

RECOMMENDATION FOR SEASONAL INFLUENZA VACCINE COMPOSITION FOR NEW ZEALAND FOR 2021

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1. RECOMMENDATION

The Australian Influenza Vaccine Committee (AIVC) met with a New Zealand representative in Canberra on 7 October 2020 to consult on the influenza vaccine composition for 2021 for New Zealand, Australia and South Africa (Table 1).

Egg-based quadrivalent influenza vaccines:

- an A/Victoria/2570/2019 (H1N1)pdm09-like virus;
- an A/Hong Kong/2671/2019 (H3N2)-like virus;
- a B/Washington/02/2019-like (B/Victoria lineage) virus; and
- a B/Phuket/3073/2013-like (B/Yamagata lineage) virus.

Cell-based quadrivalent influenza vaccines:

- an A/Wisconsin/588/2019 (H1N1)pdm09-like virus;
- an A/Hong Kong/45/2019 (H3N2)-like virus;
- a B/Washington/02/2019 (B/Victoria lineage)-like virus; and
- a B/Phuket/3073/2013 (B/Yamagata lineage)-like virus.

The composition of trivalent influenza vaccines is recommended to include the H1N1, H3N2 and the B/Victoria lineage virus.

Table 1. Influenza vaccine recommendations for New Zealand, 1991–2021

Decision		Use year	A H3N2	A H1N1	B (Trivalent)	B (Quadrivalent)
NZ & WHO*	2020	2021	A/Hong Kong/2671/2019	A/Victoria/2570/2019	B/Washington/02/2019-like	B/Phuket/3073/2013
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	

* WHO recommendations are for the Southern Hemisphere winter

** WHO recommendations are for the Northern Hemisphere winter

2. SUMMARY

NZ is a southern hemisphere country with a temperate climate. NZ has a well-established influenza circulation pattern with peak incidences in the winter months. The influenza activity in the 2020 winter is unprecedented that no influenza epidemic or outbreak was reported with almost non-existent of influenza virus circulation. This is probably due largely to the COVID-19 related non-pharmaceutical interventions which have been implemented since 25 March 2020.

No influenza-associated severe acute respiratory illness (SARI) was detected in sentinel hospitals. The very low SARI (below the baseline) was mainly associated with rhinovirus infection.

No influenza-associated influenza-like illness (ILI) was detected in sentinel general practices. The very low ILI (below the baseline) was mainly associated with rhinovirus infection.

The laboratory-based influenza surveillance tests samples from various surveillance systems as well as samples ordered by clinicians during routine hospital diagnosis. A total of 500 influenza viruses were detected and reported through this system. Most of viruses (94.8%, 474/500) were reported during Jan-Mar (weeks 1-12) before lockdown, 20 (4%, 20/500) during lockdown and only 6 viruses (1.2%, 6/500) after lockdown (weeks 18-35)

Of those 500 influenza viruses, influenza A represented 91.2% (456/500) and influenza B 8.8% (44/500) of all influenza viruses. Among A sub-typed, 91% (141/155) were A(H1N1)pdm09 virus and 9% (14/155) were A(H3N2) virus. No influenza B viruses were lineage typed

WHO National Influenza Centre (NIC) at ESR only received 5 influenza A(H1N1)pdm09 clinical samples for further characterization. Antigenic typing was conducted using rabbit antisera A/Michigan/45/2015/A(H1N1)pdm09. All of them were antigenically related to A/Michigan/45/2015/A(H1N1)pdm09.

3. EPIDEMIOLOGY - NEW ZEALAND INFLUENZA SEASON IN 2020

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, Ministry of Health data on publicly funded hospital discharges, laboratory-based surveillance for outpatients and hospital in-patients).

3.1 COMMUNITY-BASED SURVEILLANCE

3.1.1 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 population-based surveillance for influenza-like illness (ILI) among persons enrolled in sentinel general practices (~90) who seek medical consultations. It is operated nationally by ESR and locally by influenza surveillance co-ordinators in the public health services (PHS). Each year, approximately 90 sentinel general practices participate in this surveillance, covering nationally about 10% of the New Zealand population. 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 $\geq 38^{\circ}\text{C}$, 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 characterization. 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 (<http://apps.who.int/iris/bitstream/handle/10665/259392/WHO-WHE-IHM-GIP-2017.2-eng.pdf;jsessionid=0D8C59A6D4E84A8C5AB9329CADAE374D?sequence=1>)

In 2020, the GP-based ILI surveillance has not been operated in a usual way. In response to the COVID-19 pandemic, NZ government set up a number of community-based assessment centres (CBACs) around the country to provide safe places to get patient swabs for COVID-19 testing. The usual patient consultation flow and process established for sentinel GP-based ILI surveillance was interrupted as some patients were diverted to CBACs for COVID-19 testing, instead of visiting their GPs for ILI. This would result in lower consultation and reporting.

In 2020, all 80 sentinel practices with a total patient roll of 537137 from all 20 DHBs were invited to participate in the surveillance. Influenza like illness (ILI) consultation rates in 2020 were extremely low and no influenza case was detected (Figures 1-3)

Figure 1. Weekly consultations for influenza-like illness in New Zealand, 2020

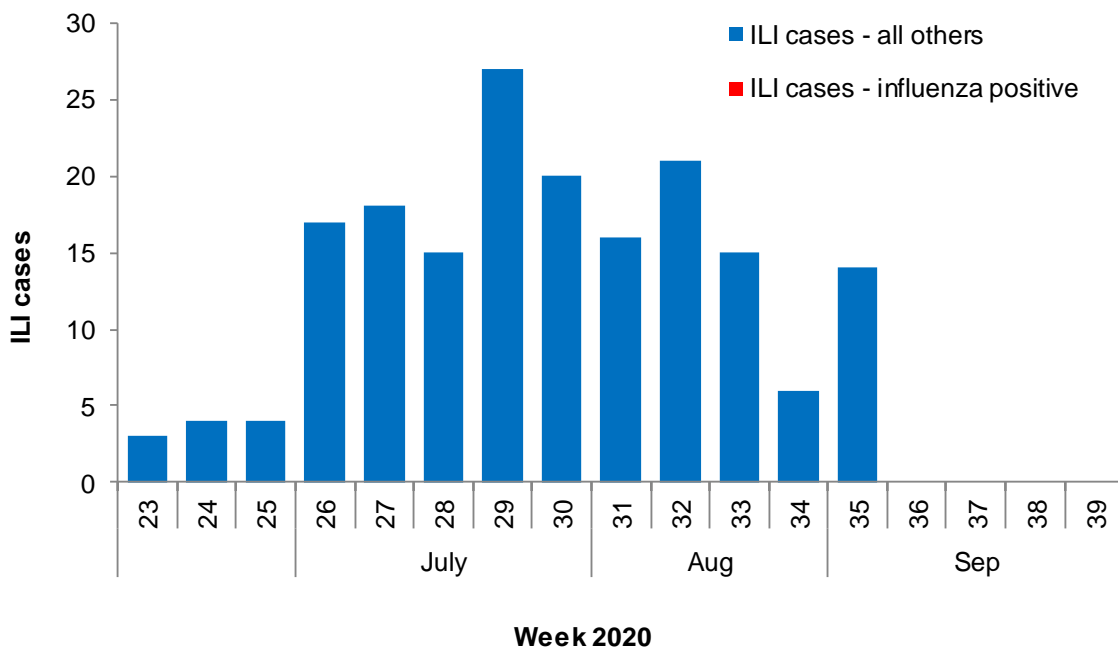


Figure 2. Weekly ILI consultation rates in 2020 compared to 2013–2019

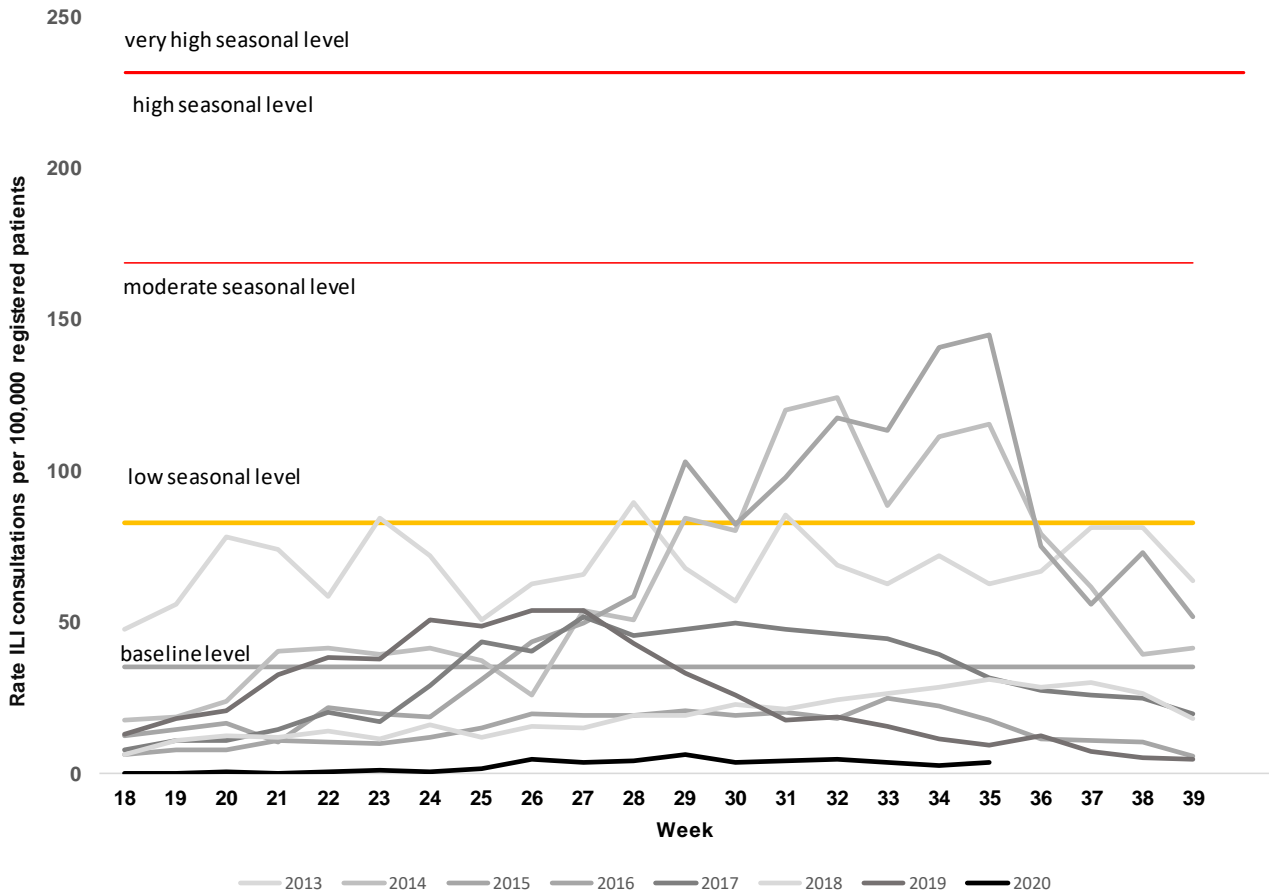
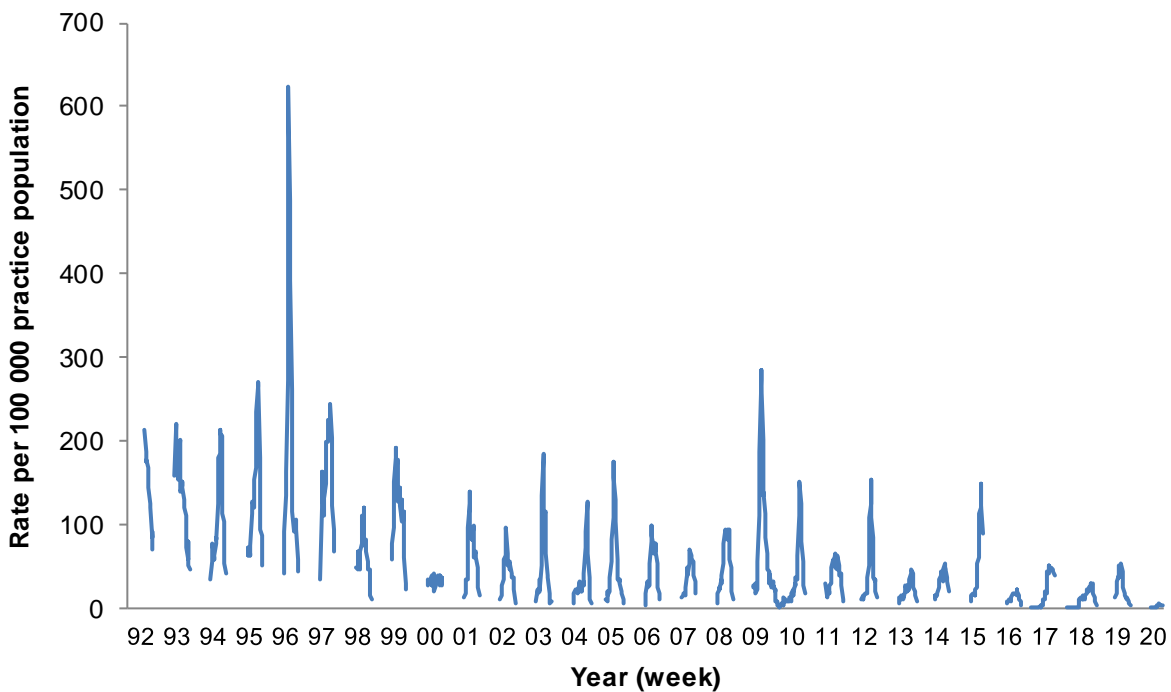


Figure 3. Weekly Consultation Rates for Influenza-like Illness in New Zealand, 1992–2020



From week 18 (commencing 27 April 2020) to week 35 (ending 30 August 2020), a total of 205 consultations for ILI were reported from the 20 DHBs. The cumulative incidence of ILI consultation during this period was 46.5 per 100,000 population. The average weekly ILI consultation rate between weeks 18 and 35 was 2.3 per 100,000 population. Among the patients that met the ILI case definition, 166 (81.0%) had a specimen tested for influenza. Of these, none (0.0%) cases had influenza viruses detected and the cumulative influenza incidence during 27 April to 30 August 2020 was 0.0 per 100,000 population (Table 1).

Table 1. Demographic characteristics of ILI and influenza cases, 27 April – 30 August 2020

Characteristics	ILI & influenza cases among sentinel practices				
	ILI cases	Influenza cases	Prop Influenza positive ¹ (%)	ILI incidence (per 100 000)	Influenza incidence ² (per 100 000)
Overall	205	0	0.0 (-)	46.5	0.0
Age group (years)					
<1	1	0	(-)	22.0	
1–4	2	0	0.0 (-)	8.3	0.0
5–19	31	0	0.0 (-)	35.1	0.0
20–34	46	0	0.0 (-)	51.0	0.0
35–49	30	0	0.0 (-)	35.6	0.0
50–64	68	0	0.0 (-)	82.4	0.0
65–79	21	0	0.0 (-)	40.5	0.0
>80	6	0	0.0 (-)	40.2	0.0
Unknown	0	0	0.0		
Ethnicity					
Māori	52	0	0.0 (-)	86.7	0.0
Pacific peoples	11	0	0.0 (-)	40.2	0.0
Asian	12	0	0.0 (-)	31.3	0.0
European and Other	130	0	0.0 (-)	41.3	0.0
Unknown	0	0	0.0		
Sex					
Female	129	0	0.0 (-)	55.8	0.0
Male	76	0	0.0 (-)	36.3	0.0
Unknown	0	0	0.0		

¹Proportion of cases tested which were positive for influenza viruses

²Adjusted to positivity of tested cases

Between 27 April to 30 August 2020, a total of 166 ILI specimens were tested for influenza viruses (Table 2) and none (0.0%) were positive. Additionally, a total of 166 ILI specimens were tested for non-influenza viruses and 76 (45.8%) were positive with non-influenza viruses.

Table 2. Influenza and non-influenza respiratory viruses among ILI cases, 27 April – 30 August 2020

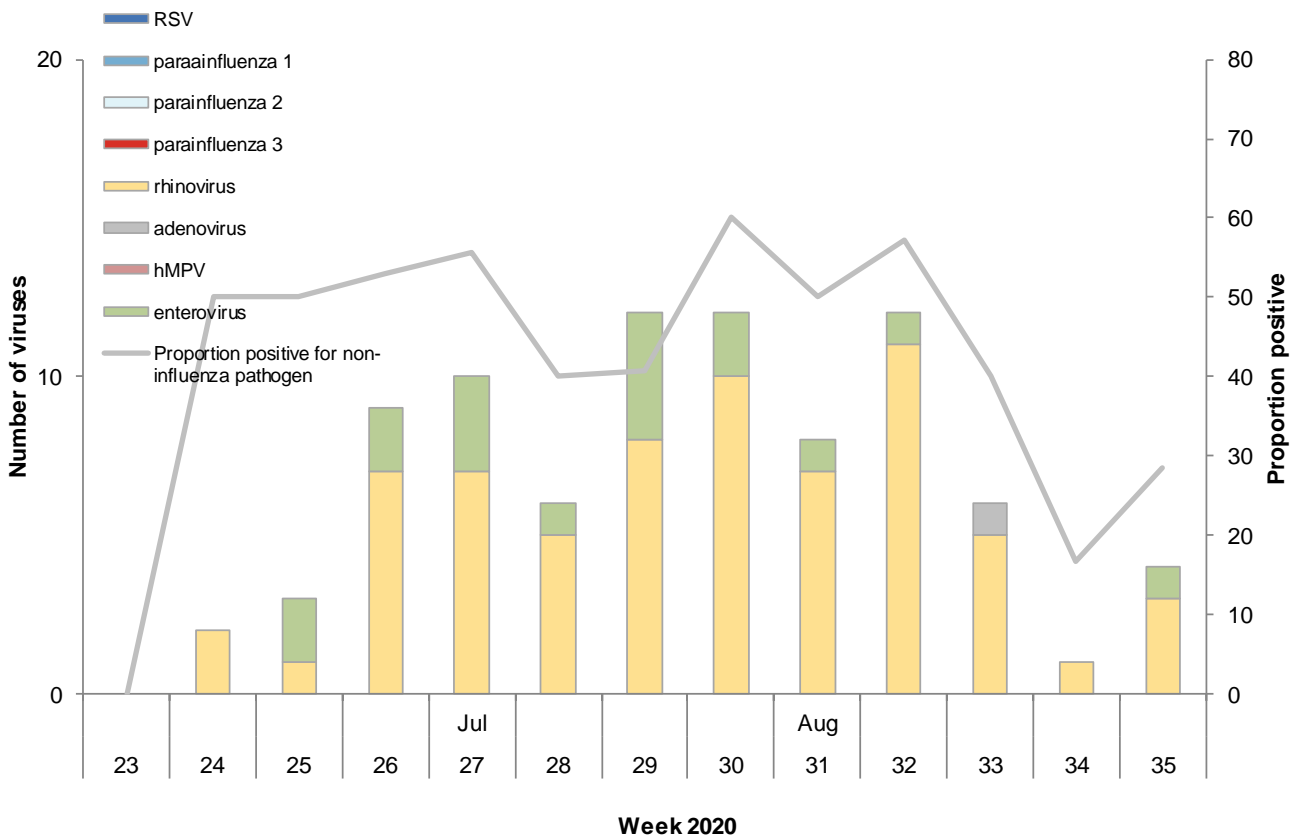
<i>Influenza viruses</i>	ILI
	Cases (%)
No. of specimens tested	166
No. of positive specimens (%) ¹	0 (0.0)
Influenza A	0
A (not subtyped)	0
A(H1N1)pdm09	0
A(H1N1)pdm09 by PCR	0
A/Brisbane/02/2018 (H1N1)pdm09 - like	0
A(H3N2)	0
A(H3N2) by PCR	0
A/South Australia/34/2019 (H3N2)-like	0
Influenza B	0
B (lineage not determined)	0
B/Yamagata lineage	0
B/Yamagata lineage by PCR	0
B/Phuket/3073/2013 - like	0
B/Victoria lineage	0
B/Victoria lineage by PCR	0
B/Washington/02/2019-like	0
Influenza and non-influenza co-detection (% +ve)	0 (-)

<i>Non-influenza respiratory viruses</i>	ILI
	Cases (%)
No. of specimens tested	166
No. of positive specimens (%) ¹	76 (45.8)
Respiratory syncytial virus (RSV)	0
Parainfluenza 1 (PIV1)	0
Parainfluenza 2 (PIV2)	0
Parainfluenza 3 (PIV3)	0
Rhinovirus (RV)	61
Adenovirus (AdV)	1
Human metapneumovirus (hMPV)	0
Enterovirus	16
SARS-Cov-2	0
Single virus detection (% of positives)	74 (97.4)
Multiple virus detection (% of positives)	2 (2.6)

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 non-influenza respiratory viruses, from 27 April to 30 August 2020, is shown in Figure 4.

Figure 4. Temporal distribution of the number and proportion of non-influenza viruses from ILI specimens, by type and week¹



3.1.2 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).

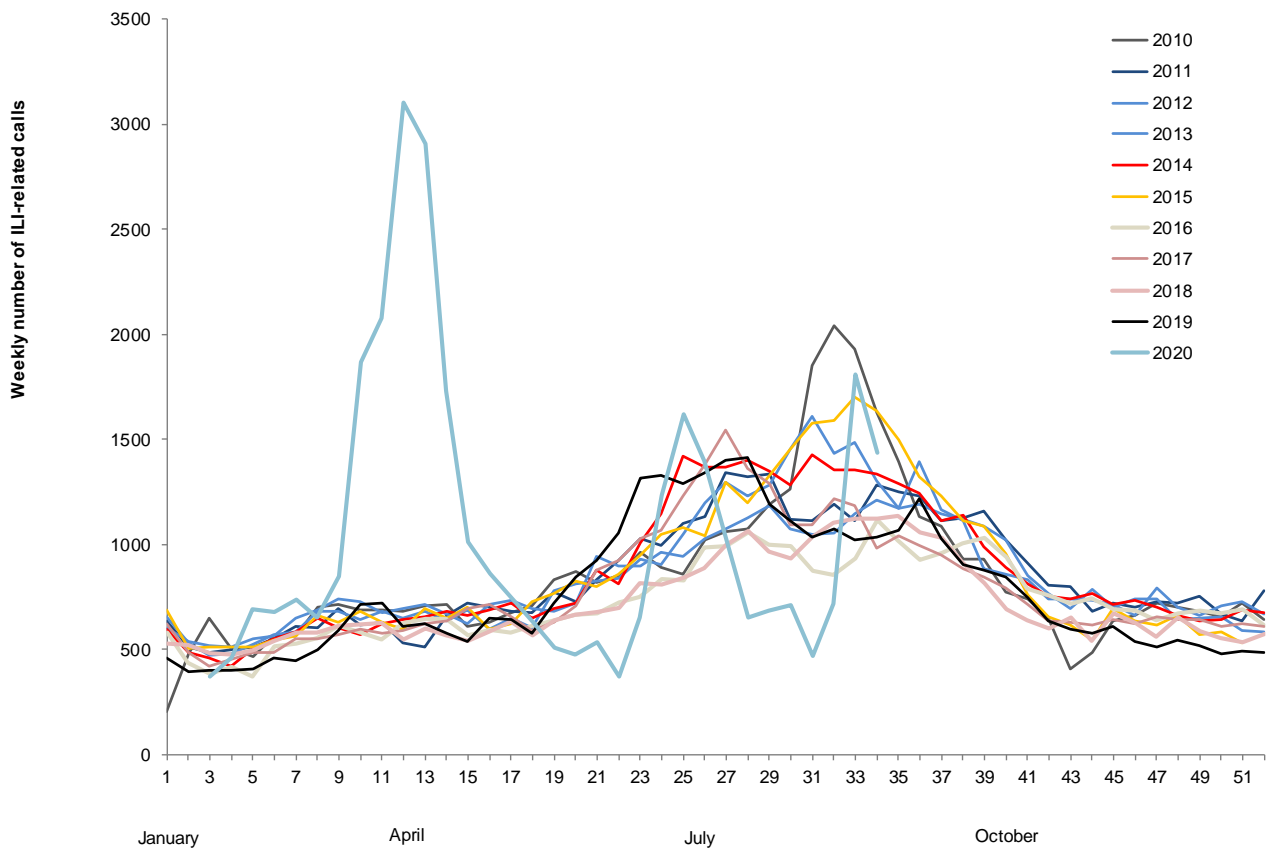
Analysis is frequency based with alarms raised by identifying statistical deviations (aberrations) from previous calls. Data are reported for all ages and in five age bands (0–4, 5–14, 15–44, 45–64, 65+ years). The analysis of the call frequency is based on the cumulative summation (CUSUM) algorithm 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 5 shows the weekly number of calls to Healthline for ILI during 2010–2020. Healthline calls in 2020 were entirely different from previous years, probably due to the COVID-19 responses.

Figure 5. Weekly number of ILI-related calls to Healthline, 2010–2020



3.2 Hospital-based surveillance

3.2.1 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 $\geq 38^{\circ}\text{C}$, 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 2020. From week 18 (commencing 27 April 2020) through week 35 (ending 30 August 2020), Severe acute respiratory illness (SARI) hospitalization rates were at a very low level and never surpassed the baseline (Figures 6 and 7). During weeks 18-35, no SARI-associated influenza hospitalization cases have been reported (Figure 8).

Figure 6. Weekly resident SARI and SARI-associated influenza incidence, 2020

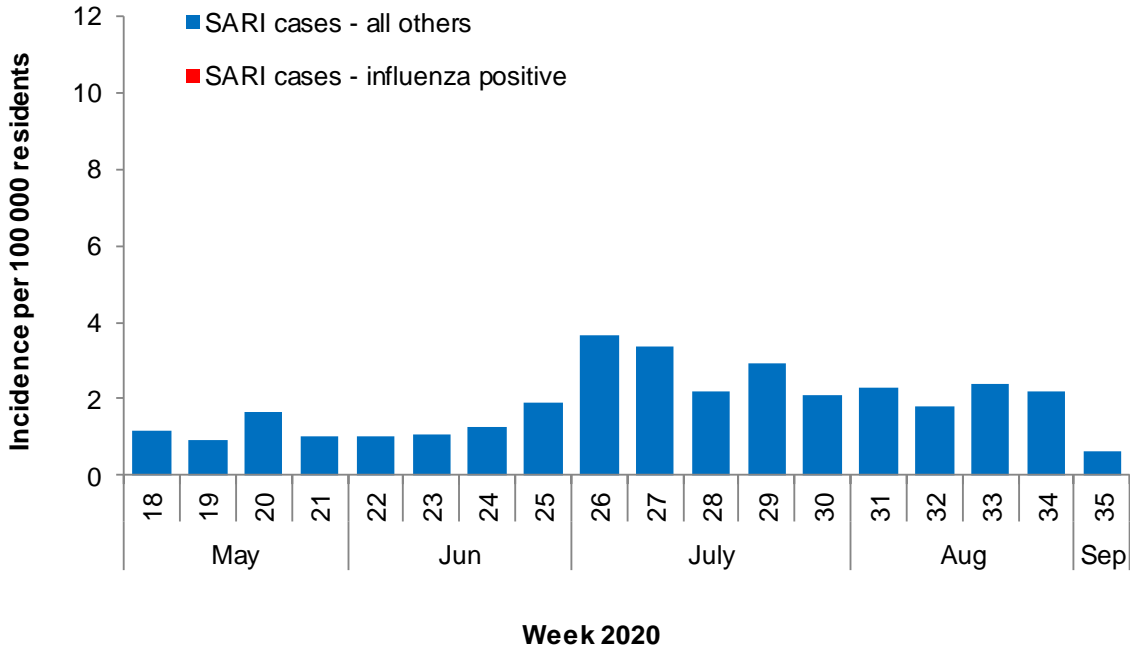


Figure 7. Weekly hospitalisation rates for SARI in 2020 compared to 2012–2019

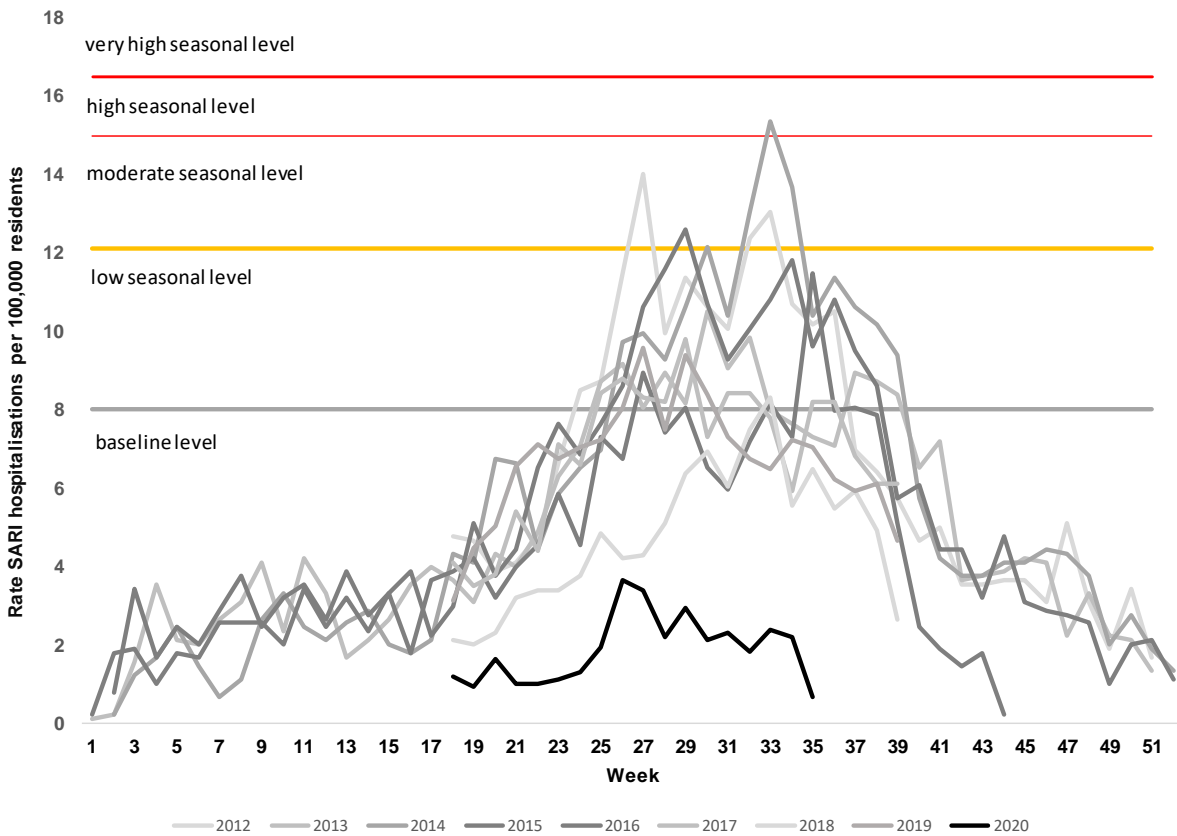
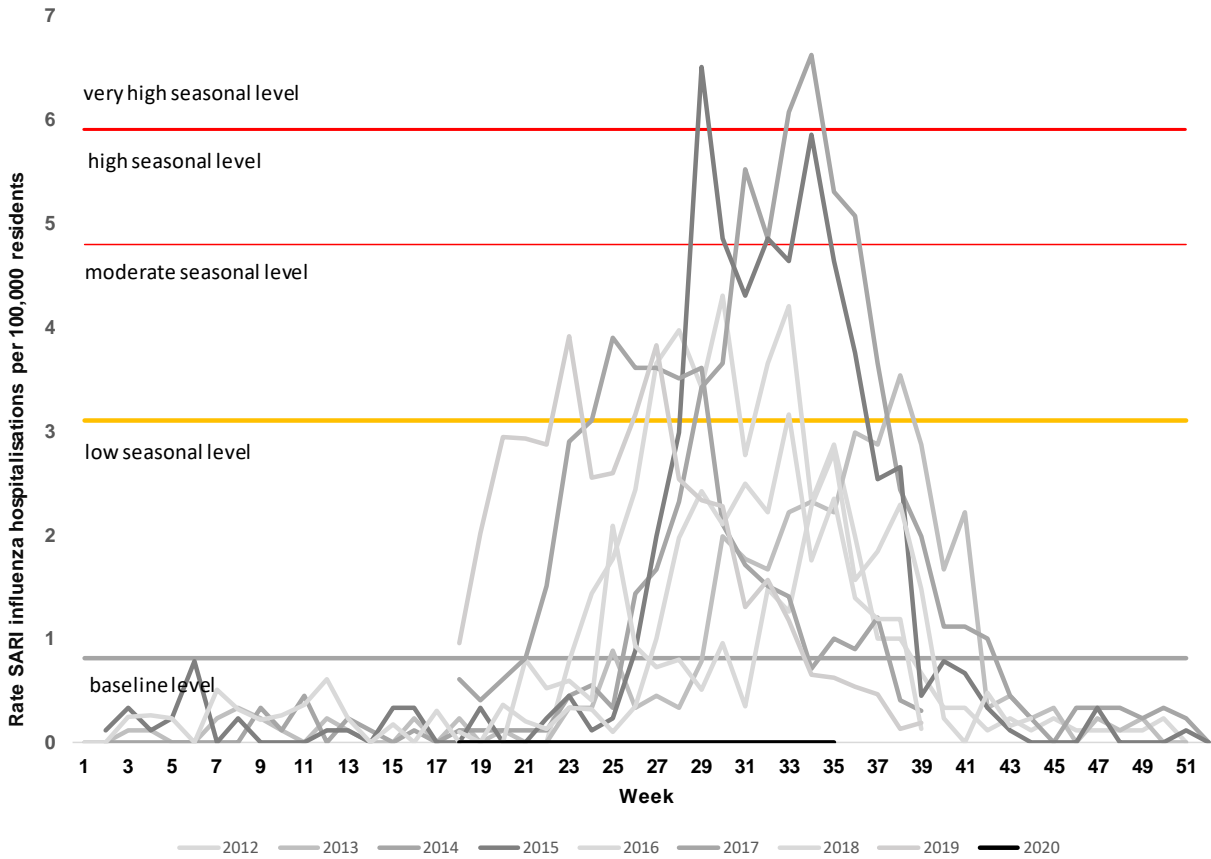


Figure 8. Weekly hospitalisation rates for SARI-associated influenza in 2020 compared to 2012–2019



From 27 April to 30 August 2020, there were 48,404 acute admissions to ADHB and CMDHB hospitals. A total of 1355 patients with suspected respiratory infections were assessed in these hospitals. Of these, 383 (28.3%) patients met the SARI case definition. Among these, 368 were residents of ADHB and CMDHB, giving the SARI incidence rate of 33.6 per 100 000 population (116.6 per 100,000 in 2019) (Table 4). Among the 304 tested SARI cases who were ADHB and CMDHB residents, none (0.0%) had positive influenza virus results. This gives a SARI related influenza incidence (adjusted for non-testing) of 0.0 per 100 000 population (Note: it was 37.4 per 100,000 in 2019).

Between 27 April and 30 August 2020, 383 SARI cases constituted 7.9 per 1000 acute hospitalisations (383/48,404) (Table 3). Of these SARI cases, 16.2% were children aged less than 5 years and 29.8% were adults 65 years and older. Of the 31 acute respiratory illness cases that were admitted to ICU, none were positive with influenza virus detection. Of the 35 deaths with acute respiratory illness, none had influenza virus infections.

Table 3. Demographic characteristics of SARI cases and related influenza cases, since 27 April 2020

Characteristics	SARI & influenza cases among all hospital patients		SARI & influenza cases among ADHB & CMDHB residents			
	SARI Cases (%)	Influenza positive ¹ (%)	SARI cases	SARI incidence (per 100 000)	Influenza Cases	Influenza incidence (per 100 000)
Overall	383 (28.3)	0 (0.0)	368	33.6	0	0.0
Age group (years)						
<1	21 (25.0)	0 (0.0)	21	144.4	0	0.0
1–4	41 (41.4)	0 (0.0)	36	66.9	0	0.0
5–19	11 (18.0)	0 (0.0)	10	4.8	0	0.0
20–34	36 (38.3)	0 (0.0)	35	11.4	0	0.0
35–49	51 (36.2)	0 (0.0)	47	22.4	0	0.0
50–64	86 (30.8)	0 (0.0)	82	46.0	0	0.0
65–79	73 (23.3)	0 (0.0)	73	75.3	0	0.0
>80	41 (19.6)	0 (0.0)	41	149.8	0	0.0
Unknown	23 (30.7)	0 ()	23	0.0	0	
Ethnicity						
Māori	97 (31.5)	0 (0.0)	90	68.8	0	0.0
Pacific peoples	124 (36.5)	0 (0.0)	122	70.3	0	0.0
Asian	28 (23.9)	0 (0.0)	28	8.7	0	0.0
European and Other	111 (21.6)	0 (0.0)	105	22.3	0	0.0
Unknown	23 (30.7)	0 ()	23		0	
Hospitals						
ADHB	131 (19.8)	0 (0.0)	119	22.1	0	0.0
CMDHB	252 (36.3)	0 (0.0)	249	44.6	0	0.0
Sex						
Female	171 (27.5)	0 (0.0)	161	29.0	0	0.0
Male	189 (28.7)	0 (0.0)	184	34.0	0	0.0
Unknown	23 (30.7)	0 ()	23	0.0	0	0.0

¹Proportion of cases tested which were positive for influenza viruses

From 27 April to 30 August 2020, 367 SARI specimens have been tested and none (0.0%) were positive for influenza viruses (Table 4). Additionally, 386 SARI specimens were tested for non-influenza respiratory viruses.

Table 4. Influenza and non-influenza respiratory viruses among SARI cases, 27 April to 30 August 2020

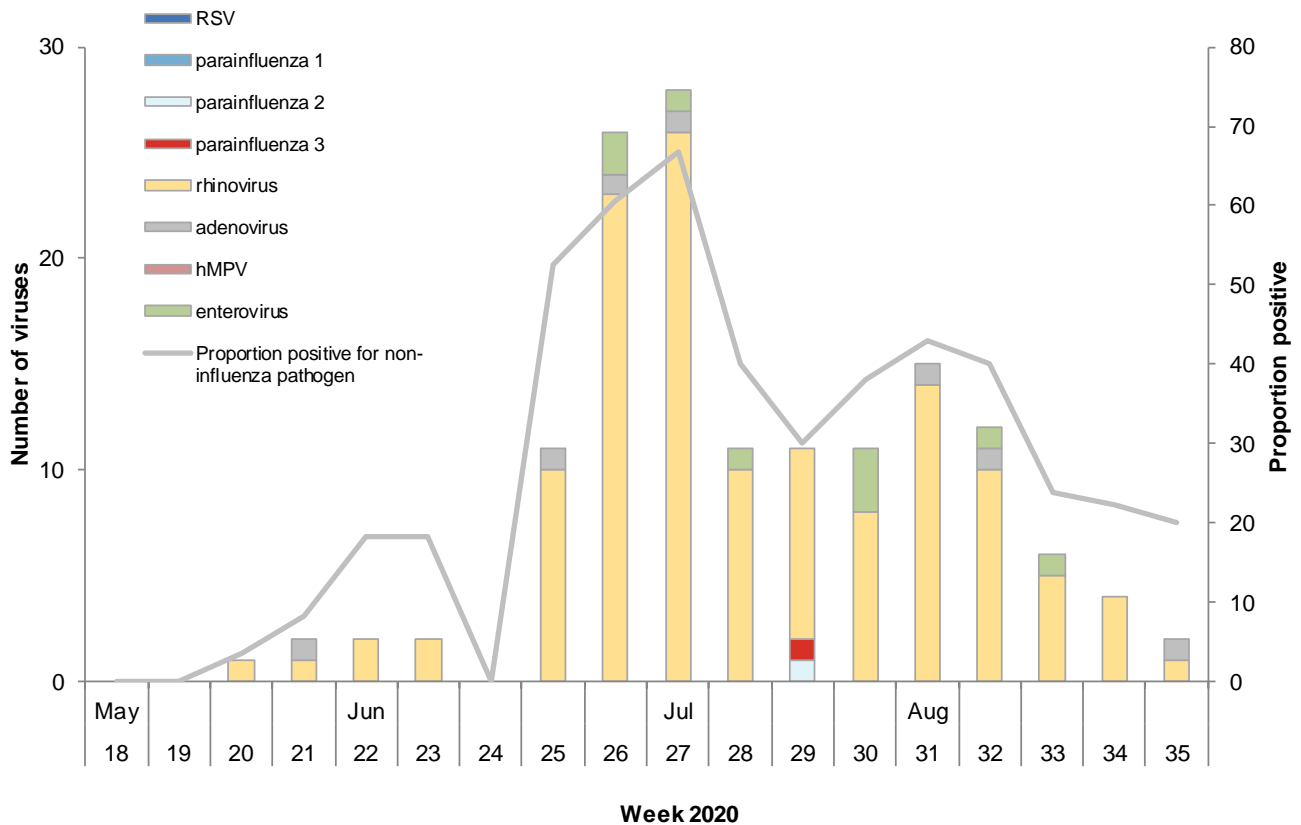
<i>Influenza viruses</i>	SARI	SARI and non-SARI	
	Cases (%)	ICU (%)	Deaths (%)
No. of specimens tested	367	32	33
No. of positive specimens (%) ¹	0 (0.0)	0 (0.0)	0 (0.0)
Influenza A	0	0	0
A (not subtyped)	0	0	0
A(H1N1)pdm09	0	0	0
A(H1N1)pdm09 by PCR	0	0	0
A/Brisbane/02/2018 (H1N1)pdm09 - like	0	0	0
A(H3N2)	0	0	0
A(H3N2) by PCR	0	0	0
A/South Australia/34/2019 (H3N2)-like	0	0	0
Influenza B	0	0	0
B (lineage not determined)	0	0	0
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	0	0	0
B/Victoria lineage by PCR	0	0	0
B/Washington/02/2019-like	0	0	0
Influenza and non-influenza co-detection (% +ve)	0 (-)	0 (-)	0 (-)

<i>Non-influenza respiratory viruses</i>	SARI	SARI and non-SARI	
	Cases (%)	ICU (%)	Deaths (%)
No. of specimens tested	386	36	38
No. of positive specimens (%) ¹	129 (33.4)	13 (36.1)	2 (5.3)
Respiratory syncytial virus (RSV)	0	0	0
Parainfluenza 1 (PIV1)	0	0	0
Parainfluenza 2 (PIV2)	1	0	0
Parainfluenza 3 (PIV3)	1	0	0
Rhinovirus (RV)	128	13	1
Adenovirus (AdV)	7	1	1
Human metapneumovirus (hMPV)	0	0	0
Enterovirus	9	2	0
SARS-Cov-2	0	0	0
Single virus detection (% of positives)	113 (87.6)	10 (76.9)	2 (100.0)
Multiple virus detection (% of positives)	16 (12.4)	3 (23.1)	0 (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 non-influenza respiratory viruses is shown in Figure 9.

Figure 9. Temporal distribution of the number and proportion of non-influenza viruses from SARI specimens, by type and week¹



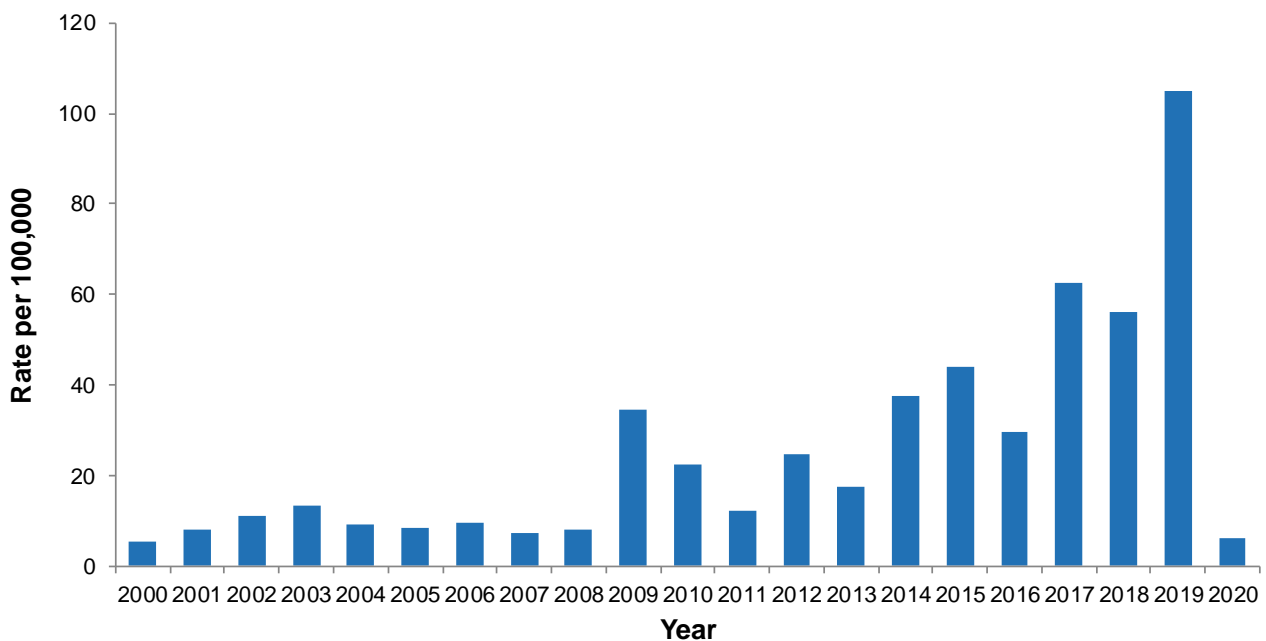
¹Figures for recent weeks will be underestimates due to time lag in receiving laboratory test results

3.2.2 MINISTRY OF HEALTH DATA ON PUBLICLY FUNDED HOSPITAL DISCHARGES

Hospitalisation data for influenza (ICD-10AM-VI code I (J09-J11) for 2020 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 2000–2020. 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 29 July, there were a total of 291 hospitalisations (6.0 per 100,000) for influenza (Figure 10). Influenza hospitalisation coding has not been completed for the year. This data only captured a proportion of influenza-coded cases for the winter season of 2020.

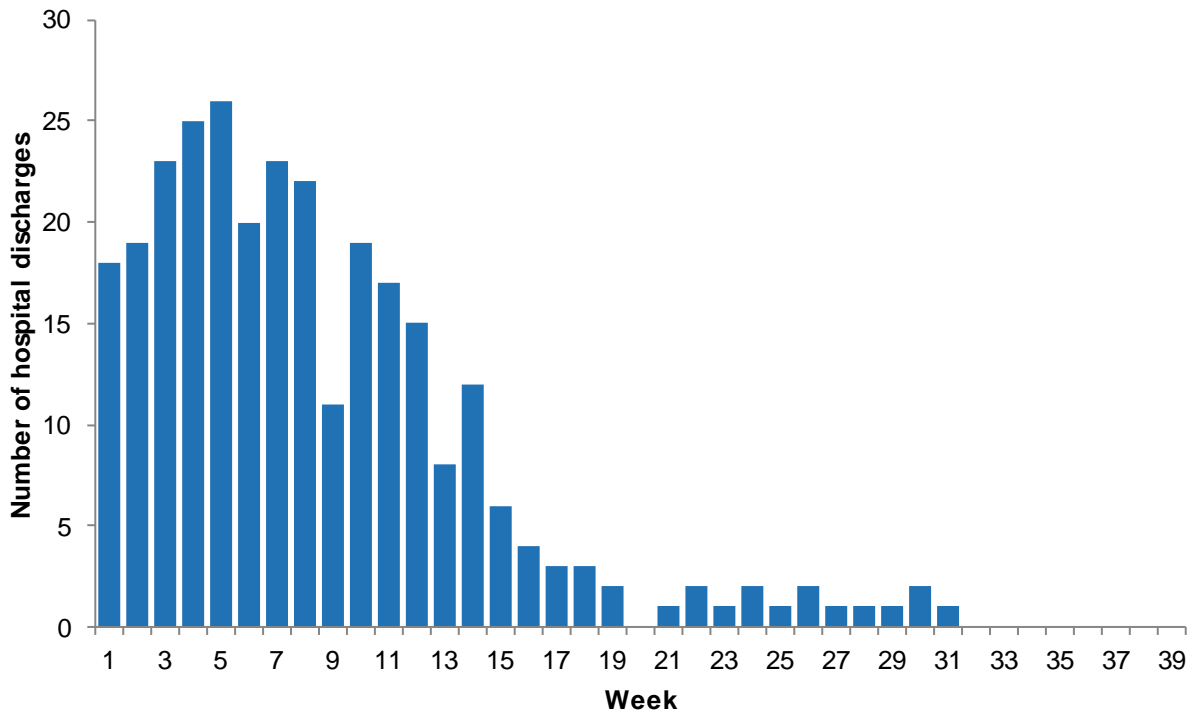
Figure 10. Influenza hospital discharge rates, 2000–2020*



*2020 data from 1 Jan to 29 July only; Source: Ministry of Health, NMDS (Hospital Events)

Figure 11 shows influenza hospitalisations by week discharged. The highest number of hospitalisations (25) occurred in week 5 (week ending 2 February).

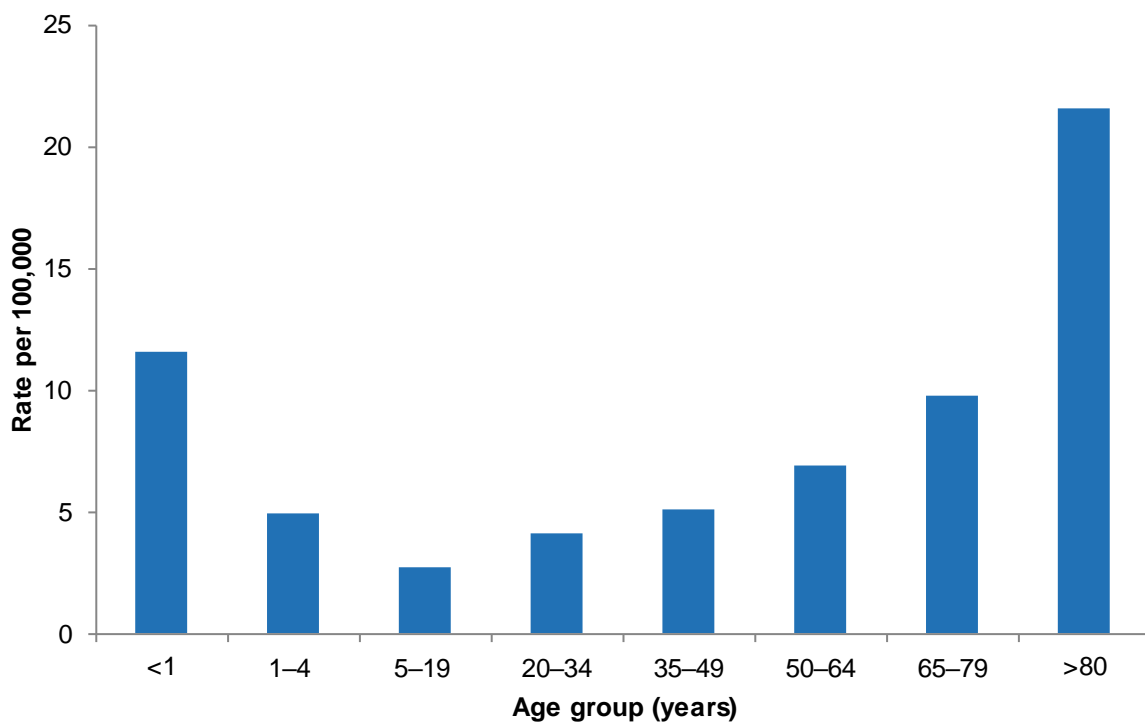
Figure 11. Influenza hospital discharges by week, 2020*



*Data from 1 Jan to 29 July only; Source: Ministry of Health, NMDS (Hospital Events)

From 1 January to 29 July, the highest influenza hospitalisation rates were recorded among adults >80 years (21.6 per 100,000) followed by infants \leq year (11.6 per 100,000) (Figure 12).

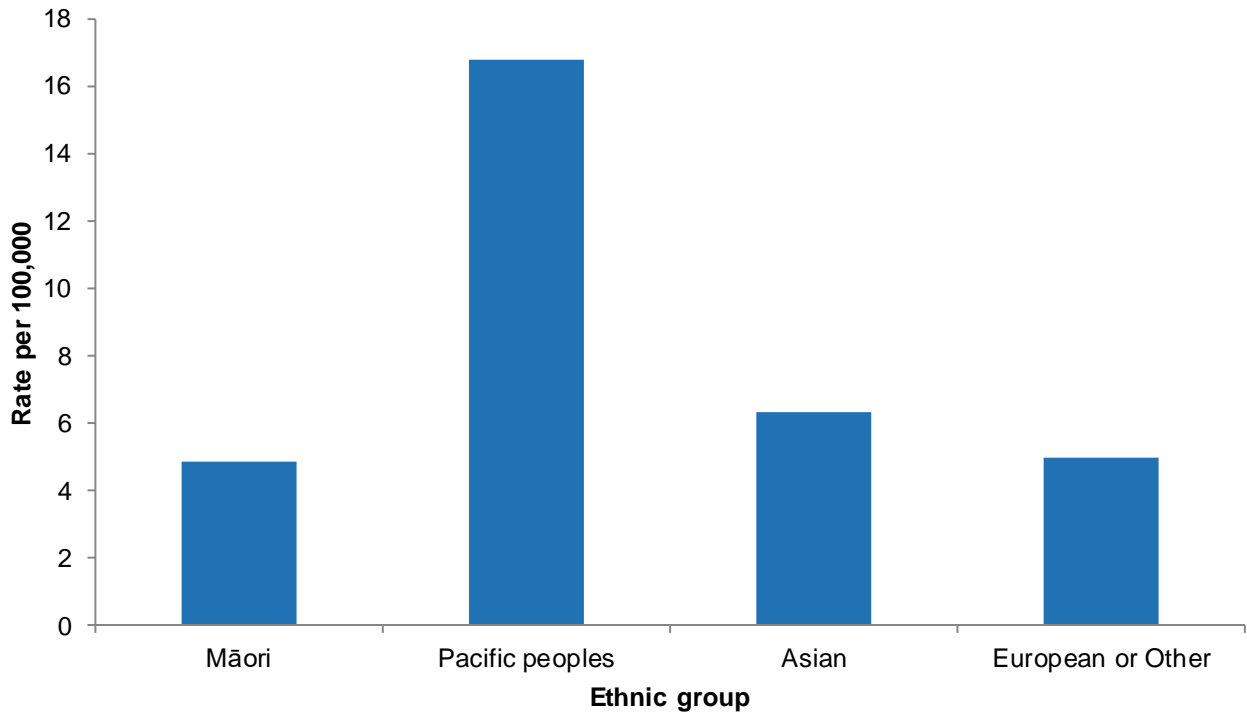
Figure 12. Influenza hospital discharge rates by age group, 2020*



*Data from 1 Jan to 29 July only; Source: Ministry of Health, NMDS (Hospital Events)

The ethnic distribution of influenza hospitalisations in 2020 is shown in Figure 13. Pacific peoples had the highest hospitalisation rate (16.8 per 100,000, 53 hospitalisations) followed by Asian (6.3 per 100,000 populations, 46). European or Other (4.9 per 100,000, 152 hospitalisations) and Maori (4.8 per 100,000 populations, 37 hospitalisations) ethnic groups+ had the lowest rates of hospitalisations.

Figure 13. Hospital discharge rates by prioritised ethnic group, 2020*



*Data from 1 Jan to 29 July only; Source: Ministry of Health, NMDS (Hospital Events)

4. RECENT STRAIN CHARACTERISATIONS

The laboratory-based surveillance for influenza is carried out all-year-around by the New Zealand virus laboratory network consisting of the WHO National Influenza Centre (NIC) at ESR and 6 hospital laboratories at Auckland, Waikato, Wellington, Christchurch and Dunedin, serving nearly 70% of the NZ population. This laboratory network tests specimens ordered by clinicians for hospital in-patient and outpatients during routine viral diagnosis. In addition, this laboratory network conducts testing for public health surveillance including hospital based SARI and sentinel GP-based ILI surveillance.

The WHO National Influenza Centre at ESR receives samples from local hospital laboratories 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).

4.1 CIRCULATING STRAINS IN 2020

A total of 500 influenza viruses were detected and reported through any surveillance system in 2020. Of them, influenza A represented 91.2% (456/500) and influenza B 8.8% (44/500) of all influenza viruses (Table 5). Among A sub-typed, 91% (141/155) were A(H1N1)pdm09 virus and 9% (14/155) were A(H3N2) virus. No influenza B were lineage typed.

Table 5. Influenza virus identifications by type and sub-type and lineage-typed, 2020

Viruses	All viruses (%)	Sub-typed and lineage-typed (%)
Influenza A	456 (91.2%)	155
Influenza A (not sub-typed)	301	
Influenza A(H1N1)pdm09	141	141 (91%)
A(H1N1)pdm09 by PCR	136	136
A/Michigan/45/2015 (H1N1)pdm09-like	5	5
Influenza A(H3N2)	14	14 (9%)
A(H3N2) by PCR	14	14
A/Switzerland/8060/2017 (H3N2)- like	0	0
Influenza B	44 (8.8%)	2
Influenza B (not lineage-typed)	44	
B/Yamagata lineage	0	0
B/Yamagata lineage by PCR	0	0
B/Phuket/3073/2013-like	0	
B/Victoria lineage	0	0
B/Colorado/6/2017-like	0	
B/Victoria lineage by PCR	0	
Total	500	155

Figure 14 shows the influenza virus identifications by type and sub-type for each week throughout 2020. NZ is a southern hemisphere country with a temperate climate. NZ has a well-established influenza circulation pattern with peak incidences in the winter months. The 2020 winter is unprecedented that no influenza epidemic or outbreak was reported during the winter influenza surveillance period. This is probably due largely to the COVID-19 related non-pharmaceutical interventions which have been implemented since 25 March 2020.

A total of 500 influenza viruses were detected and reported through laboratory-based surveillance. Most of viruses (94.8%, 474/500) were reported during Jan-Mar (weeks 1-12) before lockdown, 20 (4%, 20/500) during lockdown and only 6 viruses (1.2%, 6/500) after lockdown (weeks 18-35)

Figure 14. Total influenza viruses by type and week specimen taken, 2020

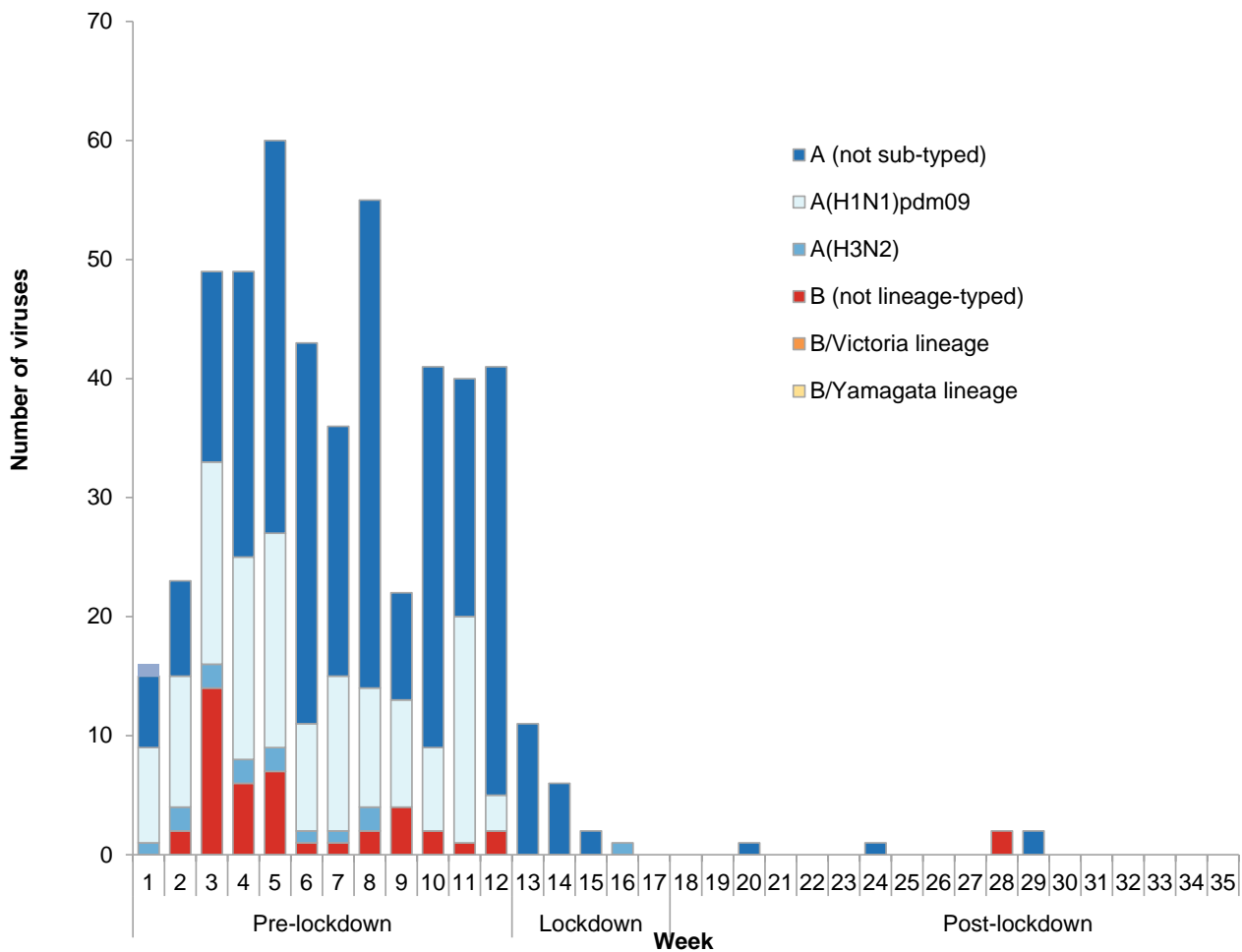


Figure 15 shows the number and percentage of typed influenza viruses from 1997 to 2020. Influenza A is the most frequent predominant influenza type. Of 24 influenza seasons during 1997–2020, influenza A predominated in 23 seasons whereas influenza B only predominated in three seasons (2005, 2008 and 2015). There was one season (1997) with equal proportion of influenza A and B circulation.

Figure 15. Influenza viruses by type, 1997–2020

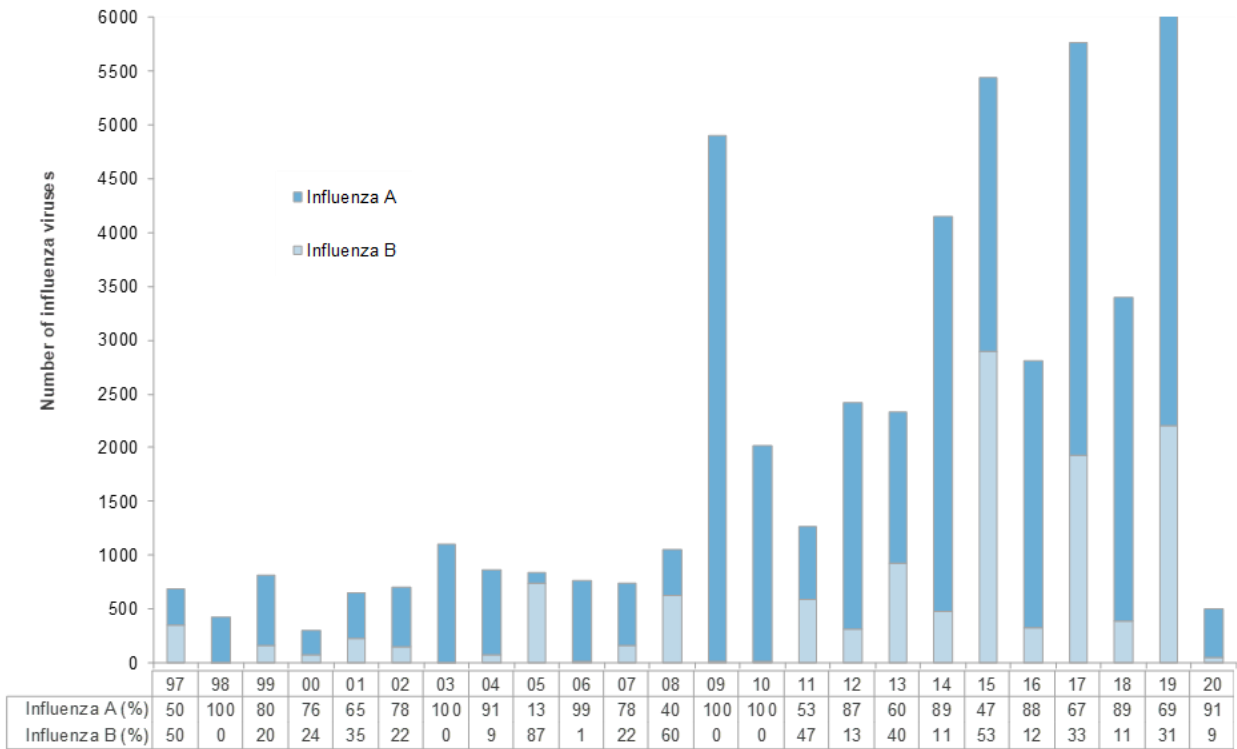


Figure 16 shows the number and percentage of all sub-typed influenza A viruses from 1997 to 2020 (excluding influenza A not sub-typed). Overall, the patterns of the predominant influenza A subtypes among all sub-typed A viruses during 1997–2020 are described below:

- Influenza A(H3N2) strain predominated for 17 seasons (1997–1999, 2002–2008, 2011–2013, 2015–2017, 2019).
- Influenza A(H1N1)pdm09 strain has become the predominant strain for four seasons in 2009, 2010, 2014, 2018 and 2020.
- Seasonal influenza A(H1N1) strain predominated in two seasons (2000 and 2001) with associated relatively low hospitalisations (228 in 2000 and 379 in 2001). It has not been detected in New Zealand since 2010.

Figure 16. Influenza A viruses by subtypes 1997–2020

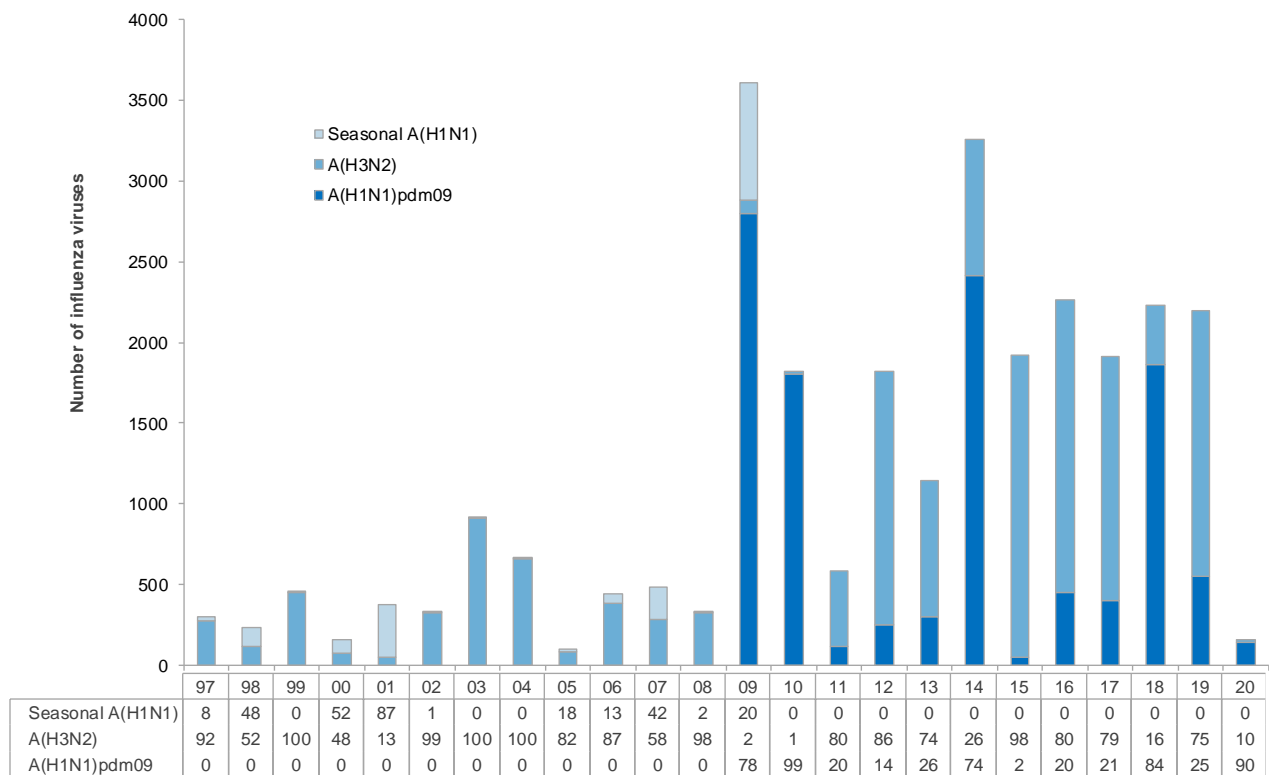
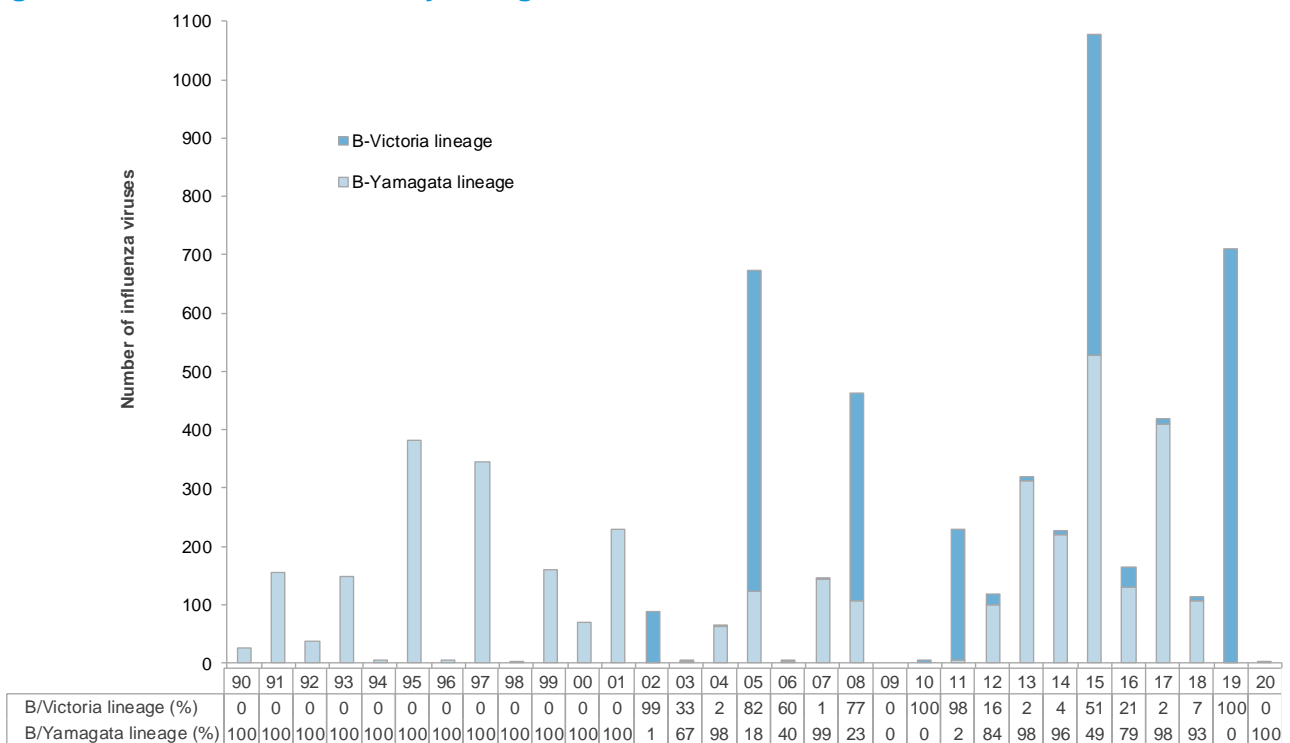


Figure 17 shows the number and percentage of all B viruses from 1990 to 2020 (excluding influenza B not lineage-typed). Overall, the patterns of the predominant influenza B among all lineage-typed B viruses during 1990–2020 are described below:

- Influenza B/Yamagata lineage was the only lineage circulating in New Zealand during 1990–2001. Relatively high number of influenza B viruses were recorded in 1995 and 1997.
- Since the introduction of the B/Victoria lineage viruses into New Zealand in 2002, this lineage has co-circulated with B/Yamagata lineage viruses. During 2002–2011, B/Victoria lineage viruses predominated over the B/Yamagata lineage viruses in every three years in New Zealand (2002, 2005, 2008 and 2011). In 2005, the disease burden was high in children aged 5–19 years with associated deaths in 3 children. In 2019, B/Victoria lineage was the predominant B lineage
- B/Yamagata lineage viruses was the predominant lineage over B/Victoria lineage virus during 2012–2014, 2016, 2017 and 2018.
- In 2015, there were almost equal proportions of B/Yamagata and B/Victoria lineage viruses.

Figure 17. Influenza B viruses by lineages, 1990–2020



4.2 INFLUENZA A(H1N1)PDM09

WHO National Influenza Centre (NIC) at ESR only received 5 influenza A(H1N1)pdm09 clinical samples (all collected in January 2020). Antigenic typing was conducted using rabbit antisera A/Michigan/45/2015/A(H1N1)pdm09 supplied by the WHO Collaborating Centre (WHOCC) in Melbourne. All of them were antigenically related to A/Michigan/45/2015/A(H1N1)pdm09.

715 A(H1N1)pdm09 viruses with collection dates between January to March 2020 were characterized at the Melbourne WHOCC from 7 countries with most coming from Australia. All A(H1N1)pdm09 viruses belonged to phylogenetic subclade 6B.1A with subclades 5A, 5B and 7 detected. The vast majority belonged to subclade 5A and have continued to diversify and the majority of viruses circulating since February 2020 fell into three genetic groups: the progenitor 5A subclade (i.e. 6B.1A5A) and two recently designated groups, 5A-V187A and 5A-N156K.

The antigenic characteristics of A(H1N1)pdm09 viruses were assessed with post-infection ferret antisera in haemagglutination inhibition (HI) assay. Only the subgroup of the 5A group viruses with V187A, representing approximately half of viruses tested, were well inhibited by ferret antisera raised against egg or MDCK-propagated A/Brisbane/02/2018 viruses. The other half of the 5A group viruses with N156K were poorly inhibited with ferret antisera to A/Brisbane/02/2018-like viruses but were well inhibited by ferret antisera raised to A/Victoria/2570/2019-like viruses.

Human serology studies were conducted using several serum panels from children (6 months to 17 years), adults (18-64 years) and elderly adults (≥65 years) who had received the 2019-2020 northern hemisphere vaccines, and two serum panels from adults who had received the 2020 southern hemisphere vaccine. Both vaccines contained A/Brisbane/02/2018-like viruses as the A(H1N1)pdm09 vaccine component. Geometric mean titres (GMTs) against recent representative cell culture-propagated 6B.1A viruses were determined by HI assays. When compared to titres against egg- and cell culture- propagated A/Brisbane/02/2018-like vaccine viruses, post-vaccination GMTs against most viruses representing HA groups 5A-V187A, 5A-N156K and subclade 5B were significantly reduced. Notably, the 5A-N156K viruses had the lowest GMTs among all the viruses tested across all serum panels.

(Abridged from the Weekly Epidemiological Record (WER), 2020 95(42):497-508 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 before March 2022. The majority of influenza A(H1N1)pdm09 viruses were antigenically and genetically drifted from the 2020 vaccine virus A/Brisbane/02/2018. Based on all of the available data, the WHO consultation recommended vaccines containing an A/Victoria/2570/2019 (H1N1)pdm09-like strain. The AIVC accepted this recommendation.

4.3 INFLUENZA A(H3N2)

The WHO National Influenza Centre at ESR did not receive any A(H3N2) positive clinical samples of 2020.

126 A(H3N2) viruses with collection dates between January to April 2020 were characterized at the Melbourne WHOCC from 8 countries with most coming from Australia. The majority of A(H3N2) viruses belonged to the phylogenetic clade 3C.2a1b (the largest group having HA T135K and T131K substitutions), however a significant number of 3C3a viruses were also detected.

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 such as the microneutralisation or plaque reduction assays or focus reduction assays (FRA) can be used where the NA binding is not relevant. Overall, Ferret antisera raised against egg-propagated A/South Australia/34/2019-like viruses (group 3C.2a1b+T131K) inhibited few recently circulating viruses well. However, the majority of viruses within subgroups of 3C.2a1b+T131K and 3C.2a1b+T135K were well inhibited by ferret antisera raised against cell culture-propagated A/Hong Kong/45/2019-like viruses, and less well by antisera raised against egg-propagated A/Hong Kong/2671/2019-like viruses.

Human serology studies using serum panels from adults who had received vaccine containing an A/South Australia/34/2019-like virus (southern hemisphere 2020 vaccine recommendation) in HI and Virus Neutralization (VN) assays showed significant reductions in GMTs against most recent representative cell culture-propagated A(H3N2) viruses from clade 3C.3a and subclade 3C.2a1b when compared to titres against egg-propagated A/South Australia/34/2019-like reference viruses. VN GMTs against cell culture-propagated circulating viruses were reduced to varying degrees when compared to cell culture-propagated A/South Australia/34/2019-like reference viruses. *(Abridged from the Weekly Epidemiological Record (WER), 2020 95(42):497-508 and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne).*

In summary, influenza A(H3N2) subclades 3C.2a1b viruses predominated in most countries globally but clade 3C.3a viruses were reported in Asia, Europe and Oceania. The great majority of these recently circulating viruses were poorly recognised by ferret antisera raised against A/South Australia/34/2019, the A(H3N2) vaccine component for the 2020 southern hemisphere influenza season. However, most of the viruses in subclade 3C.2a1b with additional HA1 amino acid substitutions at T131K or T135K were well inhibited by ferret antisera raised against cell culture-propagated A/Hong Kong/45/2019, though less well by ferret antisera raised against egg-propagated A/Hong Kong/2671/2019, the recommended vaccine components for the 2020-2021 northern hemisphere influenza season. Based on all available data, the WHO Consultative Group recommended the H3 component of the vaccines containing a cell-propagated A/Hong Kong/45/2019-like strain. AIVC accepted this recommendation.

4.4 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.

In 2020, the WHO National Influenza Centre at ESR did not receive any influenza B positive clinical samples.

90 influenza B isolates (including 89 B/Victoria and 1 B/Yamagata virus) with collection dates between January to March 2020 were characterized at the Melbourne WHOCC. Sequence analysis of the HA1 gene of the recent B/Victoria lineage viruses showed that they mostly belonged to genetic group V1A. All B/Victoria viruses had a HA triple deletion (162-164). 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 a large majority of viruses with the triple deletion were inhibited well by post-infection ferret antisera raised against both cell culture- and egg-propagated triple deletion viruses, such as B/Washington/02/2019. These viruses with the triple deletion were generally poorly inhibited by post-infection ferret antisera raised against both egg- and cell culture-propagated double deletion viruses (B/Colorado/06/2017-like, a former vaccine virus). Additionally, in HI assays the recently circulating B/Yamagata lineage viruses were inhibited well by post-infection ferret antisera raised against either cell culture- or egg-propagated B/Phuket/3073/2013.

Human serology studies used four serum panels from adults (26-64 years of age), two that received vaccines containing B/Washington/02/2019-like (triple deletion) and B/Phuket/3073/2013-like viruses (southern hemisphere 2020 vaccine recommendation) and two that received vaccines with the northern hemisphere 2019-2020 formulation (B/Colorado/06/2017 (double deletion) and B/Phuket/3073/2013). Post-vaccination HI GMTs against recent viruses of the B/Victoria lineage, representing the dominant HA1 triple deletion genetic group, were not significantly reduced when compared to titres against egg- or cell culture-propagated B/Washington/02/2019-like vaccine viruses. Post-vaccination HI GMTs against the great majority of recent representative B/Yamagata lineage viruses were not significantly reduced when compared to the cell culture-propagated B/Phuket/3073/2013 reference virus.

(Abridged from the Weekly Epidemiological Record (WER), 2020 95(42):497-508 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 the B/Yamagata/16/88 lineages co-circulated, with the B/Victoria lineage being dominant in all regions. The vast majority of B/Victoria lineage viruses contained a triple amino acid deletion in HA1 (positions 162-164) and were inhibited well by post-infection ferret antisera raised against both cell culture- and egg-propagated triple deletion viruses, such as B/Washington/02/2019. The few circulating B/Yamagata lineage viruses were antigenically and genetically closely related to the egg-propagated and cell-propagated vaccine virus B/Phuket/3073/2013. Based on all available data, the WHO Consultative Group recommended the B/Washington/2/2019-like virus (B/Victoria/2/87-lineage) and B/Phuket/3073/2013-like virus (B/Yamagata/16/88-lineage) as quadrivalent vaccine strains. AIVC accepted this recommendation.

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Local influenza coordinators within each Public Health Unit

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Research nurses and clinicians in SARI surveillance

Practice nurses and GPs in sentinel GP surveillance programme

WHO Collaborating Centre, Melbourne

WHO Collaborating Centre, Atlanta