

RECOMMENDATION FOR SEASONAL INFLUENZA VACCINE COMPOSITION FOR NEW ZEALAND FOR 2020

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The Australian Influenza Vaccine Committee (AIVC) met with a New Zealand representative (Appendix 1) in Canberra on 9 October 2019 to consult on the influenza vaccine composition for 2020 for New Zealand, Australia and South Africa (Table 1).

The recommended composition for quadrivalent vaccines was:

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

The recommended composition for trivalent vaccines was:

- A(H1N1) an A/Brisbane/02/2018 (H1N1)pdm09-like virus
- A(H3N2) an A/South Australia/34/2019 (H3N2)-like virus
- B a B/Washington/02/2019-like virus (belonging to B/Victoria lineage)

Decision		Use vear	A H3N2	A H1N1	B (Trivalent)	B (Quadrivalent)
NZ & WHO*	2019	2020	A/South Australia/34/2019	A/Brisbane/02/2018	B/Phuket/3073/2013	B/Washington/02/2019
NZ & WHO*	2018	2019	A/Switzerland/8060/2017	A/Michigan/45/2015	B/Phuket/3073/2013	B/Colorado/06/2017
NZ & WHO*	2017	2018	A/Singapore/INFIMH-16- 0019/2016	A/Michigan/45/2015	B/Phuket/3073/2013	B/Brisbane/60/2008
NZ & WHO*	2016	2017	A/Hong Kong/4801 /2014	A/Michigan/45/2015	B/Brisbane/60/2008	B/Phuket/3073/2013
NZ & WHO*	2015	2016	A/Hong Kong/4801 /2014	A/California/7/2009	B/Brisbane/60/2008	B/Phuket/3073/2013
NZ & WHO*	2014	2015	A/Switzerland/97152 93/2013	A/California/7/2009	B/Phuket/3073/2013	B/Brisbane/60/2008
NZ & WHO*	2013	2014	A/Texas/50/2012	A/California/7/2009	B/Massachusetts/2/2012	B/Brisbane/60/2008
NZ & WHO*	2012	2013	A/Victoria/361/2011	A/California/7/2009	B/Wisconsin/1/2010	
NZ & WHO*	2011	2012	A/Perth/16/2009	A/California/7/2009	B/Brisbane/60/2008	
NZ & WHO*	2010	2011	A/Perth/16/2009	A/California/7/2009	B/Brisbane/60/2008	
NZ & WHO*	2009	2010	A/Perth/16/2009	A/California/7/2009	B/Brisbane/60/2008	
NZ & WHO*	2008	2009	A/Brisbane/10/2007	A/Brisbane/59/2007	B/Florida/4/2006	
NZ & WHO*	2007	2008	A/Brisbane/10/2007	A/Solomon Islands/3/20	B/Florida/4/2006	
NZ & WHO*	2006	2007	A/Wisconsin/67/2005	A/New Caledonia/20/99	B/Malaysia/2506/2004	
NZ & WHO*	2005	2006	A/California/7/2004	A/New Caledonia/20/99	B/Malaysia/2506/2004	
NZ & WHO*	2004 2005		A/Wellington/1/2004	A/New Caledonia/20/99	B/Shanghai/361/2002	
NZ & WHO*	2003	2004	A/Fujian/411/2002	A/New Caledonia/20/99	B/Hong Kong/330/2001	
NZ & WHO*	2002	2003	A/Moscow/10/99	A/New Caledonia/20/99	B/Hong Kong/330/2001	
NZ & WHO*	2001	2002	A/Moscow/10/99	A/New Caledonia/20/99	B/Sichuan/379/99	
NZ	2000	2001	A/Sydney/5/97	A/New Caledonia/20/99	B/Beijing/184/93	
WHO*	2000	2001	A/Moscow/10/99	A/New Caledonia/20/99	B/Beijing/184/93	
NZ & WHO*	1999	2000	A/Sydney/5/97	A/Beijing/262/95	B/Beijing/184/93	
NZ	1998	1999	A/Sydney/5/97	A/Bayern/7/95	B/Beijing/184/93	
WHO**	1997–98		A/Wuhan/359/95	A/Bayern/7/95	B/Beijing/184/93	
NZ	1997	1998	A/Wuhan/359/95	A/Texas/36/91	B/Beijing/184/93	
WHO**	1996–97		A/Wuhan/359/95	A/Singapore/6/86***	B/Beijing/184/93	
NZ	1996	1997	A/Johannesburg/33/94	A/Texas/36/91	B/Beijing/184/93	
WHO**	1995-96		A/Johannesburg/33/94	A/Singapore/6/86	B/Beijing/184/93	
NZ	1995	1996	A/Guangdong/25/93	A/Texas/36/91	B/Panama/45/90	
WHO**	1994–95		A/Shangdong/9/93	A/Singapore/6/86	B/Beijing/184/93	
NZ	1994	1995	A/Beijing/32/92	A/Texas/36/91	B/Panama/45/90	
WHO**	1993–94		A/Beijing/32/92	A/Singapore/6/86	B/Panama/45/90	
NZ	1993	1994	A/Shanghai/24/90	A/Texas/36/91	B/Panama/45/90	
WHO**	1992–93		A/Beijing/353/89	A/Singapore/6/86	B/Yamagata/16/88 or B/Panama/45/90	
NZ	1992	1993	A/Beijing/353/89	A/Victoria/36/88	B/Yamagata/16/88 or B/Panama/45/90	
WHO**	1991–92		A/Beijing/353/89	A/Singapore/6/86	B/Yamagata/16/88 or B/Panama/45/90	

Table 1. Influenza vaccine recommendations for New Zealand, 1991–2020

* WHO recommendations are for the Southern Hemisphere winter

* * WHO recommendations are for the Northern Hemisphere winter

In 2019, influenza activity in New Zealand is described at a low level. Virus transmissibility which reflects the ease of movements of the influenza virus between individuals and communities was low as measured by influenza-associated influenza-like illness (ILI) consultations. Overall impact on healthcare in hospitals and ICU admissions was moderate as measured by influenza-associated severe acute respiratory illness (SARI). Seriousness of disease (i.e. severity) that indicates the extent to which individuals get sick when infected with the influenza virus was low as measured by the ratio of influenza-associated ICU admission over influenza-associated hospitalization.

The national sentinel GP-based influenza-like illness was low based on the ILI data during 2013–2019. The ILI peak consultation rate of 50 per 100,000 in 2019 was the fourth lowest when compared to that of the 2013–2018 data. Again, the ILI activity in 2019 had uneven geographical distribution. The influenza-associated ILI disease burden was higher in children aged 1–19 years and young adults (35–49 years) compared to other age groups. Influenza-associated ILI consultations were high in Asian, Maori and Europeans ethnic groups.

The hospital-based severe acute respiratory illness was low based on the SARI data during 2012–2019. However, influenza–associated SARI hospitalization in 2019 was at a moderate level. Influenza–associated SARI hospitalizations were higher in both young children (0–4 years) and elderly (\geq 65) compared to other age groups; also higher in Pacific Peoples and Maori ethnic groups compared to other ethnic groups.

A total of 1545 influenza viruses were detected and reported through sentinel ILI and SARI surveillance system in 2019, with influenza A representing 54.4% (840/1545) and influenza B 45.6% (705/1545) of all influenza viruses. Influenza B/Victoria lineage and A(H3N2) viruses were two predominant strains co-circulating during the winter. Among the 539 lineage-typed influenza B viruses, all (100%) belonged to the Victoria lineage. Among the 648 sub-typed influenza A viruses, 80.2% (520/648) were A(H3N2) viruses and 19.8% (128/648) were A(H1N1)pdm09 viruses.

During 1 January to 1 September 2019, sequencing analysis as well as antigenic characterization were carried out. WHOCC-Melbourne's hemagglutination inhibition (HAI) reference kit for 2018 was used before July 2019 and WHOCC's HAI kit for 2019 was used after July 2019 once it was received:

- A(H1N1)pdm09: Of the 48 A(H1N1)pdm09 viruses that were antigenically typed, all were antigenically related to the reference strain A/Michigan/45/2015. Genetically, the NZ influenza A(H1N1)pdm09 viruses fell into subclade 6B.1.
- A(H3N2): Of 91 A(H3N2) viruses that were antigenically typed using antisera against A/Singapore/INFIMH-16-0019/2016, 89 (98%, 89/91) were antigenically related to the reference strain A/Singapore/INFIMH-16-0019/2016, 2 (2%, 2/91) had reduced reactivity against the reference vaccine strain. Of 49 A(H3N2) viruses that were antigenically typed using antisera against A/Switzerland/8060/2017, 13 (27%, 13/49) were antigenically related to the reference strain A/Switzerland/8060/2017, and 36 (73%, 36/49) had reduced reactivity against the reference vaccine strain. Genetically, most of NZ influenza A(H3N2) viruses mainly fell into subclade 3C.2a1b.
- B/Victoria lineage: Of the 167 B/Victoria lineage viruses that were antigenically typed using antisera against B/Brisbane/60/2008-like virus, 75 (45%, 75/167) were antigenically related to the reference strain, and 92 (55%, 92/167) had reduced reactivity against the reference strain. Of the 179 B/Victoria lineage isolates were antigenically typed using antisera against B/Colorado/6/2017-like virus, 118 (66%, 118/179) were antigenically related to the reference

strain, and 61 (34%, 61/179) had reduced reactivity against the reference strain. Genetically, NZ influenza B/Victoria lineage viruses in 2019 fell into genetic clade V1A.

• B/Yamagata lineage: Of the 2 B/Yamagata lineages viruses that were antigenically typed using antisera against B/Phuket/3073/2013-like virus, all were antigenically related to the reference strain B/Phuket/3073/2013.

All of the 45 tested influenza viruses in 2019 were sensitive to oseltamivir and zanamivir.

Overall, the vaccine effectiveness was moderate. Influenza vaccination provided 50% (95% CI: 22 to 68) protection against laboratory-confirmed influenza-associated SARI hospitalisations. Influenza vaccination provided 25% (95% CI: 3 to 42) protection against influenza-associated ILI consultations.

The national influenza surveillance system in New Zealand is an essential public health tool for assessing and implementing strategies to control influenza. The surveillance system includes community-based surveillance (National sentinel general practice surveillance, Healthline - telephone health advice service) and hospital-based surveillance (SARI surveillance and Ministry of Health data on publicly funded hospital discharges).

3.1 NATIONAL SENTINEL GENERAL PRACTICE SURVEILLANCE

New Zealand's longitudinal sentinel GP-based surveillance system was established in 1989 as part of the World Health Organization's (WHO) Global Influenza Surveillance and Response System. It is operated nationally by ESR and locally by influenza surveillance co-ordinators in the public health services (PHS). Previously (1989–2015), every week during the influenza season from May to September (weeks 18–39), GPs are required to record the number of consultations for influenza-like illness (ILI) each week and the age group of the patient (<1, 1–4, 5–19, 20–34, 35–49, 50–64, 65+), for each case patient who meets the case definition for ILI, on a standardised form.

While the sentinel GP-based surveillance system has been operating successfully for a number of years, the manual method of data collection is outdated and time-consuming. The process adds extra time to the sentinel practices during the busy winter season and only provides the surveillance system with very limited consultation data.

In 2016, a modernised electronic data collection was introduced, enhanced influenza-like illness surveillance (e-ILI). It used an interactive advance form designed by HealthLink to record a consultation-seeking patient with ILI. Symptoms and onset dates including demography (age, sex, and ethnicity), clinical information, medication, vaccination status, and specimen collection were collected electronically and data was sent directly to ESR.

The ILI case definition was also modified to "an acute respiratory illness with a history of fever or measured fever of \geq 38°C, AND cough, AND onset within the past 10 days".

The syndromic eILI surveillance was all-year-round. The virological specimen collection and testing for those ILI patients was only during the influenza season, May-September inclusive.

Each participating practice from the Auckland and Wellington regions collected respiratory samples (ie, a nasopharyngeal or throat swab) from all ILI patients seen. For the remaining areas, three respiratory samples, one each from the first ILI patient examined on Monday, Tuesday and Wednesday were collected weekly.

All practices forwarded these samples to the WHO National Influenza Centre at ESR apart for those in the Canterbury, South Canterbury and West Coast DHBs who forwarded their samples to Canterbury Health Laboratories for virus characterisation. Laboratory identification included molecular detection using the polymerase chain reaction (PCR), isolation of the virus or direct detection of viral antigens. Influenza viruses were typed as A or B. Influenza A viruses were further sub-typed as A(H3N2) or A(H1N1)pdm09. Influenza B viruses were further lineage-typed as B/Yamagata or B/Victoria lineage. Eight non-influenza respiratory viruses were also tested: respiratory syncytial virus, parainfluenza virus types 1, 2 and 3, rhinovirus, adenovirus, human metapneumovirus and enterovirus.

Canterbury Health Laboratory reported to ESR weekly on the total number of swabs received from each DHB and the influenza viruses identified, and updated details on influenza types and sub-types from previous weeks. ESR reports national information on epidemiological and virological surveillance of influenza weekly and yearly to relevant national and international organisations, including the WHO, with reports published on the ESR website: https://surv.esr.cri.nz/virology.php.

Consultation rates were calculated using the registered patient populations of the participating practices as a denominator.

The values for the different intensity levels were based on the framework from Pandemic Influenza Severity Assessment (PISA) – a WHO guide to assess the severity of influenza in seasonal epidemics and pandemics (<u>http://apps.who.int/iris/bitstream/handle/10665/259392/WHO-WHE-IHM-GIP-2017.2-eng.pdf;jsessionid=0D8C59A6D4E84A8C5AB9329CADAE374D?sequence=1</u>)

In 2019, 80 sentinel practices were recruited from all 20 DHBs under ESR's sentinel GP-based surveillance with a total patient roll of 537137. Virus transmissibility which reflects the ease of movements of the influenza virus between individuals and communities was low in 2019. Influenza like illness (ILI) and ILI-associated influenza consultation rates in 2019 were low and the activity was earlier than previous years of 2013-2018 (Figures 1-4). From week 18 (commencing 29 April) through week 21 (ending 26 May), consultation activity was below the low seasonal threshold but increased steadily, then it passed the baseline level and reached peak at week 26 (ending 30 June). Since then, it started to decline steadily and below the baseline level after week 29 (ending 21 July).



Figure 1. Weekly consultation rates for influenza-like illness in New Zealand, 2019

Week 2019



Figure 2. Weekly ILI consultation rates in 2019 compared to 2013–2018

Figure 3. Weekly ILI-associated influenza rates in 2019 compared to 2013–2018





Figure 4. Weekly Consultation Rates for Influenza-like Illness in New Zealand, 1992–2019

From week 18 (commencing 29 April 2019) through week 35 (ending 1 September 2019), a total of 2898 consultations for ILI were reported from the 20 DHBs. The cumulative incidence of ILI consultation during this period was 657.7 per 100,000 population. The average weekly ILI consultation rate between weeks 18 and 35 was 36.5 per 100,000 population. Among the patients that met the ILI case definition, 2025 (70.6%) had a specimen tested for influenza. Of these, 1148 (56.7%) cases had influenza viruses detected and the cumulative influenza incidence during 29 April to 1 September 2019 was 369.2 per 100,000 population (Table 2).

	ILI & influenza cases among sentinel practices								
Characteristics	ILI cases	Influenza cases	Prop Influenza positive ¹ (%)	ILI incidence (per 100 000)	Influenza incidence ² (per 100 000)				
Overall	2898	1148	56.1 (100.0)	657.7	369.2				
Age group (years)									
<1	26	4	28.6 (0.3)	572.6	163.6				
1-4	240	70	50.7 (6.1)	994.7	504.6				
5–19	877	477	73.4 (41.6)	992.9	728.6				
20–34	635	232	53.7 (20.2)	704.2	378.2				
35–49	474	166	47.7 (14.5)	562.9	268.5				
50–64	410	123	43.6 (10.7)	497.0	216.8				
65–79	184	59	42.4 (5.1)	354.9	150.6				
>80	52	17	40.5 (1.5)	348.2	140.9				
Unknown	0	0	0.0						
Ethnicity									
Māori	379	161	59.4 (14.0)	632.0	375.5				
Pacific peoples	110	37	61.7 (3.2)	401.5	247.6				
Asian	286	115	55.3 (10.0)	745.7	412.3				
European and Other	2123	835	55.4 (72.7)	674.1	373.8				
Unknown	0	0	0.0						
Sex									
Female	1614	608	53.5 (53.0)	698.0	373.6				
Male	1284	540	59.4 (47.0)	613.2	364.3				
Unknown	0	0	0.0						

Table 2. Demographic characteristics of ILI and influenza cases, 29 April – 1 September 2019

¹Proportion of cases tested which were positive for influenza viruses

²Adjusted to positivity of tested cases

As in previous years, 2019 consultation rates for ILI varied greatly among DHBs (Table 3). From week 18 (commencing 29 April) through week 35 (ending 1 September), Auckland DHB had the highest average consultation rate (76.7 per 100,000), followed by South Canterbury (71.5 per 100,000), and Whanganui (68.8 per 100,000).

рир								R	ate pei	r 100 0	00								Average
טווט	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	rate
Auckland	40.4	49.8	59.2	87.5	127.8	138.6	153.4	170.9	153.4	121.1	64.6	56.5	37.7	30.9	35.0	25.6	18.8	9.4	76.7
Bay of Plenty	4.5	4.5	22.7	22.7	40.8	9.1	58.9	49.8	36.3	45.3	77.0	40.8	13.6	9.1	4.5	0.0	0.0	4.5	24.7
Canterbury	6.4	12.8	16.7	32.1	28.2	30.8	47.5	41.1	47.5	59.1	66.8	42.4	42.4	25.7	15.4	24.4	12.8	16.7	31.6
Capital and Coast	7.5	27.4	22.4	39.8	27.4	39.8	32.4	42.3	59.7	94.6	42.3	52.3	24.9	29.9	37.3	32.4	27.4	10.0	36.1
Counties Manukau	2.2	11.0	6.6	11.0	4.4	8.8	6.6	4.4	4.4	4.4	4.4	2.2	4.4	0.0	2.2	2.2	2.2	2.2	4.6
Hawke's Bay	33.6	20.2	6.7	20.2	40.3	13.4	20.2	40.3	73.9	40.3	26.9	26.9	40.3	20.2	13.4	13.4	13.4	13.4	26.5
Hutt Valley	0.0	0.0	8.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.5
Lakes	45.9	22.9	45.9	22.9	45.9	0.0	137.6	91.8	160.6	160.6	45.9	68.8	68.8	45.9	68.8	0.0	45.9	68.8	63.7
MidCentral	0.0	4.0	4.0	2.0	0.0	14.0	10.0	8.0	6.0	4.0	6.0	14.0	6.0	6.0	4.0	4.0	2.0	2.0	5.3
Nelson Marlborough	13.2	33.0	19.8	39.6	26.4	33.0	46.2	59.4	59.4	26.4	13.2	13.2	0.0	19.8	19.8	6.6	6.6	0.0	24.2
Northland	17.3	43.4	65.1	39.0	65.1	30.4	21.7	17.3	21.7	47.7	39.0	26.0	21.7	17.3	21.7	4.3	8.7	4.3	28.4
South Canterbury	18.0	9.0	9.0	117.0	81.0	81.0	171.0	117.0	108.0	135.0	144.0	27.0	72.0	9.0	45.0	45.0	36.0	63.0	71.5
Southern	12.9	12.9	7.7	15.5	5.2	7.7	31.0	18.1	33.6	41.3	56.8	46.5	38.7	25.8	25.8	25.8	18.1	7.7	23.9
Tairawhiti	0.0	16.4	32.8	49.2	114.7	65.5	32.8	16.4	81.9	81.9	81.9	16.4	0.0	0.0	16.4	32.8	0.0	0.0	35.5
Taranaki	0.0	0.0	4.3	8.6	4.3	0.0	0.0	0.0	0.0	12.8	4.3	4.3	8.6	4.3	0.0	0.0	0.0	0.0	2.9
Waikato	11.9	3.0	6.0	9.0	20.9	17.9	38.8	23.9	32.8	44.8	35.8	41.8	35.8	14.9	17.9	6.0	11.9	17.9	21.7
Wairarapa	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Waitemata	12.3	0.0	12.3	41.1	53.5	37.0	61.7	57.6	102.8	65.8	69.9	32.9	24.7	8.2	20.6	16.4	4.1	0.0	34.5
West Coast	5.8	5.8	0.0	0.0	5.8	5.8	17.3	5.8	11.6	17.3	11.6	17.3	5.8	5.8	11.6	0.0	0.0	0.0	7.1
Whanganui	0.0	190.5	95.2	95.2	0.0	0.0	190.5	0.0	95.2	0.0	0.0	0.0	95.2	190.5	95.2	0.0	95.2	95.2	68.8
New Zealand	12.7	17.9	20.7	32.4	38.4	37.6	50.6	48.4	53.8	53.8	43.0	32.8	25.7	17.5	18.6	15.1	11.4	9.3	30.0

Table 3. Weekly consultation rate for influenza-like illness by District Health Board, 2019

Between 29 April to 1 September 2019, a total of 2063 ILI specimens were tested for influenza viruses (Table 4) and 1156 (56.0%) were positive, with similar numbers of influenza B (595) and influenza A (561) viruses detected. Additionally, a total of 1959 ILI specimens were tested for non-influenza viruses and 382 (19.5%) were positive with non-influenza viruses.

Table 4. Influenza and non-influenza respiratory viruses among ILI cases, 29 April – 1 September 2019

Influenza viruses	ILI
	Cases (%)
No. of specimens tested	2063
No. of positive specimens (%) ¹	1156 (56.0)
Influenza A	561
A (not subtyped)	59
A(H1N1)pdm09	86
A(H1N1)pdm09 by PCR	64
A/Michigan/45/2015 (H1N1)pdm09 - like	22
A(H3N2)	416
A(H3N2) by PCR	380
A/Switzerland/8060/2017 (H3N2)-like	36
Influenza B	595
B (lineage not determined)	60
B/Yamagata lineage	0
B/Yamagata lineage by PCR	0
B/Phuket/3073/2013 - like	0
B/Victoria lineage	535
B/Victoria lineage by PCR	271
B/Colorado/06/2017-like	264
Influenza and non-influenza co-detection (% +ve)	54 (4.7)

Non-influenza respiratory viruses	ILI
	Cases (%)
No. of specimens tested	1959
No. of positive specimens (%) ¹	382 (19.5)
Respiratory syncytial virus (RSV)	98
Parainfluenza 1 (PIV1)	1
Parainfluenza 2 (PIV2)	18
Parainfluenza 3 (PIV3)	22
Rhinovirus (RV)	164
Adenovirus (AdV)	28
Human metapneumovirus (hMPV)	58
Enterovirus	13
Single virus detection (% of positives)	363 (95.0)
Multiple virus detection (% of positives)	19 (5.0)

¹Number of specimens positive for at least one of the listed viruses; note a specimen may be positive for more than one virus.

The temporal distribution of the number and proportion of the influenza viruses and non-influenza respiratory viruses, from 29 April to 1 September 2019, is shown in Figures 5 & 6. Influenza A(H3N2) and B/Victoria lineage viruses were the two main predominant strains co-circulating at almost the same level during this period.









3.2 HEALTHLINE

Healthline is the free national 0800 24 hour telephone health advice service funded by the Ministry of Health. Calls made to Healthline are triaged using electronic clinical decision support software. Data collected are daily counts of all symptomatic calls made to Healthline and those triaged for Influenza-Like-Illness (ILI). Note that about 70% of all calls to Healthline are symptomatic (other calls not part of this analysis include queries for information etc).

Analysis is frequency based with alarms raised by identifying statistical deviations (aberations) from previous calls. Data are reported for all ages and in five age bands (0–4, 5–14, 15–44, 45–64, 65+ years). The analysis of the call frequency is based on the cumulative summation (CUSUM) algorithm implemented in Early Aberration Reporting System (EARS) application developed by the Centres for Disease Control and Prevention (CDC), Atlanta, United States. EARS has three sensitivity thresholds (high, medium and low). If the daily call count exceeds a threshold a flag is signalled.

Cases of ILI are defined as those that are recorded in the Healthline database as having one of the following 18 guidelines: adult fever; breathing problems; breathing difficulty – severe (paediatric); colds (paediatric); cough (paediatric); cough – adult; fever (paediatric); flu-like symptoms or known/suspected influenza; flu like symptoms pregnant; influenza (paediatric); headache; headache (paediatric); muscle ache/pain; sore throat (paediatric); sore throat/hoarseness; sore throat/hoarseness pregnant; upper respiratory tract infections/colds; upper respiratory tract infections/colds – pregnant.

Figure 7 shows the weekly number of calls to Healthline for ILI during 2010–2019. Healthline calls in 2019 were in the middle range, similar to the level in 2014.



Figure 7. Weekly number of ILI-related calls to Healthline, 2010–2019

4. HOSPITAL-BASED SURVEILLANCE

4.1 HOSPITAL-BASED SEVERE ACUTE RESPIRATORY ILLNESS (SARI) SURVEILLANCE

Inpatients with suspected respiratory infections admitted overnight to any of the four hospitals (Auckland City Hospital and the associated Starship Children's Hospital, Middlemore Hospital and the associated Kidz First Children's Hospital) in the Auckland District Health Board (ADHB) and Counties Manukau DHB, were screened by research nurses each day. Overnight admission is defined as: "*A patient who is admitted under a medical team, and to a hospital ward or assessment unit*". Case ascertainment followed a surveillance algorithm. The presence of the components of the case definition was determined through a combination of reviewing the clinician's admission diagnoses and by interviewing patients about their presenting symptoms. Records of all patients admitted overnight to medical wards were reviewed daily to identify anyone with a suspected respiratory infection. These patients were categorised into one of ten admission diagnostic syndrome groups. Research nurses then interviewed the patients and documented the components of the case definition of severe acute respiratory illness (SARI) that were present and ascertain the patients whether meeting the SARI case definition.

The case definition being used is the World Health Organisation (WHO) SARI case definition: "an acute respiratory illness with a history of fever or measured fever of ≥38°C, and cough, and onset within the past 10 days, and requiring inpatient hospitalisation". If a patient with suspected respiratory infection met the SARI case definition, a respiratory sample was collected to test for influenza and other respiratory pathogens. In addition, patient information was captured via a case report form which included patient demographics, presenting symptoms and illness, pre-hospital healthcare, medication usage, influenza vaccination history, co-morbidities, disease course and outcome, including major treatments, ICU admission and mortality, epidemiologic risk factors and laboratory results.

The total numbers of all new hospital inpatient acute admissions and newly assessed and tested patients, including ICU admissions and deaths were collected. This allowed calculation of population-based incidence for SARI and associated influenza cases overall and stratified by age, sex, ethnicity and socio-economic status among the ADHB and CMDHB resident population (from 2013 census data). Incidence rates were calculated along with 95% confidence intervals (95%CI). In addition, this allowed the calculation of the proportion of SARI and associated influenza cases, including ICU admissions and deaths, by overall and stratified patients, among all acute admissions regardless of residence status. An acute admission is defined as an unplanned admission on the day of presentation at the admitting health care facility. Admission may have been from the emergency or outpatient departments of the health care facility, a transfer from another facility or a referral from primary care.

Overall impact on healthcare use in hospitalizations and ICU admissions was low in 2019. From week 18 (commencing 29 April 2019) through week 35 (ending 1 September 2019), Severe acute respiratory illness (SARI) hospitalization rates were at a low level (Figures 8 and 9). From week 18 through week 26 (ending 30 June), SARI hospitalisation rates was below the seasonal threshold, then it passed the baseline and reached the peak at week 27 (ending 7 July) with a second peak at week 29 (ending 21 July August). Since then, it started to decline steadily and below the baseline level after week 29.

From the start of week 18, SARI-associated influenza hospitalization rate has already passed the baseline. The influenza hospitalization rate remained low but climbed up quickly between week 18 through week 20 (ending 19 May), then it reached moderate seasonal level with the first peak at week 23 (ending 9 June) and the second peak at week 27 (ending 7 July). Since then, it started to decline gradually and below the baseline level after week 34 (ending 25 August) (Figure 10).



Figure 8. Weekly resident SARI and SARI-associated influenza incidence, 2019

Week 2019





Figure 10. Weekly hospitalisation rates for SARI-associated influenza in 2019 compared to 2012–2018

7



Seriousness of disease (i.e. severity) that indicates the extent to which individuals get sick when infected with the influenza virus was low in 2019 as measured by the ratio of influenza-associated ICU admission over influenza-associated hospitalization. It has been noticed that when influenza A(H1N1)pdm09 virus was the predominant strain in 2018, 2014, it tended to be associated with high level of severity (Figure 11).



Figure 11. Seriousness of disease indicator in 2019 compared to 2012–2018

From 29 April to 1 September 2019, there were 51,220 acute admissions to ADHB and CMDHB hospitals. A total of 3260 patients with suspected respiratory infections were assessed in these hospitals. Of these, 1456 (44.7%) patients met the SARI case definition. Among these, 1279 were residents of ADHB and CMDHB, giving the SARI incidence rate of 116.6 per 100 000 population (Table 5). Among the 1102 tested SARI cases who were ADHB and CMDHB residents, 353 (32.0%) had positive influenza virus results. This gives a SARI related influenza incidence (adjusted for non-testing) of 37.4 per 100 000 population.

Between 29 April and 1 September 2019, 1456 SARI cases constituted 28.4 per 1000 acute hospitalisations (1456/51220) (Table 4). Of these SARI cases, 37.8% were children aged less than 5 years and 24.9% were adults 65 years and older. Of the 104 acute respiratory illness (ARI) cases admitted to ICU, 16 were positive with influenza virus detection. Of the 34 deaths with acute respiratory illness, 7 had influenza virus infections.

	SARI & inf among all he	luenza cases ospital patients	SARI & influenza cases among ADHB & CMDHB residents					
Characteristics	SARI Cases (%)	Influenza positive ¹ (%)	SARI cases	SARI incidence (per 100 000)	Influenza Cases	Influenza incidence (per 100 000)		
Overall	1456 (44.7)	372 (31.5)	1279	116.6	353	37.4		
Age group (years)								
<1	317 (57.0)	41 (15.8)	290	1994.5	36	300.4		
1-4	234 (74.5)	38 (21.7)	203	377.5	33	83.0		
5–19	94 (64.4)	29 (41.4)	86	41.0	26	17.2		
20–34	103 (60.9)	50 (51.0)	93	30.3	48	16.4		
35–49	101 (56.1)	38 (41.3)	100	47.8	38	19.9		
50–64	159 (42.3)	52 (35.9)	151	84.8	50	30.3		
65–79	213 (39.6)	69 (34.8)	208	214.6	68	75.6		
>80	149 (32.5)	54 (38.8)	148	540.7	54	211.6		
Unknown	86 (16.4)	1 (20.0)	0	0.0	0			
Ethnicity								
Māori	278 (51.5)	50 (22.1)	252	192.8	47	44.4		
Pacific peoples	473 (55.1)	130 (32.7)	456	262.8	128	87.4		
Asian	153 (58.6)	55 (39.9)	145	45.0	53	18.2		
European and Other	466 (43.2)	136 (32.8)	426	90.7	125	29.7		
Unknown	86 (16.4)	1 (20.0)	0		0			
Hospitals								
ADHB	743 (40.9)	200 (33.8)	597	110.8	186	38.7		
СМДНВ	713 (49.3)	172 (29.2)	682	122.3	167	35.8		
Sex								
Female	695 (49.7)	202 (34.2)	653	117.6	192	40.5		
Male	670 (50.5)	169 (29.1)	621	114.7	161	34.3		
Unknown	91 (17.0)	1 (10.0)	5	0.0	0	0.0		

Table 5. Demographic characteristics of SARI cases and related influenza cases, since 29 April2019

¹Proportion of cases tested which were positive for influenza viruses

From 29 April to 1 September 2019, 1271 SARI specimens have been tested and 389 (30.6%) were positive for influenza viruses with more influenza A (279) than influenza B (110) viruses (Table 6). Additionally, 1272 SARI specimens were tested for non-influenza respiratory viruses.

Table 6. Influenza and non-influe	nza respiratory viruse	es among SARI cases,	29 April to 1
September 2019			

Influenza viruses	SARI	SARI and non-SARI		
	Cases (%)	ICU (%)	Deaths (%)	
No. of specimens tested	1271	96	31	
No. of positive specimens (%) ¹	389 (30.6)	19 (19.8)	7 (22.6)	
Influenza A	279	12	3	
A (not subtyped)	133	3	2	
A(H1N1)pdm09	42	1	0	
A(H1N1)pdm09 by PCR	42	1	0	
A/Michigan/45/2015 (H1N1)pdm09 - like	0	0	0	
A(H3N2)	104	8	1	
A(H3N2) by PCR	104	8	1	
A/Switzerland/8060/2017 (H3N2)-like	0	0	0	
Influenza B	110	7	4	
B (lineage not determined)	106	7	4	
B/Yamagata lineage	0	0	0	
B/Yamagata lineage by PCR	0	0	0	
B/Phuket/3073/2013 - like	0	0	0	
B/Victoria lineage	4	0	0	
B/Victoria lineage by PCR	3	0	0	
B/Colorado/06/2017-like	1	0	0	
Influenza and non-influenza co-detection (% +ve)	31 (8.0)	4 (21.1)	1 (14.3)	

Non-influenza respiratory viruses	SARI	SARI and n	on-SARI
	Cases (%)	ICU (%)	Deaths (%)
No. of specimens tested	1272	92	31
No. of positive specimens (%) ¹	547 (43.0)	62 (67.4)	9 (29.0)
Respiratory syncytial virus (RSV)	260	30	2
Parainfluenza 1 (PIV1)	0	0	0
Parainfluenza 2 (PIV2)	10	6	0
Parainfluenza 3 (PIV3)	48	3	0
Rhinovirus (RV)	248	33	7
Adenovirus (AdV)	40	5	0
Human metapneumovirus (hMPV)	48	0	1
Enterovirus	6	0	1
Single virus detection (% of positives)	448 (81.9)	49 (79.0)	0 (-)
Multiple virus detection (% of positives)	99 (18.1)	13 (21.0)	0 (-)

¹Number of specimens positive for at least one of the listed viruses; note a specimen may be positive for more than one virus

The temporal distribution of the number and proportion of the influenza viruses and non-influenza respiratory viruses is shown in Figures 12 & 13.





Figure 13. Temporal distribution of the number and proportion of non-influenza viruses from SARI specimens, by type and week¹



Week (2019) ¹Figures for recent weeks will be underestimates due to time lag in receiving laboratory test results

4.2 MINISTRY OF HEALTH DATA ON PUBLICLY FUNDED HOSPITAL DISCHARGES

Hospitalisation data for influenza (ICD-10AM-VI code I (J09-J11) for 2019 which correlate with previous versions of ICD-10AM codes J10-J11, were extracted from the New Zealand Ministry of Health's NMDS (by discharge date). In this dataset, people who received less than 1 day of hospital treatment in hospital emergency departments were excluded from any time series analysis of influenza hospitalisations during 2000–2019. Influenza-related hospitalisations were conservatively taken to include only those cases where influenza was the principal diagnosis. Repeat admissions were included, as infections with another influenza A subtype or B virus are possible.

From 1 January to 3 September, there were a total of 4093 hospitalisations (83.8 per 100,000) for influenza (Figure 14). Influenza hospitalisation coding has not been completed for the year. This data only captured a proportion of influenza cases for the winter season of 2019.



Figure 14. Influenza hospital discharge rates, 2000–2019*

*2019 data from 1 Jan to 3 Sep only; Source: Ministry of Health, NMDS (Hospital Events)

Figure 15 shows influenza hospitalisations by week discharged. The high number of hospitalisations (360 each) occurred in weeks 26 (week ending 30 June) and 27 (week ending 7 July).



Figure 15. Influenza hospital discharges by week, 2019*

*Data from 1 Jan to 3 Sep only; Source: Ministry of Health, NMDS (Hospital Events)

From 1 January to 3 September, the highest influenza hospitalisation rates were recorded among adults <1 year (375.3 per 100,000) followed by \geq 80 years (442.9 per 100,000) (Figure 16).





*Data from 1 Jan to 3 Sep only; Source: Ministry of Health, NMDS (Hospital Events)

The ethnic distribution of influenza hospitalisations in 2019 is shown in Figure 17. Pacific peoples had the highest hospitalisation rate (205.2 per 100,000, 645 hospitalisations) followed by Maori (108.7 per 100,000 populations, 828). European or Other (68.7 per 100,000, 2168 hospitalisations) and Asian (67.0 per 100,000 populations, 384 hospitalisations) ethnic groups had the lowest rates of hospitalisations.



Figure 17. Hospital discharge rates by prioritised ethnic group, 2019*

*Data from 1 Jan to 3 Sep only; Source: Ministry of Health, NMDS (Hospital Events)

5. VIROLOGY - RECENT STRAIN CHARACTERISATIONS

5.1 CIRCULATING STRAINS IN 2019

A total of 1545 influenza viruses were detected and reported through sentinel ILI and SARI surveillance system in 2019, with influenza A representing 54.4% (840/1545) and influenza B 45.6% (705/1545) of all influenza viruses (Table 7). Influenza B/Victoria lineage and A(H3N2) viruses were two predominant strains co-circulating during the winter. Among the 539 lineage-typed influenza B viruses, all (100%) belonged to Victoria lineage. Among the 648 sub-typed influenza A viruses, 80.2% (520/648) were A(H3N2) viruses and 19.8% (128/648) were A(H1N1)pdm09 viruses.

Table 7. Influenza virus identifications by type and sub-type and lineage-typed, 2019

Viruses	All viruses (%)	Sub-typed and lineage- typed (%)		
Influenza A	840 (54.4)	648		
Influenza A (not sub-typed)	192			
Influenza A(H1N1)pdm09	128	128		
A(H1N1)pdm09 by PCR	106	106		
A/Michigan/45/2015 (H1N1)-like	22	22		
Influenza A(H3N2)	520	520		
A(H3N2) by PCR	484	484		
A/Switzerland/8060/2017 (H3N2)-like	36	36		
Influenza B	705 (45.6)	539		
Influenza B (not lineage-typed)	166			
B/Yamagata lineage	0	0		
B/Yamagata lineage by PCR	0	0		
B/Phuket/3073/2013-like	0	0		
B/Victoria lineage	539	539		
B/Victoria lineage by PCR	274	274		
B/Colorado/06/2017-like	265	265		
Total	1545	1187		

5.2 ANTIGENIC AND GENETIC TYPING

The WHO National Influenza Centre received samples for further typing from active surveillance (sentinel ILI and SARI) as well as passive surveillance (i.e. mainly hospital in-patient and outpatients during routine viral diagnosis).

WHOCC-Melbourne's hemagglutination inhibition (HAI) reference kit for 2018 was used before July 2019 and WHOCC's HAI kit for 2019 was used after July 2019 once it was received.

5.2.1 INFLUENZA A(H1N1)PDM09

Representative of influenza A(H1N1)pdm09 isolates were antigenically typed at the WHO National Influenza Centre at ESR using rabbit antisera supplied by the WHO Collaborating Centre (WHOCC) in Melbourne. Some of these isolates were also sent to WHOCC-Melbourne. During 1 January to 1 September 2019, a total of 48 influenza A(H1N1)pdm09 isolates were antigenically typed using antisera A/Michigan/45/2015/A(H1N1)pdm09 at ESR. All of them were antigenically related to A/Michigan/45/2015/A(H1N1)pdm09. Genetically, the NZ influenza A(H1N1)pdm09 viruses in 2019 fell into genetic subclade 6B.1 (CDC designations, Figure 18)

During the 2019 influenza season, 731 A(H1N1)pdm09 viruses were received at the Melbourne WHOCC from 13 countries with most coming from Australia and New Zealand. All A(H1N1)pdm09 viruses belonged to phylogenetic subclade 6B.1 with substitutions in the HA at N129D and T185I. The heterogeneity of the viral HA genes was reduced with the dominance of the S183-P5 group viruses.

The antigenic characteristics of A(H1N1)pdm09 viruses were assessed with post-infection ferret antisera in haemagglutination inhibition (HI) assay. Most of the viruses tested reacted well with ferret antisera raised against egg and cell-propagated A/Michigan/45/2015 (the H1 component of the 2019 southern hemisphere and 2018/2019 northern hemisphere vaccine). These viruses were also were well inhibited by ferret antisera raised against egg and cell-propagated A/Brisbane/02/2018 (the recommended vaccine component for the 2019-20 Northern Hemisphere vaccine).

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.

(Abridged from the Weekly Epidemiological Record (WER), 2019 94(42):473-496 and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne).

In summary, influenza A(H1N1)pdm09 viruses were circulating in many countries including New Zealand and Australia. The majority of influenza A(H1N1)pdm09 viruses were antigenically indistinguishable from the current vaccine virus A/Michigan/45/2015. However, human serology studies showed reduction of reactivity to many recent representative influenza A(H1N1)pdm09 viruses. Based on all of the available data, the WHO consultation recommended vaccines containing an A/Brisbane/02/2018 (H1N1)pdm09-like strain. The AIVC accepted this recommendation.

5.2.2 INFLUENZA A(H3N2)

Influenza A(H3N2) has frequently been associated with severe disease and excess mortality in highrisk groups. This subtype has also shown the greatest tendency for antigenic drift as illustrated by the frequency of vaccine formulation changes recommended by the WHO and AIVC (Table 1). Representative influenza A(H3N2) isolates were antigenically typed at the WHO National Influenza Centre at ESR using HAI typing kit supplied by the WHO Collaborating Centre (WHOCC) in Melbourne. Some of these isolates were also sent to WHOCC-Melbourne. Antisera against A/Switzerland/8060/2017 (H3N2)-like virus was used after receiving the 2019 HAI typing kit in July 2019 and antisera against A/Singapore/INFIMH-16-0019/2016 was used for typing prior to receipt of the 2019 kit. During 1 January to 1 September 2019, a total of 91 influenza A(H3N2) isolates were antigenically typed at ESR using antisera against A/Singapore/INFIMH-16-0019/2016. 89 (98%, 89/91) were antigenically related to the reference strain A/Singapore/INFIMH-16-0019/2016, 2 (2%, 2/91) had reduced reactivity against the reference vaccine strain. In addition, a total of 49 influenza A(H3N2) isolates were antigenically typed using antisera against A/Switzerland/8060/2017 (H3N2)-like virus. 13 (27%, 13/49) were antigenically related to the reference strain A/Switzerland/8060/2017, and 36 (73%, 36/49) had reduced reactivity against the reference vaccine strain Genetically, the NZ influenza A(H3N2) viruses in 2019 fell into subclade 3C.2a1b (CDC designations, Figure 19)

During the 2019 influenza season, 714 A(H3N2) viruses were received at the Melbourne WHOCC from 13 countries with most coming from Australia and New Zealand. The majority of A(H3N2) viruses belonged to the phylogenetic clade 3C.2a1b with 131K substitution. There was reduced heterogeneity within each group/subgroup.

A(H3N2) viruses have become increasingly difficult to test with the haemagglutination inhibition assay (HI). Some viruses have low or no HA titre with guinea pig RBC even though there is ample virus present (as detected by other methods). Particular mutations or polymorphisms in the NA of recent H3N2 viruses (especially the D151G) appear to allow some level of binding to red blood cells (RBC), thus interfering with the inhibition of viruses between HA and RBC using post-infection ferret sera. To overcome this problem a number of WHOCCs have been performing their HI assays in the presence of 20nM oseltamivir carboxylate in order to prevent this NA binding. This appears to improve the discrimination between antigenically drifted vs not-drifted viruses. However, about 35% of these viruses have a drop in HA titre to a point whereby these viruses cannot be assayed by HI anymore. Alternatively, virus neutralization assays (FRA) can be used where the NA binding is not relevant. Overall, A(H3N2) viruses tested by HAI and FRA were well inhibited by ferret antisera raised against cell culture-propagated A/Switzerland/8060/2017, but were not well inhibited by ferret antisera

Human serology studies, using the serum panels described above, showed that geometric mean HI and virus neutralization titres of antibodies against most recent A(H3N2) viruses were reduced compared to HI and virus neutralization titres against the egg-propagated and cell-propagated vaccine viruses A/Singapore/INFIMH-16- 0019/2016 and A/Switzerland/8060/2017. (*Abridged from the Weekly Epidemiological Record, 2019 94(42):473-496 and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne*).

In summary, influenza A(H3N2) viruses were predominant and associated with outbreaks in several countries. The majority of A(H3N2) viruses fell into the phylogenetic subclades 3C.2a1b. The majority of recent viruses were inhibited well by ferret antisera raised against cell culture-propagated A/Switzerland/8060/2017 viruses but were not well inhibited by ferret antisera raised against egg-propagated A/Switzerland/8060/2017 viruses. In addition, human serology studies showed reduction of reactivity to many recent representative influenza A(H3N2) viruses. Based on all available data, the WHO Consultative Group recommended the H3 component of the vaccines containing an A/South Australia/34/2019-like strain. AIVC accepted this recommendation.

5.2.3 INFLUENZA B

Two distinct lines of influenza B have co-circulated in many countries during recent years. This dates from the late 1980s when the B/Panama/45/90 variant of influenza B was first observed. This strain and its further variants of the Yamagata/16/88 lineage (most recently representative strain-B/Phuket/3073/2013) spread worldwide, whereas strains of the previous B/Victoria/2/87-like viruses

continued to circulate in Asia and subsequently underwent independent evolution as an antigenically distinct lineage (most recent representative strain-B/Colorado/6/2017). For reasons not wholly understood, these remained geographically restricted to Asia until 2001. In 2002 the B/Victoria/2/87 lineage viruses were the predominant viruses worldwide.

Both B/Yamagata-like strains (B/Phuket/3073/2013) and B/Victoria-like strains (B/Colorado/06/2017) continued to be isolated worldwide in 2019 with variable proportions in different regions globally. More B/Victoria than B/Yamagata lineage viruses circulated in New Zealand and Australia in 2019.

Representative influenza B/Yamagata lineage isolates and B/Victoria lineage isolates were antigenically typed at the WHO National Influenza Centre at ESR using HAI typing kit supplied by the WHO Collaborating Centre (WHOCC) in Melbourne. Some of these isolates were also sent to WHOCC-Melbourne and CDC-Atlanta. Antisera against B/Colorado/6/2017 was used after receiving the 2019 HAI typing kit in July 2019 and antisera against B/Brisbane/60/2008 was used for typing prior to receipt of the 2019 kit.

During 1 January to 1 September 2019, a total of 167 B/Victoria lineage isolates were antigenically typed at ESR using antisera against B/Brisbane/60/2008. Of them, 75 (45%, 75/167) were antigenically related to the reference strain B/Brisbane/60/2008, and 92 (55%, 92/167) had reduced reactivity against the reference strain. In addition, a total of 179 B/Victoria lineage isolates were antigenically typed using antisera against B/Colorado/6/2017-like virus. 118 (66%, 118/179) were antigenically related to the reference strain B/Colorado/6/2017, and 61 (34%, 61/179) had reduced reactivity against the reference strain B/Colorado/6/2017. Genetically, the NZ influenza B/Victoria lineage viruses in 2019 fell into genetic clade V1A (CDC designations, Figure 20). During 1 January to 1 September 2019, a total of 2 B/Yamagata lineages isolates were antigenically typed at ESR using antisera against B/Phuket/3073/2013-like virus. These two isolates were antigenically related to the reference 300 and 300 antisera against B/Phuket/3073/2013.

373 influenza B isolates (including 339 B/Victoria and 34 B/Yamagata viruses) were received in 2019 by the Melbourne WHOCC from 13 countries. Sequence analysis of the HA1 gene of the recent B/Victoria lineage viruses showed that they mostly belonged to genetic group V1A. The majority of the B/Victoria viruses had a HA triple deletion (162-164). Only smaller proportion of double deletions (162-163), similar to B/Colorado/6/2017, were detected. In addition, sequence analysis of the HA1 gene of the recent B/Yamagata lineage viruses showed that they belonged to genetic subgroup Y3.

The antigenic characterization of B/Victoria-lineage viruses showed that about 71% of the viruses were well inhibited by ferret antisera raised against cell-propagated B/Colorado/06/2017-like virus, but less well inhibited by ferret antisera against egg-propagated B/Colorado/06/2017-like virus. In addition, the antigenic characterization of B/Yamagata-lineage viruses showed indistinguishable antigenic profiles between circulating viruses and B/Phuket/3073/2013-like reference strain. The majority of recent viruses were well inhibited by ferret sera raised against cell-propagated B/Phuket/3073/2013-like viruses but were poorly inhibited by ferret sera raised against egg propagated B/Phuket/3073/2013-like viruses.

Human serology studies, using the same 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. (*Abridged from the Weekly Epidemiological Record, 2019 94(42):473-496 and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne*).

In summary, influenza B viruses of the B/Victoria/2/87 and B/Yamagata/16/88 lineages cocirculated, with viruses of the B/Victoria lineage predominating in many countries including New Zealand and Australia. The majority of recent B/Yamagata lineage viruses were antigenically and genetically closely related to B/Phuket/3073/2013-like virus. Influenza B viruses of the B/Victoria lineage were detected with increasing proportion of these viruses containing a 3 amino acid deletion in the HA (represented by B/Washington/2/2019-like virus). They were different from those double deletion viruses represented by B/Colorado/6/2017-like vaccine viruses. Human serology studies showed some reduction of reactivity to many recent representative influenza B/Victoria viruses. Based on all available data, the WHO Consultative Group recommended the B/Phuket/3073/2013like virus (B/Yamagata/16/88-lineage) and B/Washington/2/2019-like virus (B/Victoria/2/87-lineage) as quadrivalent vaccine strains. The AIVC accepted this recommendation. In addition, AIVC recommended B/Washington/2/2019-like virus as the B component of the trivalent vaccines.

Figure 18. Phylogenetic relationships among influenza A(H1N) haemagglutinin genes



Figure 19. Phylogenetic relationships among influenza A(H3N2) haemagglutinin genes



Figure 20. Phylogenetic relationships among influenza B (Victoria) haemagglutinin genes



5.3 ANTIVIRAL RESISTANCE

The WHO National Influenza Centre at ESR employed a phenotypic method (fluorometric neuraminidase inhibition assay) for the surveillance of anti-viral drug resistance in influenza viruses. In addition, NIC at ESR employed a molecular method (PCR and sequencing) to monitor the H275Y mutation (histidine-to-tyrosine mutation at the codon of 275 in N1 numbering) which is known to confer resistance to oseltamivir.

In 2019, fluorometric neuraminidase inhibition assay was used to test 45 influenza viruses against oseltamivir and zanamivir. The preliminary results showed that all were sensitive to both oseltamivir and zanamivir (Tables 8 & 9).

Influenza	NA inhibition	No. of Influenza Viruses							
		2014	2015	2016	2017	2018	2019		
A(H1N1)pdm09	Normal	665	12	48	103	75	12		
	Reduced	1	-	-	-	-	-		
	Highly reduced	1	-	-	-	-	-		
A(H3N2)	Normal	164	110	93	254	6	32		
	Reduced	-	-	-	-	-	-		
	Highly reduced	-	-	-	-	-	-		
Influenza B	Normal	167	730	30	548	46	1		
	Reduced	-	-	-	-	1	-		
	Highly reduced	-	-	-	-	-	-		

Table 8. Antiviral susc	eptibility to	o oseltamivir fo	r influenza	viruses,	2014 <mark>-20</mark> 19′
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^ Jan-Aug 2019

Neuraminidase inhibition was defined as:

Normal inhibition = IC_{50} values which are within or close to the median IC_{50} of the type/subtype matched viruses as detailed in the table above.

Reduced inhibition = IC_{50} values which are 10 to 100 fold above the median value of viruses with normal inhibition (5 to 50 fold for influenza B viruses)

Highly reduced inhibition = IC_{50} values which are greater than 100 fold above the median value of viruses with normal inhibition (above 50 fold for influenza B viruses)

Influenze	NA inhibition to Zanamivir	No. of Influenza Viruses						
IIIIuenza		2014	2015	2016	2017	2018	2019	
A(H1N1)pdm09	Normal	671	12	48	125	75	12	
	Reduced	-	-	-	-	-	-	
	Highly reduced	-	-	-	-	-	-	
A(H3N2)	Normal	157	110	93	284	6	32	
	Reduced	-	-	-	-	-	-	
	Highly reduced	-	-	-	-	-	-	
Influenza B	Normal	168	735	30	641	47	1	
	Reduced	-	-	-	-	-	-	
	Highly reduced	-	-	-	-	-	-	

Table 9. Antiviral susceptibility to zanamivir for influenza viruses, 2014–2019^

^ Jan-Aug 2019

Neuraminidase inhibition was defined as:

Normal inhibition = IC50 values which are within or close to the median IC50 of the type/subtype matched viruses as detailed in the table above.

Reduced inhibition = IC50 values which are 10 to 100 fold above the median value of viruses with normal inhibition (5 to 50 fold for influenza B viruses)

Highly reduced inhibition = IC50 values which are greater than 100 fold above the median value of viruses with normal inhibition (above 50 fold for influenza B viruses)

6. INFLUENZA VACCINE EFFECTIVENESS

In New Zealand seasonal trivalent influenza vaccine is offered annually free of charge to all adults aged 65 years and over, pregnant women and all those over six months of age with chronic medical conditions that are likely to increase the severity of the infection. Since 2013, free influenza vaccines have been offered to children (6-months to 4-years) who have been hospitalised or have a history of significant respiratory illness. Influenza vaccines are also available on the private market for all others over six months of age. The influenza season usually occurs between May and September.

Using the case test-negative design to estimate propensity-adjusted VE, we estimated the effectiveness of seasonal trivalent inactivated influenza vaccine in preventing laboratory-confirmed influenza in patients hospitalised with severe acute respiratory infections (SARI) and in patients presenting to general practice with an influenza-like illness (ILI) during the influenza season. The influenza season was defined as starting when there were two consecutive weeks with two or more cases; The data is contributed to I-GIVE project for the WHO vaccine strain selection meeting in September for southern hemisphere countries.

Most ILI and SARI patients with laboratory-confirmed influenza are included except those with incomplete data for vaccination status, infants under 6 months of age, children under 9 years who were only given one dose of vaccine, those vaccinated less than 14 days before admission or presentation. For patients with multiple episodes, the first influenza virus-positive episode was used for the analysis or the first illness episode if there was no influenza virus-positive episode.

The proportion vaccinated did not change throughout the season. For influenza-confirmed SARI cases, after adjustment for age and week of admission, the estimated vaccine effectiveness (VE) was 50% (95% CI: 22 to 68). For influenza-confirmed ILI cases, after adjustment for age and week of consultation, the estimated VE was 25% (95% CI: 3 to 42) (Table 10).

Table 10. Estimated influenza vaccine effectiveness, by participant age group and byinfluenza virus type and subtype in New Zealand, 2019 influenza season

Age &	Influenza Positive		Influenza Negative		crude			age and week adjusted*		
Virus	Vaccinated-	Vaccinated-	Vaccinated-	Vaccinated-						
	Yes	Not	Yes	Not	VE%	LCL	UCL	VE%	LCL	UCL
ILI										
Overall	186	910	197	550	43	28	55	25	3	42
<18y	25	484	19	183	50	2	74	50	4	74
18-64y	115	407	122	343	21	-8	41	24	-3	44
65+y	46	19	56	24	-4	-127	52	29	-69	70
H1	18	66	197	550	24	-34	59	-1	-83	44
<18y	2	24	19	183	20	-268	91	-56	-749	71
18-64y	14	40	122	343	2	-92	52	-7	-109	45
65+y	2	2	56	24	57	-530	97	100	0	100
H3	104	294	197	550	1	-31	26	-2	-40	26
<18y	9	120	19	183	28	-75	72	17	-101	66
18-64y	60	163	122	343	-3	-51	29	-1	-48	31
65+y	35	11	56	24	NA	NA	NA	NA	NA	NA
D	0	45	107	550	4.4	10	70	E 4	24	60
B <191	9	45	197	102	44	-18	76	54	34	08
18-64v	2	29	122	2/2						
10-04y	1	14	56	243						
SARI		2	50	24						
Overall	58	147	148	306	18	-19	44	50	22	68
<18v	1	54	32	186	89	32	100	86	-9	98
18-64v	16	58	47	85	50	0	76	43	-17	72
65+v	41	35	69	35	41	-14	69	52	1	72
H1	8	25	148	306	34	-56	75	60	-2	85
<18v	0	8	32	186	NA	NA	NA	NA	NA	NA
18-64y	2	14	47	85	74	-21	97	80	-1	96
65+y	6	3	69	35	NA	NA	NA	NA	NA	NA
H3	24	49	148	306	-1	-76	43	57	16	79
<18y	0	20	32	186	NA	NA	NA	NA	NA	NA
18-64y	2	12	47	85	70	-45	97	74	-46	95
65+y	22	17	69	35	34	-50	71	54	-14	81
В	6	42	148	306	70	28	90	50	-22	79
<18y	0	21	32	186	NA	NA	NA	NA	NA	NA
18-64y	4	17	47	85	57	-42	90	NA	NA	NA
65+y	2	4	69	35	75	-89	98	NA	NA	NA

*Adjusted for week in season and age

N/A: not applicable as numbers to too low to reach any significance; CI: Confidence interval; ILI: Influenza-like illness; SARI: severe acute respiratory infections. Highlighted cells indicate low numbers

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