses

Phylogenetic analysis of A(H3N2) viruses collected from February to August 2019 showed that the majority of HA genes belonged to clade 3C.2a. However, there was regional heterogeneity, with clade 3C.3a viruses predominating in several countries. There was a significant increase in the proportion of viruses within the 3C.2a1b subclade, along with further genetic diversification of the HA and NA genes. Over this period there has been a continuing decrease in detection of 3C.2a2 subclade viruses. The majority of HA genes of recent viruses belonged to genetic subclade 3C.2a1b, with viruses having either T128A and T135K or T131K substitutions. Most recent A(H3N2) viruses sequenced belonged to the 3C.2a1b+131K subgroup, but with the additional substitutions of Q197R and S219F in

HA1 and V18M (V347M) and E155G in HA2 (E484G). Within the other major subgroup 3C.2a1b+128A+135K, viruses with additional substitutions at S137F, A138S and F193S in HA have been detected in increasing proportions in Bangladesh, China and Hong Kong Special Administrative Region of China, and in smaller numbers, in other parts of the world.

Antigenic characterisation of clade 3C.2a viruses continued to be technically difficult because a large proportion of viruses did not agglutinate red blood cells, preventing HI analysis of such viruses. Virus neutralisation (VN) assays have become the preferred method for determining the antigenic characteristics of current A(H3N2) viruses.

In VN or HI assays, ferret antisera raised against cell culture-propagated A/Switzerland/8060/2017 (3C.2a2) virus inhibited the few viruses found in this subclade well, but viruses from the predominant subclade 3C.2a1b, and clade 3C.3a, were poorly inhibited. Ferret antisera raised against egg-propagated A/Switzerland/8060/2017 or A/Singapore/INFIMH-16-0019/2016-like (subclade 3C.2a1) viruses continued to poorly inhibit most of the recently circulating viruses. Ferret antisera raised against reference cell-grown viruses in subclades 3C.2a1 and 3C.2a2 or subgroup 3C.2a1b poorly inhibited genetic clade 3C.3a viruses. Likewise, ferret antisera raised against clade 3C.3a viruses propagated in cell culture, such as A/Kansas/14/2017 (the 2019-2020 northern hemisphere vaccine virus), inhibited clade 3C.3a viruses well but those in clade 3C.2a poorly. The majority of viruses from subgroups 3C.2a1b+131K and 3C.2a1b+128A+135K were well inhibited by ferret antisera raised against 3C.2a1b+131K viruses, including the cell reference virus A/South Australia/34/2019 and A/Iowa/60/2018. Ferret antisera raised against egg isolates of 3C.2a1b+131K viruses, including A/South Australia/34/2019, inhibited recent 3C.2a1b viruses better than antisera raised to the previous vaccine viruses A/Switzerland/8060/2017 and A/Singapore/INFIMH-16-0019/2016 (Table 1).

Human serology studies, using the serum panels described above, showed that geometric mean HI and VN titres of antibodies against most recent A(H3N2) viruses were reduced compared to HI and VN titres against the egg-propagated and cell-propagated vaccine viruses A/Singapore/INFIMH-16-0019/2016 and A/Switzerland/8060/2017.

One of 3424 influenza A(H3N2) viruses tested showed reduced inhibition by zanamivir. One thousand six hundred and twenty-seven A(H3N2) viruses were assessed for susceptibility to baloxavir by genetic and/or phenotypic analysis. Twelve viruses (one from the United States and 11 from Japan) contained amino acid substitutions at residue 38 in the PA which are known to confer reduced baloxavir susceptibility.

Table 1. A(H3N2) focus reduction assay

		2	3	4	5	6	8	9	10	11		
		A8538-14D	A8537-14D	A8659-14D	A8658-14D	F4381-14D	A8401-14D	A8336-14D	A/8585/14	A8493-14D		
		SIAT1	E4	SIAT3	E5	SIAT1	SIAT2/SIAT1	E6	S3,SIAT1	E8		
Reference Antigens		NEWC82	NEWC82	STHAUS34	STHAUS34	VIC653	SWITZ8060	SWITZ8060	KANS14	KANS14	**Passage Details**	**Sample Date**
	Clade	**3c.2a1b + 131K**	**3c.2a1b + 131K**	**3c.2a1b + 131K**	**3c.2a1b + 131K**	**3c.2a1b + 135K**	**3c.2a2re**	**3c.2a2re**	**3c.3a**	**3c.3a**		
A/Hong Kong/4801/2014	**3c.2a**	320	160	320	160	640	320	320	160	160	MDCKX, SIAT4	
A/Newcastle/82/2018	**3c.2a1b+131K**	**640**	160	640	80	1280	320	160	80	80	SIAT2	
A/Newcastle/82/2018	**3c.2a1b+131K**	640	**5120**	1280	5120	1280	640	2560	320	160	E4	
A/South Australia/34/2019	**3C.2a1b+131K**	2560	1280	**2560**	320	5120	2560	1280	640	640	SIAT3	
A/South Australia/34/2019	**3C.2a1b+131K**	640	2560	1280	**5120**	1280	1280	2560	160	80	E5	
A/Victoria/653/2017	**3c.2a1b+135K**	640	640	1280	320	**5120**	640	320	640	320	SIAT1	
A/Victoria/653/2017	**3c.2a1b+135K**	320	320	640	320	10240	640	1280	320	320	E5	
A/Switzerland/8060/2017	**3c.2a2re**	320	160	640	160	1280	**1280**	1280	80	80	SIAT2 SIAT2	
A/Switzerland/8060/2017	**3c.2a2re**	640	640	640	160	2560	10240	**10240**	320	320	E6	
A/Kansas/14/2017	**3c.3a**	160	320	320	160	640	160	320	**1280**	640	S3, SIAT1	
A/Kansas/14/2017	**3c.3a**	160	160	160	160	640	160	160	1280	**5120**	E8	
Test Antigens												
A/Darwin/593/2019		2560	1280	5120	320	5120	5120	1280	640	640	SIAT1	10/06/2019
A/Nakhonphanom/537/2019		2560	1280	5120	640	5120	5120	2560	1280	1280	SIAT1	07/05/2019
A/Sydney/164/2019		2560	1280	5120	640	5120	5120	1280	1280	2560	SIAT2	27/06/2019
A/Christchurch/522/2019		2560	2560	2506	640	5120	2560	1280	640	640	SIAT2	02/06/2019
A/South Australia/348/2019		1280	640	10240	320	5120	2560	640	1280	1280	SIAT2	01/07/2019
A/Victoria/1014/2019		1280	640	2560	320	5120	2560	640	320	320	SIAT1	09/07/2019
A/Victoria/157/2019		2560	1280	2560	320	5120	2560	1280	640	320	SIAT2	01/07/2019
A/Victoria/197/2019		1280	640	1280	160	5120	2560	640	320	320	SIAT2	04/06/2019
A/Victoria/206/2019		1280	640	2560	320	5120	2560	1280	640	640	SIAT2	25/07/2019
A/Canberra/217/2019		1280	640	2560	640	5120	1280	640	320	640	SIAT2	03/05/2019
A/Chanthaburi/2377/2019		2560	640	2560	160	5120	1280	640	160	160	SIAT1	05/05/2019
A/Sydney/134/2019	3c.3a	320	640	640	640	2560	1280	640	5120	5120	MDCK-Siat, SIAT1	02/07/2019
A/Victoria/130/2019		1280	1280	5120	640	5120	1280	2560	1280	1280	SIAT2	02/07/2019
A/Victoria/199/2019		1280	320	1280	160	2560	1280	320	320	320	SIAT2	26/07/2019
A/Victoria/203/2019		1280	1280	2560	320	5120	1280	640	320	320	SIAT2	30/07/2019
A/Victoria/205/2019		1280	640	1280	160	2560	1280	640	160	160	SIAT2	23/07/2019
A/Victoria/2514/2019		2560	1280	1280	640	5120	1280	1280	320	640	SIAT3	25/07/2019
A/South Australia/1070/2019		640	640	1280	160	2560	640	640	160	160	SIAT1	09/05/2019
A/Victoria/112/2019		640	160	1280	80	2560	640	320	80	80	SIAT1	13/07/2019
A/South Australia/4/2019	3c.2a1b+135K	640	320	640	160	2560	320	320	320	320	SIAT1	01/01/2019
A/Cambodia/d0619386/20	3c.2a1b+131K	640	320	640	160	1280	320	320	160	160	Siat 2, SIAT1	04/01/2019
A/Singapore/GP0866/2019		1280	640	2560	160	2560	320	640	320	320	MDCK2, SIAT1	13/06/2019
A/Tasmania/557/2019		640	160	640	160	1280	320	320	160	80	SIAT1	06/06/2019
A/South Australia/2/2019	3c.2a1b+135K	320	160	320	80	1280	160	160	160	160	SIAT1	21/06/2019
A/Perth/1015/2019		80	160	80	80	320	80	160	640	320	SIAT1	08/06/2019

Influenza B viruses

All available HA gene sequences of B/Yamagata lineage viruses belonged to genetic clade 3. In HI assays the vast majority of recently circulating B/Yamagata lineage viruses were well inhibited by post-infection ferret antisera raised against cell culture- or egg-propagated B/Phuket/3073/2013-like viruses.

The HA gene sequences of the B/Victoria lineage viruses characterized belonged to genetic clade 1A, but significant genetic diversity continued to be observed. Viruses with a three amino acid deletion in HA (amino acids 162-164) were predominant in most countries, although in some countries (e.g. Madagascar, Mozambique and many countries in Central and South America) viruses with a two amino acid deletion in HA (amino acids 162 and 163) were dominant. Viruses without HA amino acid deletions were in the minority. A majority of viruses with the three amino acid deletion were inhibited well by post-infection ferret antisera raised against both cell culture- or egg-propagated triple deletion viruses that contained HA1 G133R and K136E amino acid substitutions, such as B/Sichuan-Gaoxin/531/2018 and B/Washington/02/2019 (Table 2). Many viruses with the three amino acid deletion in HA and those without HA amino acid deletions were poorly inhibited by post-infection ferret antisera raised against both egg- and cell culture-propagated B/Colorado/06/2017-like viruses. In contrast, viruses with the two amino acid deletion were inhibited well by the antisera raised against B/Colorado/06/2017-like viruses.

Human serology studies, using the serum panels described above, showed generally minor reductions in post-vaccination HI geometric mean titres against representative recent B/Yamagata lineage viruses when compared to the cell culture-propagated B/Phuket/3073/2013 reference virus. Post-vaccination HI geometric mean titres against recent viruses of the B/Victoria lineage representing the three major genetic groups, with three, two or no amino acid deletions in the HA, showed small to medium reductions when compared to egg- or cell culture-propagated B/Colorado/06/2017 reference viruses.

Of the 1727 influenza B viruses screened for NAI susceptibility, 18 showed reduced susceptibility. A total of 755 viruses were screened for susceptibility to baloxavir, with 97 of these being tested phenotypically; all retained normal susceptibility.

Table 2. Antigenic analysis of influenza B/Victoria lineage viruses – HI assay

		REFERENCE FERRET ANTISERA					
		V1A		V1A-2DEL		V1A-3DEL	
REFERENCE VIRUSES	HA CLADE	EGG BRI/60	CELL BRI/60	EGG CO/06	CELL IA/06	EGG WA/02	CELL WA/02
B/BRISBANE/60/2008 (EGG)	V1A	**2560**	1280	320	160	320	80
B/BRISBANE/60/2008 (CELL)	V1A	2560	**1280**	160	160	80	80
B/COLORADO/06/2017 (EGG)	V1A-2DEL	320	160	**160**	160	160	80
B/IOWA/06/2017 (CELL)	V1A-2DEL	40	80	40	**320**	40	40
B/WASHINGTON/02/2019 (EGG)	V1A-3DEL	320	160	160	40	**320**	160
B/WASHINGTON/02/2019 (CELL)	V1A-3DEL	20	40	10	80	320	**640**
TEST VIRUSES							
B/IDAHO/09/2019	V1A	160	640	20	80	40	40
B/NEW YORK/11/2019	V1A	160	640	20	80	40	40
B/MANAGUA/1377-18/2019	V1A-2DEL	40	80	80	320	80	40
B/ESTELI/1349-18/2019	V1A-2DEL	20	40	40	320	40	40
B/MOZAMBIQUE/54/2019	V1A-2DEL	20	40	40	320	40	40
B/TEXAS/24/2019	V1A-2DEL	20	80	80	160	40	40
B/LOUISIANA/18/2019	V1A-3DEL	80	80	20	160	80	160
B/OMAN/2250/2019	V1A-3DEL	640	320	320	160	320	320
B/GEORGIA/05/2019	V1A-3DEL	320	320	160	80	320	320
B/MARYLAND/17/2019	V1A-3DEL	20	80	10	80	320	320
B/NIGERIA/3624/2018	V1A-3DEL	20	80	20	40	80	160
B/WISCONSIN/21/2019	V1A-3DEL	10	40	10	40	160	160

Recommended composition of influenza virus vaccines for use in the 2020 southern hemisphere influenza season

WHO recommends that quadrivalent influenza vaccines for use in the 2020 southern hemisphere influenza season contain the following:

- **an A/Brisbane/02/2018 (H1N1)pdm09-like virus;**
- **an A/South Australia/34/2019 (H3N2)-like virus;**
- **a B/Washington/02/2019-like (B/Victoria lineage) virus; and**
- **a B/Phuket/3073/2013-like (B/Yamagata lineage) virus.**

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- **a B/Washington/02/2019-like (B/Victoria lineage) virus.**

Lists of egg- or cell culture-propagated candidate vaccine viruses (CVVs) suitable for use in human vaccine production are available on the WHO website[4]. A list of reagents for vaccine standardization, including those for this recommendation, can also be found on the WHO website. CVVs for zoonotic influenza viruses are listed on the same website.

As in previous years, national or regional authorities approve the composition and formulation of vaccines used in each country. National public health authorities are responsible for making recommendations regarding the use of the vaccine. WHO has published recommendations on the prevention of influenza[5].

CVVs (including reassortants) and reagents for use in the laboratory standardisation of inactivated vaccines may be obtained from:

- Biomedicines and Influenza Vaccines Section, Laboratories Branch, Medical Devices and Product Quality Division, Therapeutic Goods Administration, P.O. Box 100, Woden, ACT, 2606, Australia (fax: +61262328564, email: influenza.reagents@health.gov.au; web site: http://www.tga.gov.au)
- Division of Virology, National Institute for Biological Standards and Control, a centre of the Medicines and Healthcare products Regulatory Agency (MHRA), Blanche Lane, South Mimms, Potters Bar, Hertfordshire, EN6 3QG, UK (fax: +441707641050, e-mail: enquiries@nibsc.org, web site: http://www.nibsc.org/science_and_research/virology/influenza_resource_.aspx
- Division of Biological Standards and Quality Control, Center for Biologics Evaluation and Research, Food and Drug Administration, 10903 New Hampshire Avenue, Silver Spring, Maryland, 20993, USA (fax: +1 301 480 9748), email: cbershippingrequests@fda.hhs.gov)
- Influenza Virus Research Center, National Institute of Infectious Diseases, 4-7-1 Gakuen, Musashi-Murayama, Tokyo 208-0011, Japan (fax: +81425616156, email: flu-vaccine@nih.go.jp)

Requests for reference viruses should be addressed to:

- WHO Collaborating Centre for Reference and Research on Influenza, VIDRL, Peter Doherty Institute, 792 Elizabeth Street, Melbourne, Victoria 3000, Australia (fax: +61393429329, web

[4] http://www.who.int/influenza/vaccines/virus/candidates_reagents/home
[5] http://www.who.int/wer/2012/wer8747.pdf

site: http://www.influenzacentre.org, email: whoflu@influenzacentre.org)

- WHO Collaborating Centre for Reference and Research on Influenza, National Institute of Infectious Diseases, 4-7-1 Gakuen, Musashi-Murayama, Tokyo 208-0011, Japan (fax: +81425616149 or +81425652498, email: whocc-flu@nih.go.jp
- WHO Collaborating Centre for Surveillance, Epidemiology and Control of Influenza, Centers for Disease Control and Prevention, 1600 Clifton Road, Mail Stop H17-5, Atlanta, GA 30329, United States (fax: +14046390080, web site: http://www.cdc.gov/flu/, email: influenzavirussurveillance@cdc.gov)
- WHO Collaborating Centre for Reference and Research on Influenza, The Francis Crick Institute, 1 Midland Road, London NW1 1AT, UK (Tel: +44 203 796 1520 or +44 203 796 2444) (website: http://www.crick.ac.uk/research/worldwide-influenza-centre email: whocc@crick.ac.uk)
- WHO Collaborating Centre for Reference and Research on Influenza, National Institute for Viral Disease Control and Prevention, China CDC, 155 Changbai Road, Changping District, 102206, Beijing, P.R. China. (tel: +86 10 5890 0851, fax: +86 10 5890 0851, email: whocc-china@cnic.org.cn, website: http://www.chinaivdc.cn/cnic/en).

WHO provides fortnightly updates[6] of global influenza activity. Other information about influenza surveillance can be found on the WHO Global Influenza Programme website[7].

Acknowledgements

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[6] http://www.who.int/influenza/surveillance_monitoring/updates/en/
[7] http://www.who.int/influenza/gip