

RECOMMENDATION FOR SEASONAL INFLUENZA VACCINE COMPOSITION FOR NEW ZEALAND FOR USE IN 2027

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1. RECOMMENDATION

The Australian Influenza Vaccine Committee (AIVC) met with a New Zealand representative in Canberra on 7 October 2026 to consult on the influenza vaccine composition for use in 2027 for New Zealand, Australia and South Africa.

Egg-based influenza vaccines:

- an A/Missouri/11/2025 (H1N1)pdm09-like virus;
- an A/Darwin/1454/2025 (H3N2)-like virus; and
- a B/Tokyo/EIS13-175/2025 (B/Victoria lineage) - like virus.

Cell-based or recombinant-based or nucleic acid-based influenza vaccines:

- an A/Missouri/11/2025 (H1N1)pdm09-like virus;
- an A/Darwin/1454/2025 (H3N2)-like virus; and
- a B/Pennsylvania/14/2025 (B/Victoria lineage)-like virus.

The continued absence of confirmed detection of naturally occurring B/Yamagata lineage viruses after March 2020 is indicative of a very low risk of infection by B/Yamagata lineage viruses. Consistent with previous four WHO recommendations since September 2023, it remains the opinion of the WHO influenza vaccine composition advisory committee that inclusion of a B/Yamagata lineage antigen is no longer warranted. **There will no longer be updated recommendations for the B/Yamagata lineage component.** The AIVC noted this position and supports the WHO committee's views.

Quadrivalent vaccines, where the transition to trivalent vaccines is not yet complete, contain a 4th component – a B/Yamagata lineage virus.

Influenza vaccine recommendations for New Zealand, 1994–2026

Decision		Use	A H3N2	A H1N1	B (Trivalent)	B (Quadrivalent)
NZ & WHO*	2026	2027	A/Darwin/1454/2025	A/Missouri/11/2025	B/Tokyo/EIS13-175/2025	B/Phuket/3073/2013
NZ & WHO*	2025	2026	A/Singapore/GP20238/2024	A/Missouri/11/2025	B/Austria/1359417/2021	B/Phuket/3073/2013
NZ & WHO*	2024	2025	A/Croatia/10136RV/2023	A/Victoria/4897/2022	B/Austria/1359417/2021	B/Phuket/3073/2013
NZ & WHO*	2023	2024	A/Thailand/8/2022	A/Victoria/4897/2022	B/Austria/1359417/2021	B/Phuket/3073/2013
NZ & WHO*	2022	2023	A/Darwin/9/2021	A/Sydney/5/2021	B/Austria/1359417/2021	B/Phuket/3073/2013
NZ & WHO*	2021	2022	A/Darwin/9/2021	A/Victoria/2570/2019	B/Austria/1359417/2021	B/Phuket/3073/2013
NZ & WHO*	2020	2021	A/Hong Kong/2671/2019	A/Victoria/2570/2019	B/Washington/02/2019	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/3/2006	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 recommendations are for the Southern Hemisphere winter; ** WHO recommendations are for the Northern Hemisphere winter;

2. SUMMARY

In 2026, influenza activity in New Zealand is described at an extraordinarily high level (above seasonal level). Overall impact on healthcare use in hospitals was extraordinarily high as measured by influenza-associated severe acute respiratory illness (SARI). Seriousness of disease (i.e. clinical 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 SARI admitted in ICU over influenza-associated SARI hospitalization. Virus transmissibility which reflects the ease of movements of the influenza virus between individuals and communities was extraordinarily high as measured by influenza-associated acute respiratory illness among the SHIVERS community cohort participants.

The hospital-based severe acute respiratory illness was extraordinarily high in 2026. Influenza-associated SARI in 2026 was the highest compared to 2022-2025. Influenza-associated SARI hospitalizations were higher in both young children (0–4 years) and elderly (≥ 65 years) compared to other age groups; also higher in Pacific Peoples and Māori ethnic groups compared to other ethnic groups.

The community cohort-based acute respiratory illness (ARI) was high in 2026, higher than 2022-2025. Influenza-associated ARI in 2026 was the highest compared to 2022-2025. The influenza-associated ARI disease burden was higher in children aged 0–19 years compared to other age groups; also higher in Pacific peoples and Māori compared to other ethnic groups.

The laboratory-based influenza surveillance tested samples from various surveillance systems as well as samples ordered by clinicians during routine hospital diagnosis. A total of 3740 influenza viruses were detected and reported through this system. Of them, influenza A represented 91.1% (3409) and influenza B 8.9% (331) of all influenza viruses. Among 1002 subtyped influenza A viruses, 54.6% (547) were A(H3N2) viruses and 45.4% (455) were A(H1N1)pdm09 viruses.

WHO National Influenza Centre (NIC) at the New Zealand Institute for Public Health and Forensic Science (formerly ESR) conducted antigenic/genetic typing: 1) 29 influenza A(H1N1)pdm09 viruses were antigenically closely related to the vaccine strain A/Missouri/11/2025(H1N1)pdm09-like virus. Genetically most of influenza A(H1N1)pdm09 viruses fell into subclade D.3.1.1; 2) 59 (98.3%) influenza A(H3N2) viruses were antigenically closely related to the vaccine strain A/Sydney/1359/2024 (H3N2)-like virus. One (1.7%) A(H3N2) virus was antigenically drifted away from the vaccine strain. Genetically most of influenza A(H3N2) viruses fell into subclade K. 3) 3 influenza B/Victoria-lineage viruses were antigenically closely related to the vaccine strain B/Austria/1359417/2021-like virus. No influenza B sequences were available for reporting.

For influenza-confirmed SARI cases among acutely admitted hospital patients, the estimated adjusted vaccine effectiveness (VE) was 46% (95% CI: 30, 58). For influenza-confirmed ILI cases among GP consultation patients, the estimated adjusted vaccine effectiveness was 58% (95% CI: 37, 72). For influenza-confirmed ARI among cohort participants, the estimated adjusted vaccine effectiveness was 45% (95% CI: 29, 57).

3. EPIDEMIOLOGY - NEW ZEALAND INFLUENZA SEASON IN 2026

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 hospital-based surveillance (hospital-based SARI surveillance and Ministry of Health data on publicly funded hospital discharges). Additionally, the surveillance system includes community-based surveillance (community-based longitudinal cohort surveillance and sentinel GP based virological surveillance). Furthermore, the surveillance includes laboratory-based surveillance for testing ordered by clinicians for hospital in-patients and outpatients during routine viral diagnosis as well as testing for public health surveillance.

3.1 HOSPITAL-BASED SURVEILLANCE

3.1.1 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.

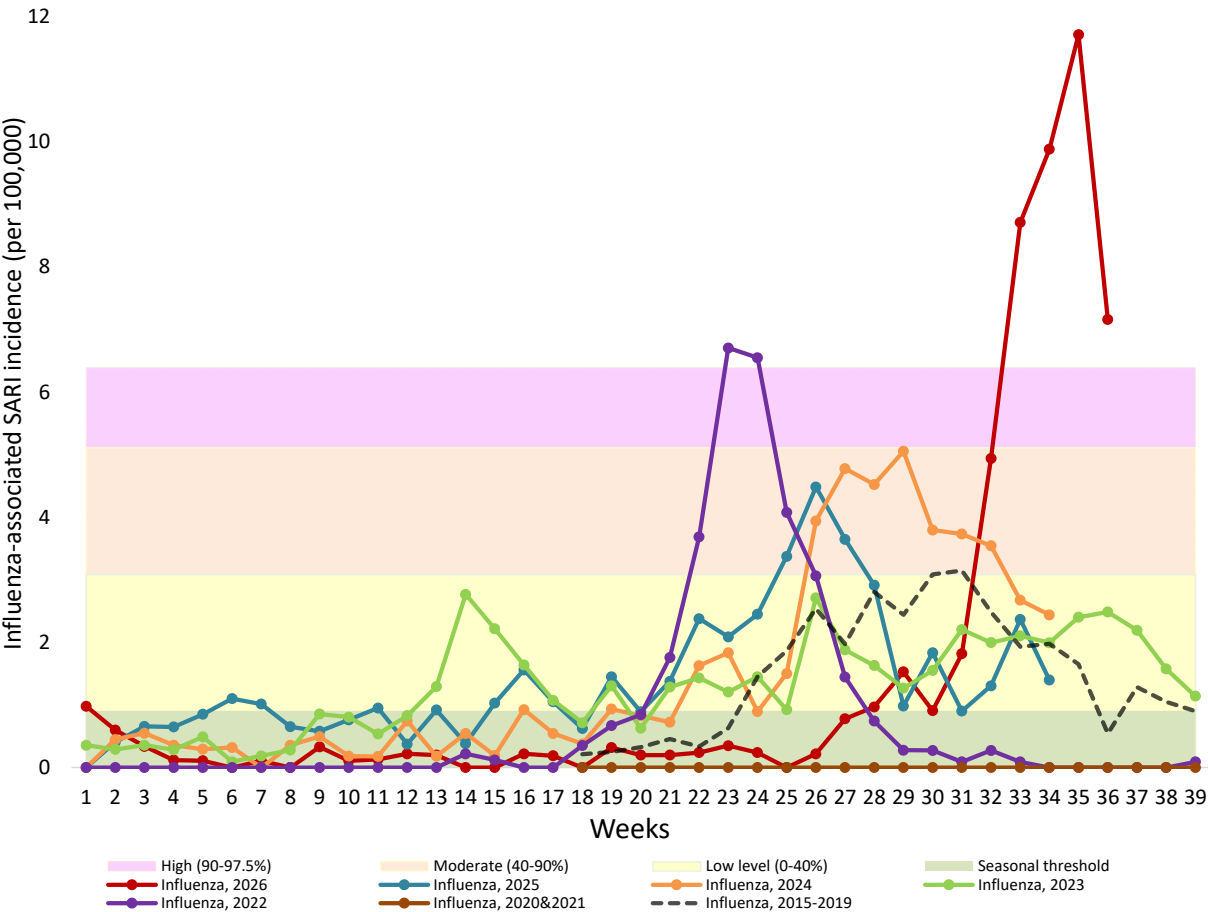
The case definition being used is the World Health Organisation (WHO) SARI case definition: *"an acute respiratory illness with a history of fever or measured fever of $\geq 38^{\circ}\text{C}$, and cough, and onset within the past 10 days, and requiring inpatient hospitalisation"*. If a patient with 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. Incidence rates were calculated along with 95% confidence intervals (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.

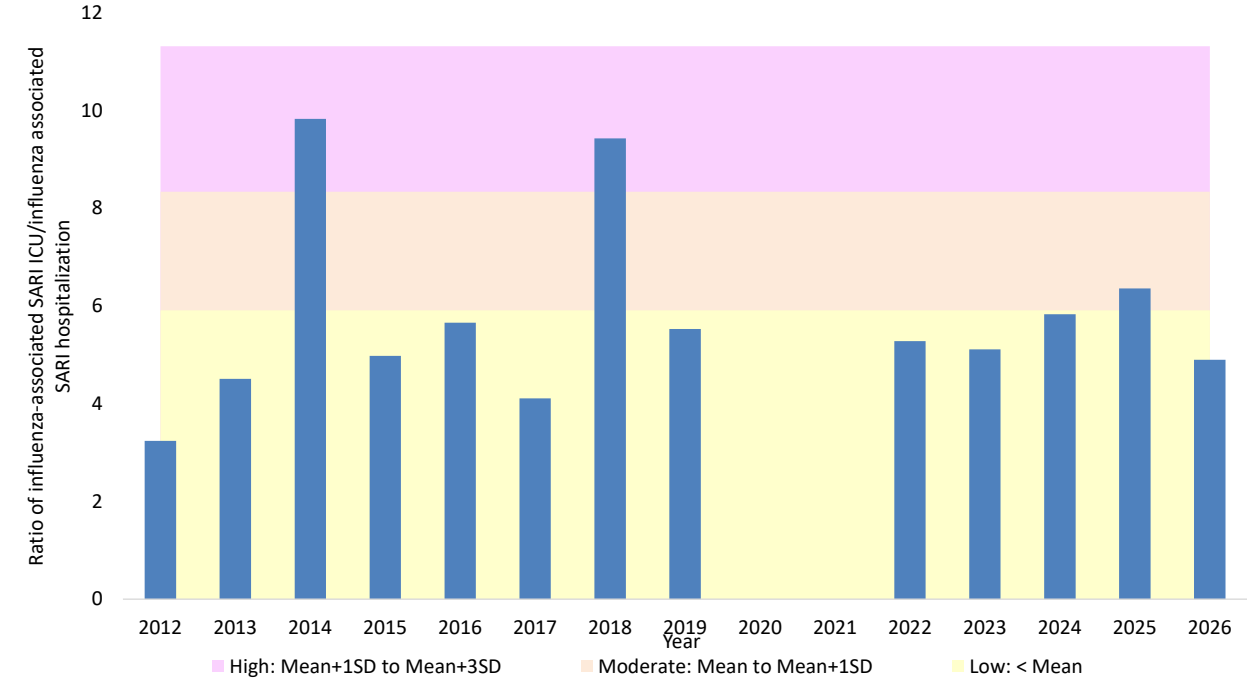
Overall impact on healthcare use is measured by influenza-associated severe acute respiratory illness (SARI) hospitalizations. Overall impact was extraordinarily high, above seasonal level. During the study period from week 1 (commencing 1 January 2026) to week 36 (ending 6 September 2026), influenza-associated SARI hospitalizations were below seasonal level from week 1 (commencing 1 January 2026) to week 27 (ending 5 July 2026). It increased sharply and above seasonal level in week 33 and peaked at week 35 (ending 30 August 2026) (Figure 1). Overall impact was extraordinarily high (above seasonal level) in 2026.

Figure 1. Influenza-associated SARI hospitalizations in 2026 compared to pre-pandemic 2015-2019, and during and post COVID-19 pandemic 2020-2025



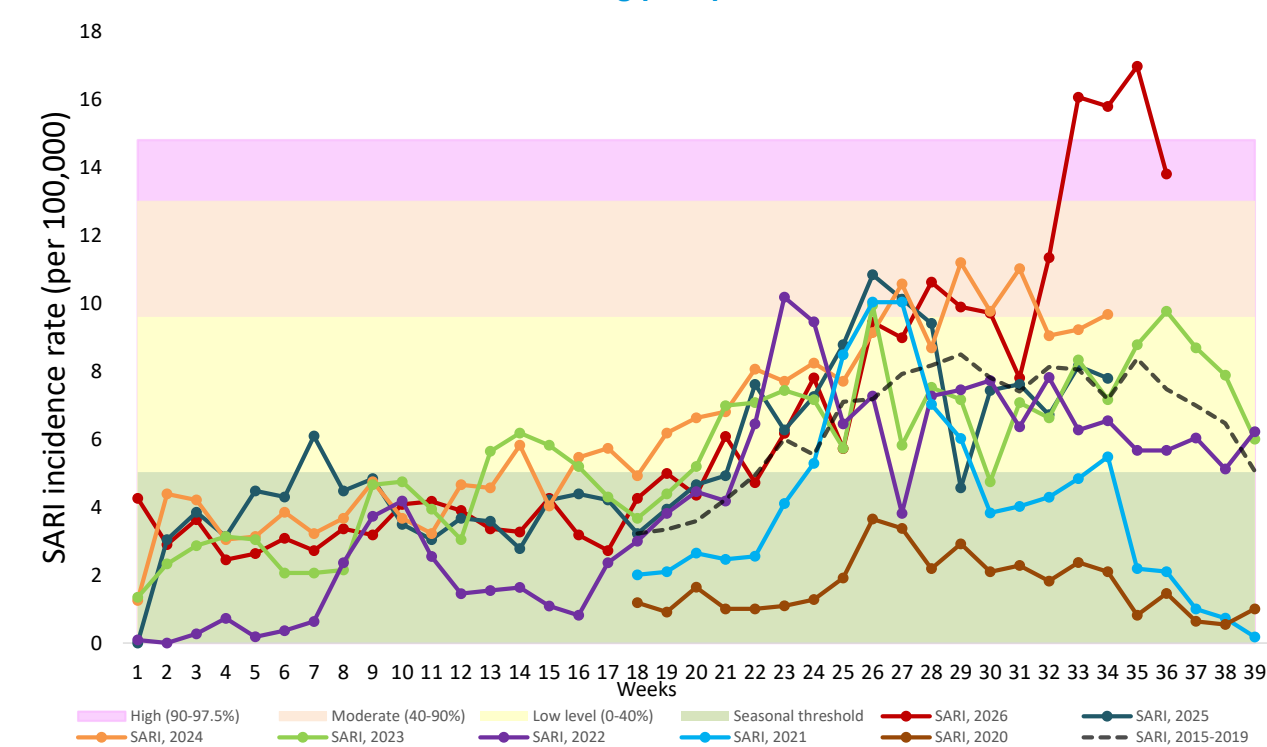
Seriousness of disease (i.e. clinical severity) indicates the extent to which individuals get sick when infected with the influenza virus as measured by the ratio of influenza-associated SARI admitted in ICU over influenza-associated SARI hospitalization. Seriousness of disease was low in 2026 (Figure 2).

Figure 2. Seriousness of disease indicator in 2026 compared to 2012–2025



SARI hospitalization was below baseline during weeks 1-20, then increased to low and moderate level during weeks 21-32, above the seasonal level at week 33, peaked at week 35 (Figure 3). SARI hospitalization was extraordinarily high (above seasonal level).

Figure 3. Weekly hospitalisation rates for SARI in 2026 compared to pre-pandemic 2015-2019, during/post pandemic 2020-2025



From 1 January to 30 August 2026, there were 110,500 acute admissions to ADHB and CMDHB hospitals (Table 1). A total of 6314 patients with suspected respiratory infections were assessed. Of these, 2589 (41%) patients met the SARI case definition, giving a proportion of SARI among acutely admitted hospital patients as 23.4 per 1000 patients. Of those SARI hospital patients, 652 were influenza positive. Among ADHB and CMDHB residents who were acutely admitted in hospitals, 2403 were SARI cases, giving the SARI incidence rate of 218.0 per 100 000 population (186.4 per 100 000 in 2025). Among the SARI cases who were ADHB and CMDHB residents, 416 had positive influenza virus. This gives influenza-associated SARI incidence of 46.3 per 100 000 population, slightly lower than 47.1 per 100 000 in 2025.

Influenza-associated SARI hospitalizations were higher in both young children (0–4 years) and elderly (≥65 years) groups compared to other age groups; also higher in Pacific Peoples and Māori ethnic groups compared to other ethnic groups.

Table 1. Demographic characteristics of SARI cases and related influenza cases, weeks 1 – 35, 2026

Characteristics	SARI & influenza					SARI & influenza cases among ADHB & CMDHB residents					
	Admissions	Assessed	SARI	SARI per 1000 patients	Influenza positive	SARI cases	SARI incidence (per 100 000)	Influenza-associated SARI	Influenza SARI incidence (per 100 000)	Influenza cases (SARI & non SARI)	Influenza incidence (SARI & non SARI) (per 100 000)
Overall	110500	6314	2589	23.4	652	2403	218.0	416	46.3	604	80.6
Age group (years)											
<1	2818	926	362	128.5	48	335	2571.0	33	306.3	42	419.7
1–4	7,288	882	486	66.7	91	424	842.1	59	151.5	79	226.4
5–19	11,372	548	261	23.0	81	223	104.7	54	29.5	71	45.6
20–34	20,165	239	125	6.2	35	119	47.7	31	15.9	35	21.6
35–49	18,649	404	182	9.8	46	175	76.0	31	16.4	45	29.5
50–64	17,429	728	298	17.1	78	286	151.0	50	32.3	73	56.5
65–79	19,658	1428	534	27.2	162	511	423.4	96	95.4	150	184.7
>80	13,120	1154	340	25.9	110	329	904.8	62	213.3	108	494.2
Unknown	1	5	1		1	1				1	
Ethnicity											
Māori	16,349	1323	521	31.9	107	474	320.4	68	54.6	102	94.4
Pacific peoples	23,709	1850	862	36.4	208	830	405.1	141	81.4	198	129.0
Asian	24,767	747	332	13.4	90	311	89.7	65	23.4	86	36.7
European and Other	44,848	2394	874	19.5	247	788	195.7	142	45.7	218	90.9
Unknown	827	0	0	0.0	0	0		0		0	
DHB of Residence											
ADHB	64520	3035	1201	18.6	209	1045	220.2	163	53.8	180	109.7
CMDHB	45980	3275	1386	30.1	441	1356	216.0	252	42.2	422	70.5
Other	0	4	2		2	2		1		2	
Sex											
Female	59,406	3010	1222	20.6	323	1137	205.5	206	45.6	301	80.3
Male	50,999	3298	1364	26.7	327	1263	230.1	209	46.8	301	80.5
Unknown	95	6	3	31.6	2	3		1		2	

From 1 January to 30 August 2026, 2097 SARI specimens have been tested and 450 (21.5%) were positive for influenza viruses (Table 2). Of the 261 specimens collected from ICU admitted patients with acute respiratory illness (SARI and non-SARI), 39 (14.9%) were positive for influenza viruses. Of the 47 specimens collected from fatal cases with acute respiratory illness (SARI and non-SARI), 5 (10.6%) were positive for influenza A viruses. Influenza A(H1N1)pdm09 and A(H3N2) viruses were two predominant co-circulating strains.

Additionally, 1080 SARI specimens were tested for non-influenza respiratory viruses (Table 2).

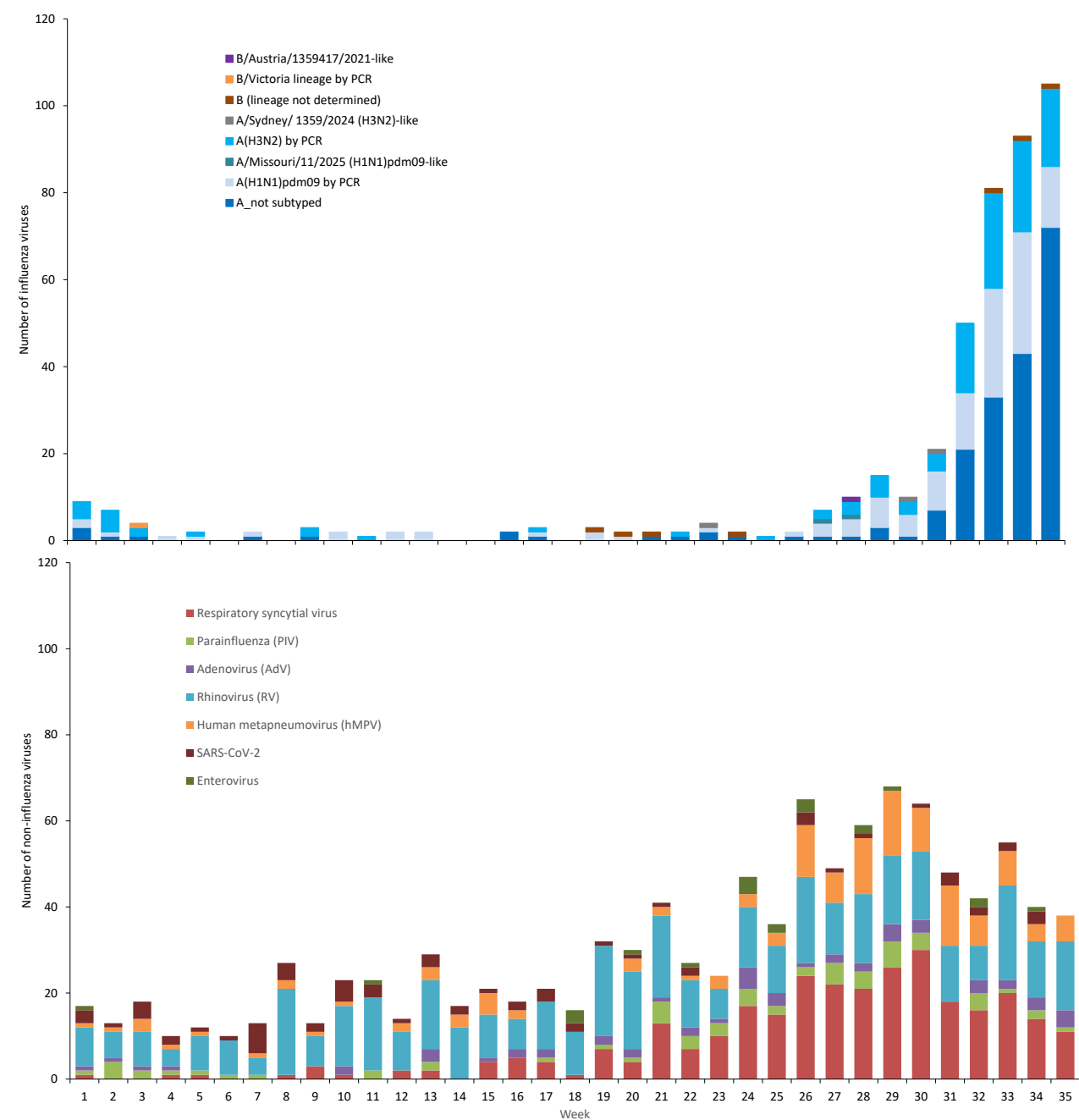
Table 2. Influenza and non-influenza respiratory viruses among SARI cases, 2026

<i>Influenza viruses</i>	<i>SARI Cases</i>	<i>SARI and non-SARI</i>	
		<i>ICU</i>	<i>Deaths</i>
No. of specimens tested	2097	261	47
No. of positive specimens	450 (21.5%)	39 (14.9%)	5 (10.6%)
Influenza A	441	39	5
A (not subtyped)	198	20	4
A(H1N1)pdm09	128	12	1
A(H1N1)pdm09 by PCR	126	12	1
A/Missouri/11/2025 (H1N1)pdm09-like	2		
A(H3N2)	115	7	0
A(H3N2) by PCR	112	7	0
A/Sydney/ 1359/2024 (H3N2)-like	3	0	0
Influenza B	9	0	0
B (lineage not determined)	7	0	0
B/Yamagata lineage	0	0	0
B/Yamagata lineage by PCR	0		
B/Phuket/3073/2013-like	0		
B/Victoria lineage	2	0	0
B/Victoria lineage by PCR	1		
B/Austria/1359417/2021-like	1		
<i>Non-influenza respiratory viruses</i>	<i>SARI cases</i>	<i>SARI and non-SARI</i>	
		<i>ICU</i>	<i>Deaths</i>
No. of positive viruses	1080	161	9
SARS-CoV-2	68	7	3
Respiratory syncytial virus (RSV)	301	30	2
Parainfluenza (PIV)	64	10	0
Rhinovirus (RV)	433	94	3
Adenovirus (AdV)	54	7	0
Human metapneumovirus (hMPV)	138	11	1
Enterovirus	22	2	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 of influenza and other respiratory viruses in 2026 is shown in Figure 4. Influenza A(H1N1)pdm09 outnumbered A(H3N2) viruses from May to July. Since August, high number of A(H3N2) viruses co-circulated with A(H1N1)pdm09 viruses and A(H3N2) viruses surpassed A(H1N1)pdm09 at the end of August (Note: Many influenza A viruses are yet to be subtyped).

Figure 4. Temporal distribution of the number and proportion of influenza and other respiratory viruses from SARI specimens, by subtype and week¹ in 2026



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

3.1.2 Ministry of Health data on publicly funded hospital discharges

Hospitalisation data for influenza (ICD-9-CMA-II code 487) for 2026 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 2000–2026. 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 6 September 2026, there were a total of 1750 hospitalisations (32.9 per 100,000) for influenza (Figure 5). 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 2026 from 1 January to 6 September 2026 only.

Figure 5. Influenza hospital discharge rates, 2000–2026*

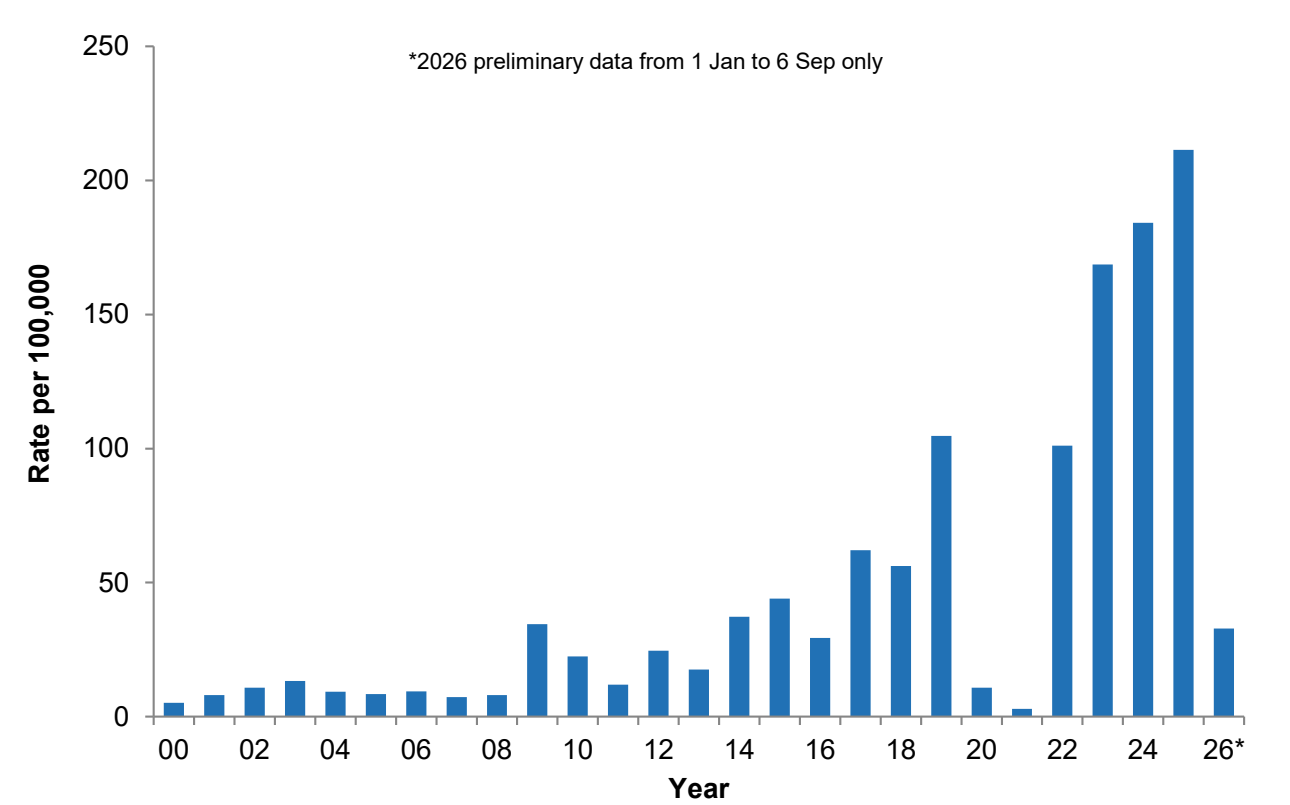
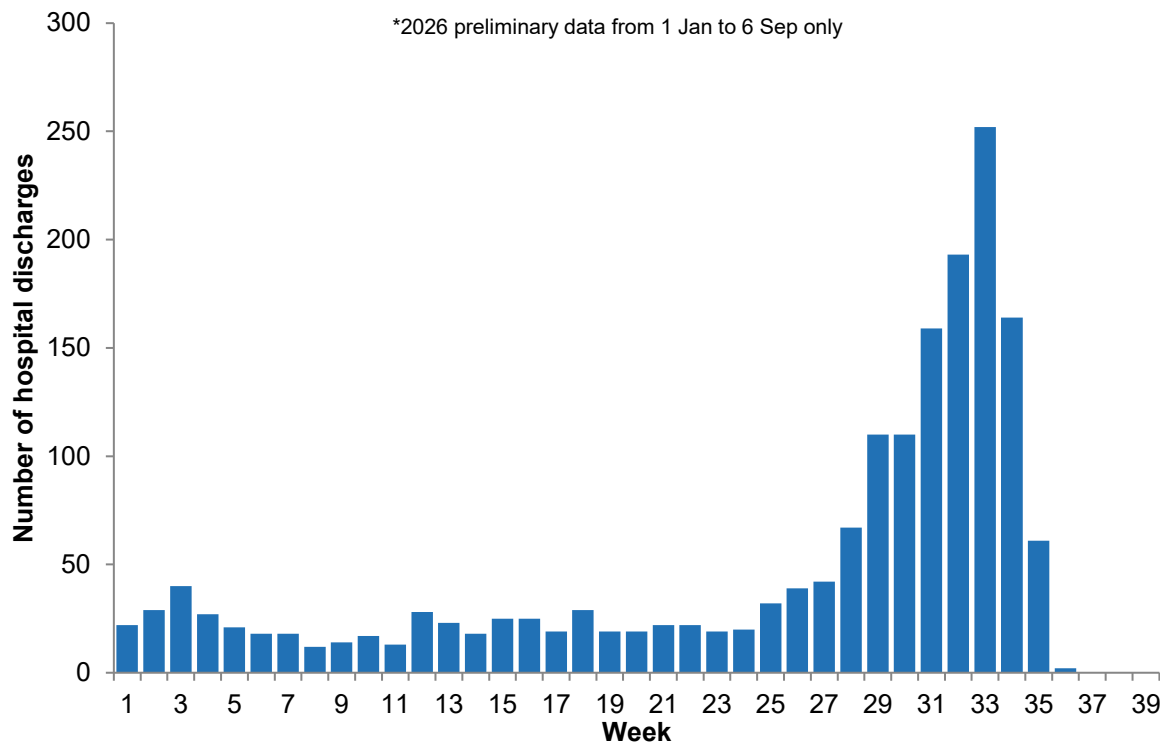


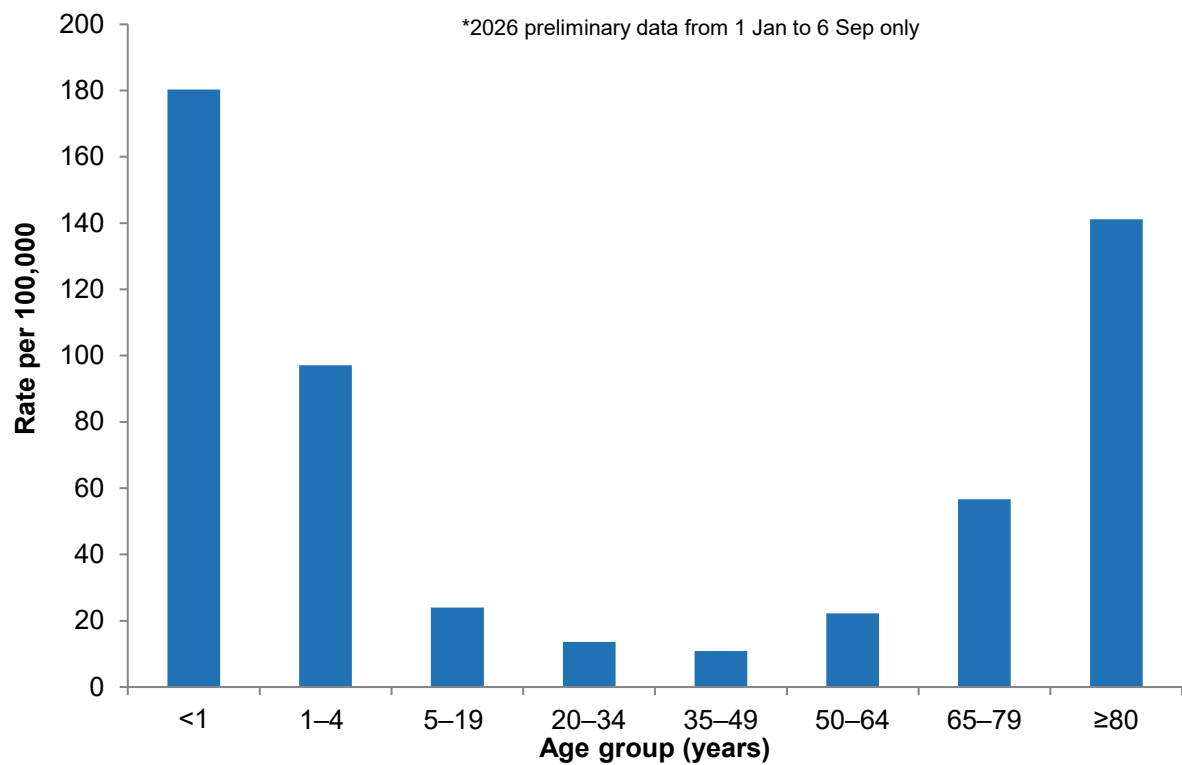
Figure 6 shows influenza hospitalisations by week discharged. The highest number of hospitalisations (252) occurred in week 33 (week ending 16 August 2026).

Figure 6. Influenza hospital discharges by week, 2026*



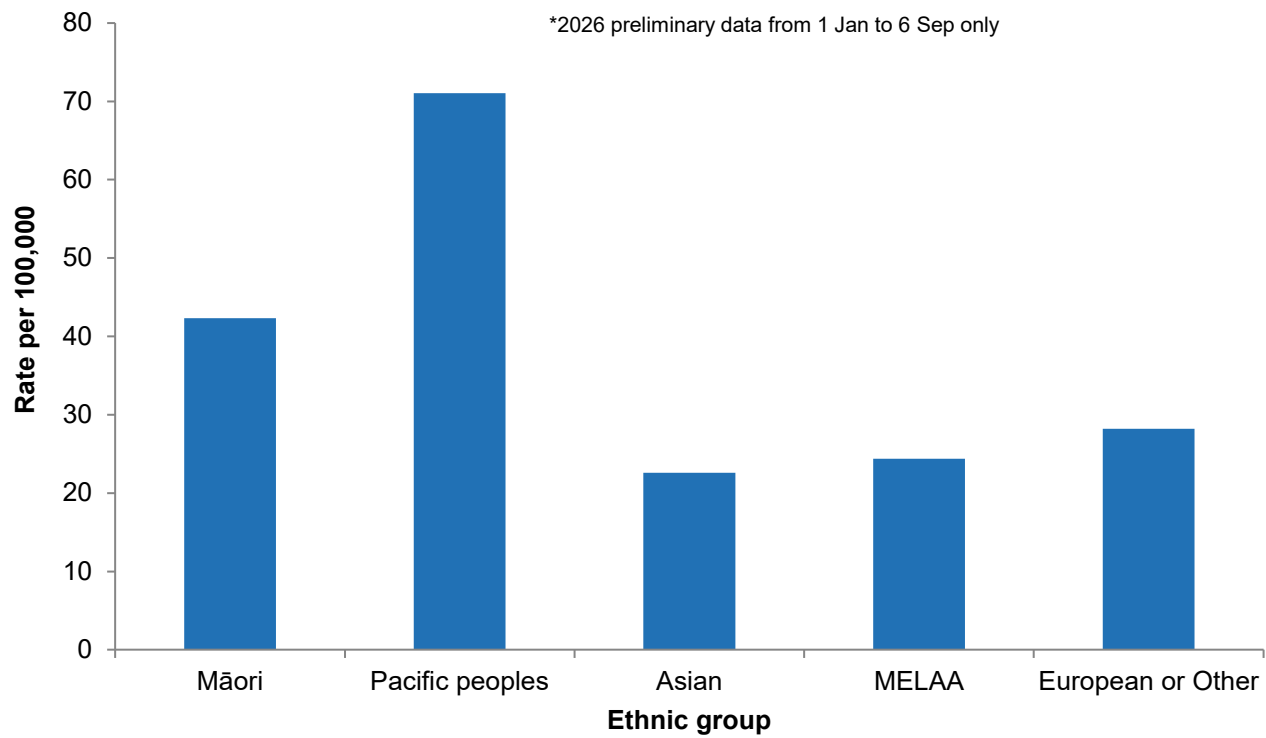
From 1 January to 6 September 2026, the highest influenza hospitalisation rates were recorded among infants <1 year (180.3 per 100,000) followed by elderly aged ≥80 years (141.2 per 100,000) (Figure 7).

Figure 7. Influenza hospital discharge rates by age group, 2026*



The ethnic distribution of influenza hospitalisations in 2026 is shown in Figure 8. Pacific peoples had the highest hospitalisation rate (71.1 per 100,000) followed by Māori (42.3 per 100,000).

Figure 8. Hospital discharge rates by prioritised ethnic group, 2026*



MELAA – Middle Eastern/Latin American/African

3.2 COMMUNITY-BASED SURVEILLANCE

3.2.1 Community-based longitudinal cohort study

SHIVERS-II (the second iteration of the Southern Hemisphere Influenza and Vaccine Effectiveness Research and Surveillance programme) is a prospective adult cohort study in Wellington, NZ. The cohort study is also called WellKiwis Adult study and has been in operation since 2018 enrolling individuals aged 20-69 years, randomly selected from those healthy individuals listed in the general practice's primary care management system. In 2026, SHIVERS-II study staff followed these participants (~800) and monitored their ILIs and acute respiratory illness (ARI)s.

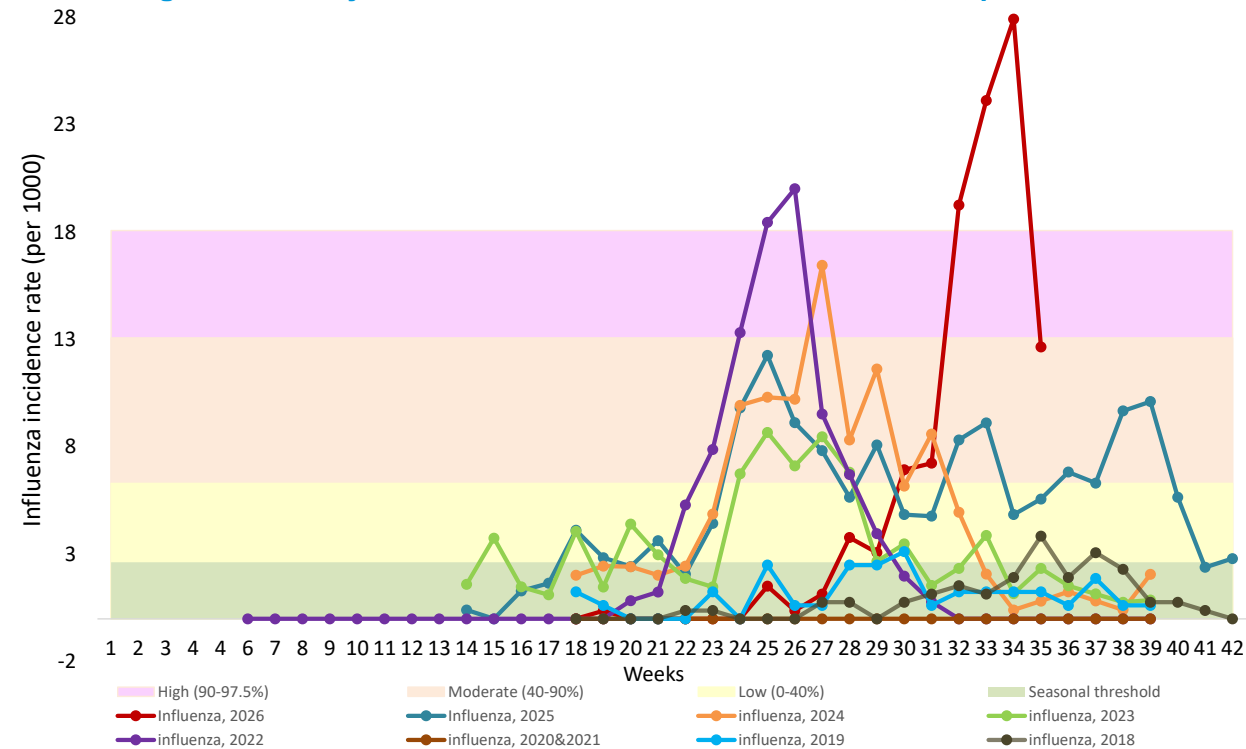
SHIVERS-III (i.e. WellKiwis Infant) is a prospective Wellington infant cohort aiming to recruit 600 infant-mother pairs from Oct 2019-Sept 2022 (200 pairs a year) and follow them until 2029. In 2026, the study staff followed up ~700 infants and monitored their ILIs and ARIs.

SHIVERS-IV (i.e. WellKiwis Household) is a prospective Wellington household cohort in Wellington, NZ. Households with at least one child aged 19 years or younger are invited to participate from SHIVERS-II and III participants and individuals randomly selected from participating general practice's patient list. Enrolled participants are to be followed for 7 years during 2021-2028. In 2026, the study staff followed up ~1700 household members and monitored their ILIs and ARIs.

During 27-April (week 18) to 30-August (week 35) 2026, the study staff sent weekly surveys to participants regarding their respiratory illness. The ARI case definition was: "acute respiratory illness with fever or feverishness and/or one of following symptoms (cough, running nose, wheezing, sore throat, shortness of breath, loss of sense of smell/taste) with onset in the past 10 days." The case definition for ILI was: "acute respiratory illness with cough and fever/measured fever of $\geq 38^{\circ}\text{C}$ and onset within the past 10 days". For those participants who met the case definition for ILI/ARI, research nurses guided the participant to take a nasal swab to test for influenza, SARS-CoV-2, RSV, rhinovirus, parainfluenza virus types 1-3, human metapneumovirus, adenovirus and enterovirus.

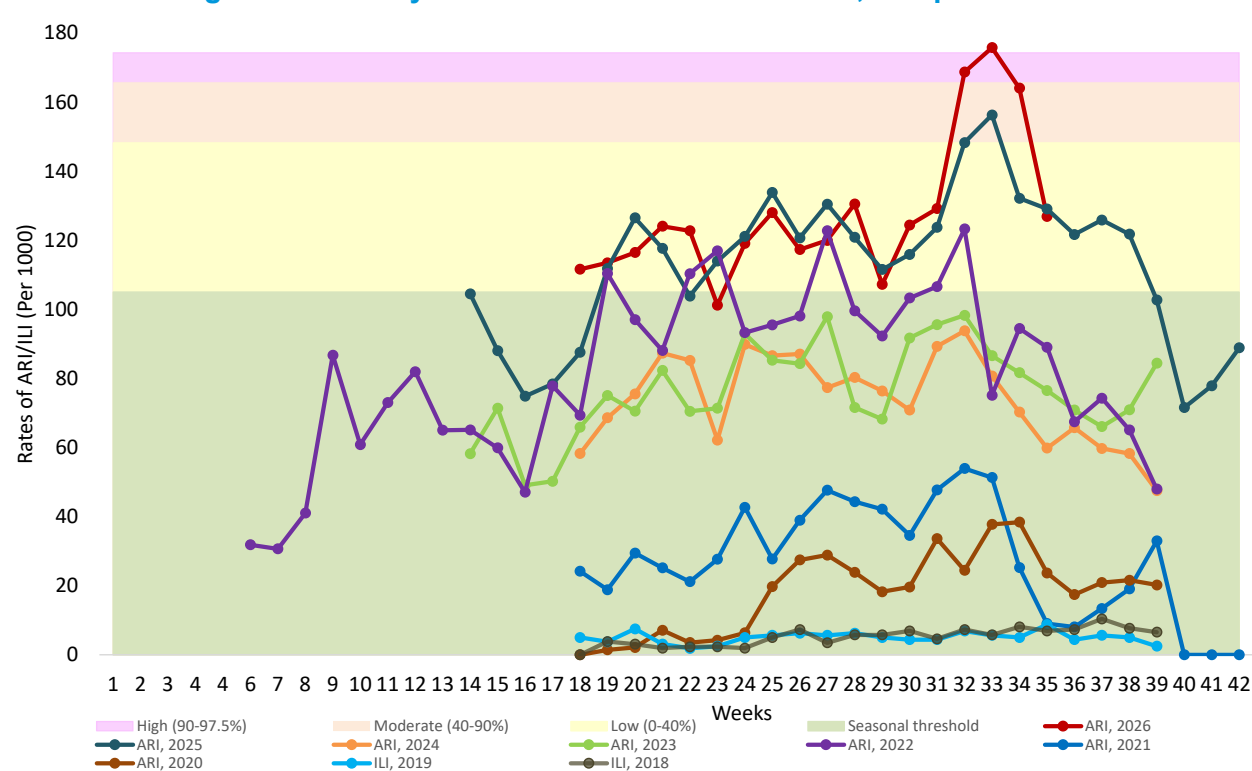
Virus transmissibility reflects the ease of movements of the influenza virus between individuals and communities, and it is measured by influenza-associated ARI. In 2026, the influenza-associated ARI was at extraordinarily high (above seasonal level), higher than other years. It peaked at week 34 (Figure 9).

Figure 9. Weekly influenza associated ARI rates in 2026, compared to 2018–2025



The ARI rates in 2026 was at high level, higher than other years. It peaked in week 33 (ending 16 August 2026) (Figure 10).

Figure 10. Weekly ARI/ILI incidence rates in 2026, compared to 2018–2025



From 27-April to 30-August 2026, a total of 6027 participants with acute respiratory illness (ARI) were reported, giving the ARI incidence rate of 192.1 per 100 population (196.9 per 100 in 2025) (Table 3). Of the ARI cases, 282 had positive influenza virus results. This gave the influenza-associated ARI incidence of 9.0 per 100, higher than 8.5 per 100 in 2025.

The influenza-associated ARI disease burden was higher in children aged 0–19 years compared to other age groups. Influenza-associated ARI were higher in Pacific peoples and Māori ethnic groups than Asians, Europeans and others ethnic groups.

Table 3. Demographic characteristics of ARI cases and related influenza cases, during 27 April to 30 August, 2026

Characteristics	ARI cases among WellKiwis participants		Influenza cases among WellKiwis participants	
	ARI Cases	ARI incidence (per 100)	Influenza Cases	Influenza incidence (per 100)
Overall	6027	192.1 (187.5, 196.7)	282	9.0 (8.0, 10.1)
<i>Age group (years)</i>				
<1	418	354.2 (324.1, 385.8)	9	7.6 (3.5, 14.5)
1–4	1817	306.4 (293.7, 319.5)	84	14.2 (11.3, 17.5)
5–19	1079	187.0 (176.6, 197.9)	90	15.6 (12.6, 19.2)
20–34	405	192.9 (175.4, 211.4)	14	6.7 (3.6, 11.2)
35–49	1703	202.7 (193.7, 212.0)	58	6.9 (5.2, 8.9)
50–64	416	86.8 (78.9, 95.4)	20	4.2 (2.6, 6.4)
≥65	189	58.9 (50.9, 67.7)	7	2.2 (0.9, 4.5)
<i>Ethnicity</i>				
Māori	590	183.8 (170.0, 198.4)	36	11.2 (7.9, 15.5)
Pacific peoples	307	251.6 (226.0, 279.0)	26	21.3 (13.9, 31.1)
Asian	560	202.2 (186.6, 218.5)	29	10.5 (7.0, 15.0)
European and Other	4570	189.0 (183.8, 194.3)	191	7.9 (6.8, 9.1)
<i>Sex</i>				
Female	3334	189.8 (183.7, 195.9)	153	8.7 (7.4, 10.2)
Male	2687	195.4 (188.5, 202.5)	129	9.4 (7.8, 11.1)
Other	6	100.0 (37.2, 210.6)	0	0.0 (0.0, 60.4)

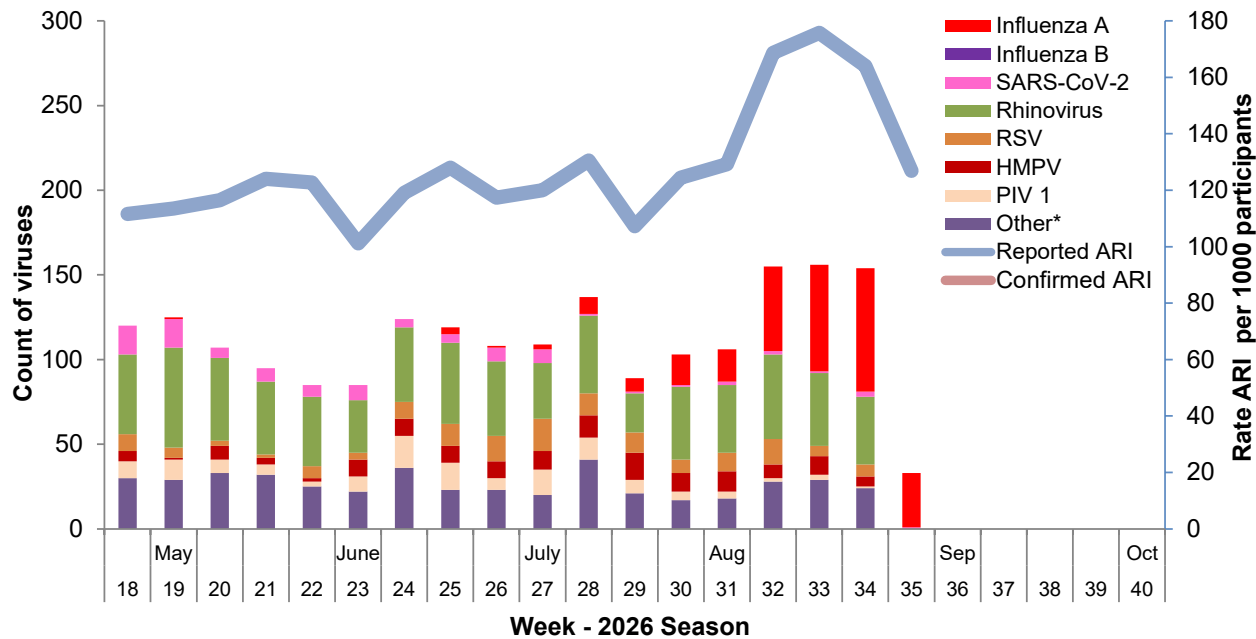
From 27-April to 30-August 2026, 3199 respiratory specimens have been tested and 282 (8.8%) were positive for influenza viruses. Of which, A(H3N2) (150) was the predominant strain. Influenza A(H1N1)pdm09 (94) co-circulated throughout this period (Table 4). Additionally, 3200 specimens were tested for non-influenza respiratory viruses.

Table 4 Influenza and Non-influenza respiratory viruses among ARI cases, 27 April to 30 August 2026

<i>Influenza viruses</i>	WellKiwis Households	Wellkiwis Adults	WellKiwis Infants	Total
No. of specimens tested	1760	245	1194	3199
No. of positive specimens (%) ¹	162 (9.2)	25 (10.2)	95 (8.0)	282 (8.8)
Influenza A	162	25	95	282
A (not subtyped)	19	6	13	38
A(H1N1)pdm09	55	10	29	94
A(H1N1)pdm09 by PCR	55	10	29	94
A/Missouri/11/2025 (H1N1)pdm09-like	0	0	0	0
A(H3N2)	88	9	53	150
A(H3N2) by PCR	88	9	53	150
A/Sydney/ 1359/2024 (H3N2)-like	0	0	0	0
Influenza B	0	0	0	0
B (lineage not determined)	0	0	0	0
B/Yamagata lineage	0	0	0	0
B/Yamagata lineage by PCR	0	0	0	0
B/Phuket/3073/2013 – like	0	0	0	0
B/Victoria lineage	0	0	0	0
B/Victoria lineage by PCR	0	0	0	0
B/Austria/1359417/2021-like	0	0	0	0
Influenza and non-influenza co-detection (% +ve)	13 (8.0)	1 (4.0)	13 (13.7)	27 (9.6)
<i>Non-influenza respiratory viruses</i>	WellKiwis Households	Wellkiwis Adults	WellKiwis Infants	Total
No. of specimens tested	1761	245	1194	3200
No. of positive specimens (%) ¹	705 (40.0)	90 (36.7)	719 (60.2)	1514 (47.3)
Respiratory syncytial virus (RSV)	59	7	95	161
Parainfluenza 1 (PIV1)	55	7	79	141
Parainfluenza 2 (PIV2)	25	0	23	48
Parainfluenza 3 (PIV3)	6	0	25	31
Rhinovirus (RV)	375	57	292	724
Adenovirus (AdV)	65	1	134	200
Human metapneumovirus (HMPV)	54	8	87	149
Enterovirus	87	9	76	172
SARS-CoV-2	55	5	42	102
Single virus detection (% of positives)	633 (89.8)	86 (95.6)	594 (82.6)	1313 (86.7)
Multiple virus detection (% of positives)	72 (10.2)	4 (4.4)	125 (17.4)	201 (13.3)

Figure 11 shows the weekly rate of acute respiratory illness (ARI) and associated viruses detected among the SHIVERS-II, III, IV cohort participants during the active surveillance period in 2026.

Figure 11. Weekly incidence rate of acute respiratory illness and associated viruses in 2026



Note: other viruses include enterovirus, adenovirus and parainfluenza virus types 2 and 3. The left axis indicates number of respiratory viruses detected among participants each week. The different coloured bars on the graph represent the count of the different respiratory viruses detected. The right axis shows weekly ARI rates - the blue line is the weekly rate of ARI reported by participants (per 1000). (Note: The case definition since 2020 has been widened compared to previous years, 2018–2019. This is to increase the sensitivity to detecting influenza as well as SARS-CoV-2 that causes COVID-19 infection). X-axis is based on the date of symptom onset.

3.2.2 Community-based sentinel GP virological surveillance

Virological surveillance at sentinel GP sites provides insight into the prevalence of respiratory viruses circulating in the community at any one time. GPs that participate in virological ILI surveillance take a nasopharyngeal or throat swab from some of the ILI patients they see each week.

In 2026, 34 sentinel general practices participated in this surveillance. Between 1 January to 30 August 2026, a total of 965 ILI specimens were tested for influenza viruses (Table 5) and 169 (17.5%) were positive for influenza with 116 influenza A(H3N2) and 42 A(H1N1)pdm09 detected.

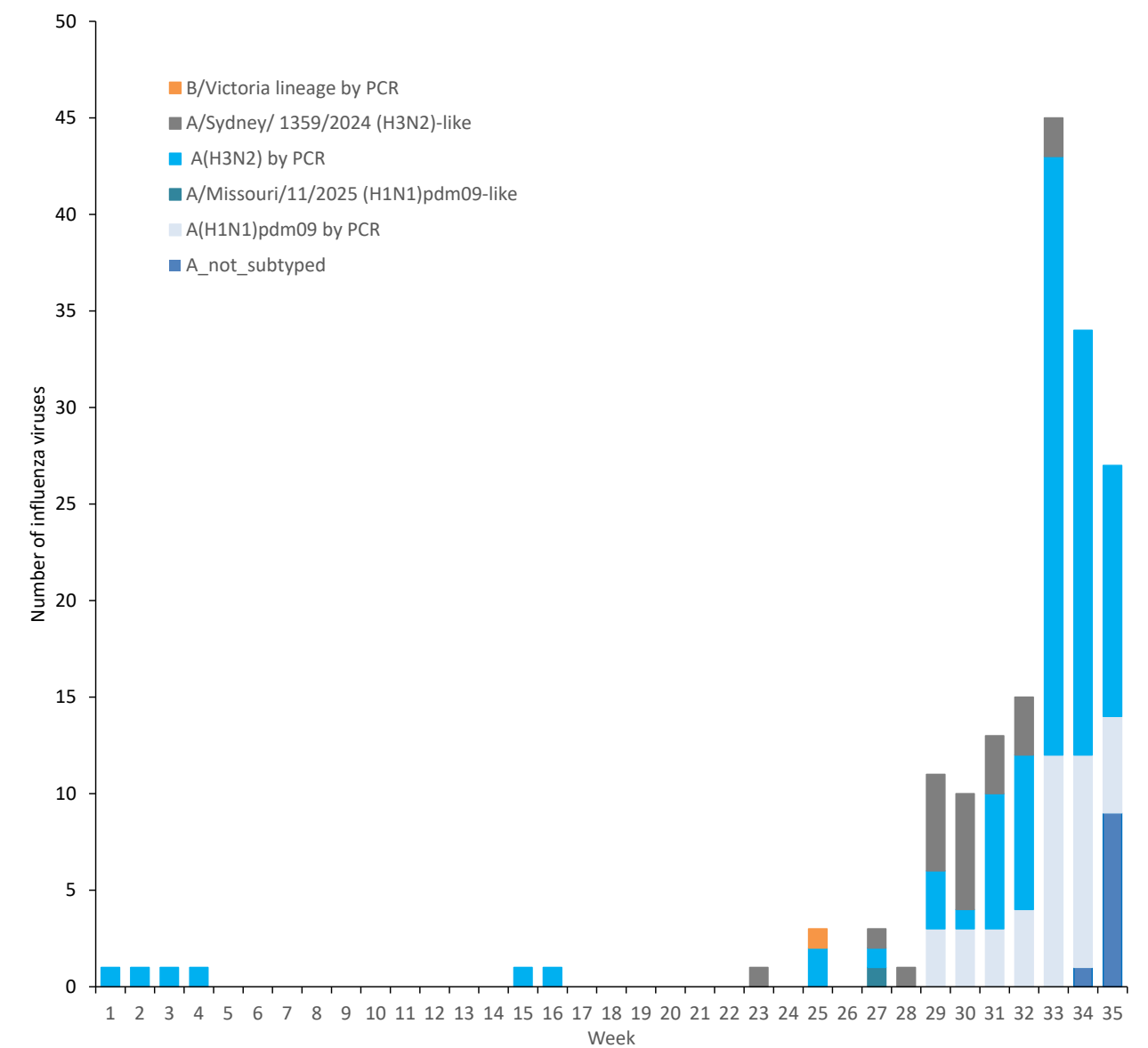
Additionally, a total of 965 ILI specimens were tested for non-influenza viruses and 356 (34.3%) were positive with non-influenza viruses.

Table 5. Influenza and non-influenza respiratory viruses among ILI cases, 1 January to 30 August, 2026

<i>Influenza viruses</i>	Total
No. of specimens tested	965
No. of positive specimens	169 (17.5%)
Influenza A	
A (not subtyped)	10
A(H1N1)pdm09	42
A(H1N1)pdm09 by PCR	41
A/Missouri/11/2025 (H1N1)pdm09-like	1
A(H3N2)	116
A(H3N2) by PCR	94
A/Sydney/ 1359/2024 (H3N2)-like	22
Influenza B	1
B (lineage not determined)	0
B/Yamagata lineage	0
B/Yamagata lineage by PCR	0
B/Phuket/3073/2013-like	0
B/Victoria lineage	1
B/Victoria lineage by PCR	1
B/Austria/1359417/2021-like	0
Non-influenza respiratory viruses	Total
No. of specimens tested	965
No. of positive viruses	356 (34.3%)*
SARS-CoV-2	20
Respiratory syncytial virus (RSV)	56
Parainfluenza 1 (PIV1)	22
Parainfluenza 2 (PIV2)	10
Parainfluenza 3 (PIV3)	9
Rhinovirus (RV)	126
Adenovirus (AdV)	31
Human metapneumovirus (hMPV)	57
Enterovirus	25

The temporal distribution of the number and proportion of the influenza viruses from 1 January to 30 August 2026, is shown in Figure 12. Influenza A(H3N2) was the predominant virus and peaked in week 33. A(H1N1)pdm09 co-circulated with A(H3N2).

Figure 12. Temporal distribution of the number and proportion of influenza viruses from ILI specimens, by type and week, 2026



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 PHF Science and six 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 surveillance and SHIVERS research.

The WHO National Influenza Centre at PHF Science 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 2026

During 1-Jan to 30-August-2026, a total of 3740 influenza viruses were detected and reported through any surveillance system, with influenza A representing 91.1% (3409/3740) and influenza B 8.9% (331/3740) of all influenza viruses (Table 6). Among 1002 subtyped influenza A viruses, 54.6% (547/1002) were A(H3N2) viruses and 45.4% (455/1002) were A(H1N1)pdm09 viruses.

Table 6. Influenza virus identifications by type and sub-type and lineage-typed, 2026

Viruses	All viruses		Influenza A sub-typed	
	N.	Col%	N.	%
Influenza virus	3740	100.0		
Influenza A	3409	91.1	1002	100.0
Influenza A (not sub-typed)	2407	64.4		
Influenza A(H1N1)pdm09	455	12.2	455	45.4
A(H1N1)pdm09 by PCR	429	11.5		
A/Missouri/11/2025 (H1N1)pdm09-like	26	0.7		
Influenza A(H3N2)	547	14.6	547	54.6
A(H3N2) by PCR	490	13.1		
A/Sydney/ 1359/2024 (H3N2)-like	57	1.5		
Influenza B	331	8.9		
Influenza B (not lineage-typed)	323	8.6		
B/Victoria lineage	8	0.2		
B/Victoria lineage by PCR	5	0.1		
B/Austria/1359417/2021-like	3	0.1		
B/Yamagata lineage	0	0.0		
B/Yamagata lineage by PCR	0	0.0		
B/Phuket/3073/2013-like	0	0.0		

Figure 13 shows the influenza virus identifications by type, sub-type, antigenic type and lineage for each week throughout 2026. A(H3N2) is the predominant virus and cocirculated with A(H1N1)pdm09 and influenza B viruses throughout the season. A(H3N2) increased with intensity from July to August.

Figure 13. Total influenza A and B viruses by week specimen taken, 2026

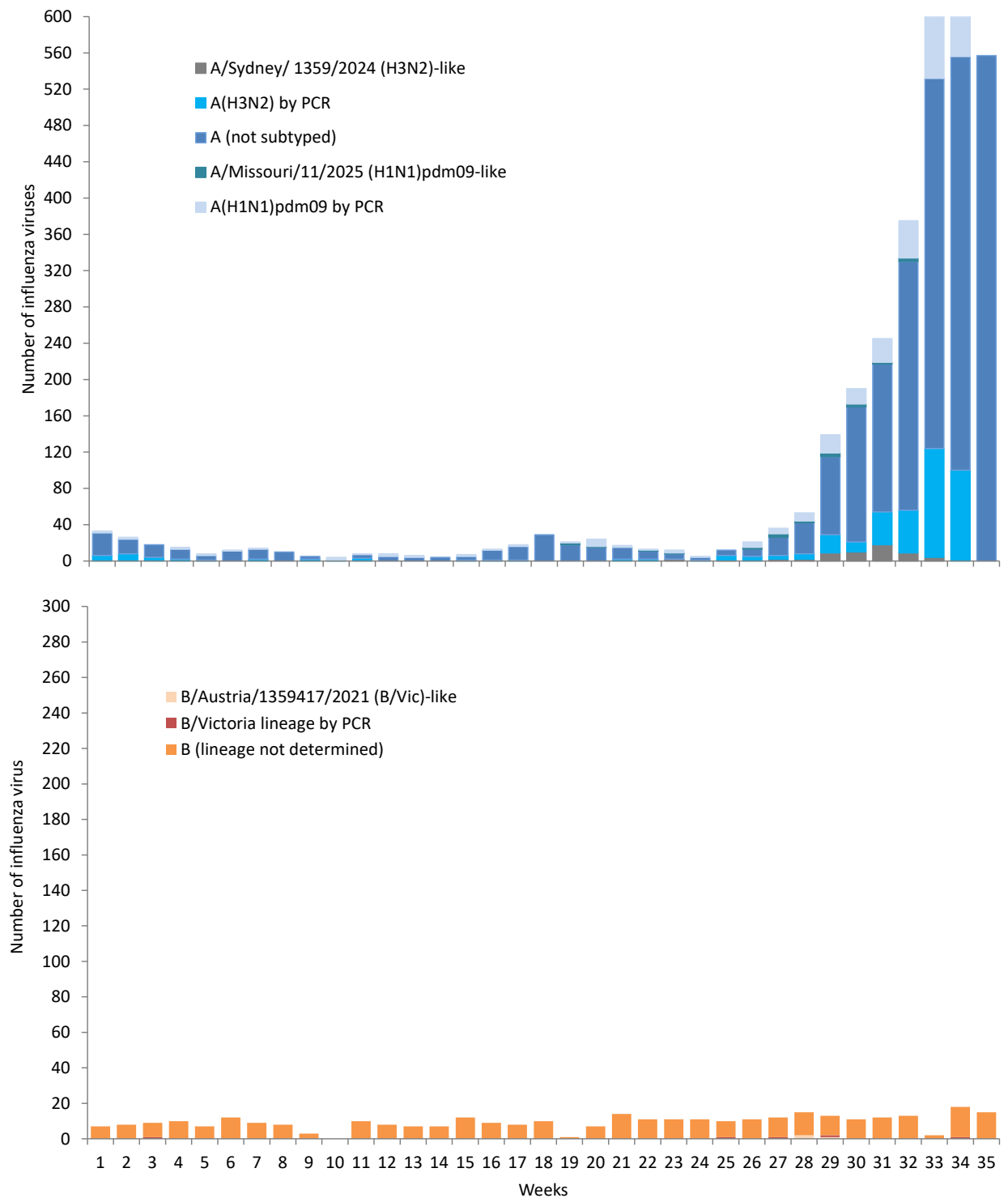


Figure 14 shows the number and percentage of typed influenza viruses from 1997 to 2026. Influenza A is the most frequent predominant influenza type. Of 29 influenza seasons during 1997–2026 excluding 2021 (Note: 2021 – no influenza circulation due to COVID-19 elimination strategy), influenza A predominated in 25 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 14. Influenza viruses by type, 1997–2026

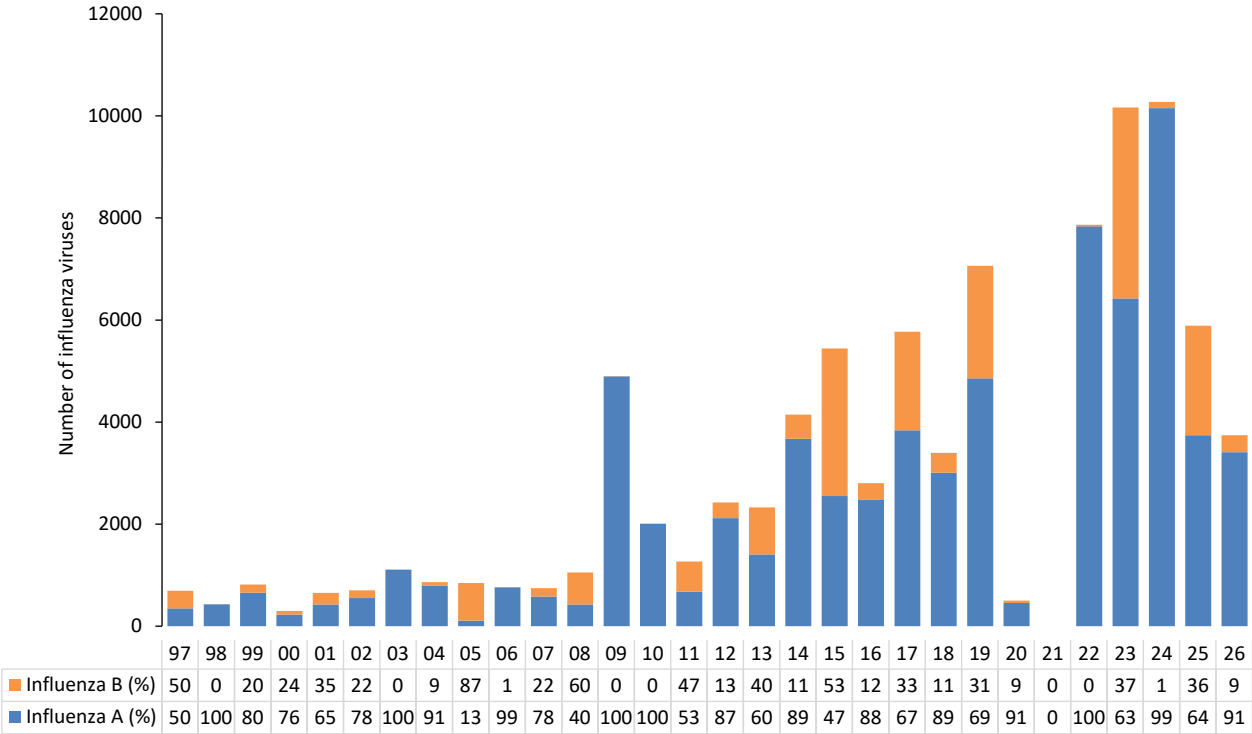


Figure 15 shows the number and percentage of all sub-typed influenza A viruses from 1997 to 2026 (excluding influenza A not sub-typed). Overall, the patterns of the predominant influenza A subtypes among all sub-typed A viruses during 1997–2026 are described below:

- Influenza A(H3N2) strain predominated for 20 seasons [1997-1999 (3), 2002–2008 (7), 2011–2013 (3), 2015–2017 (3), 2019, 2022, 2024, 2026).
- Influenza A(H1N1)pdm09 strain has become the predominant strain for 7 seasons in 2009, 2010, 2014, 2018, 2020, 2023, 2025.
- Seasonal influenza A(H1N1) strain predominated in two seasons (2000 and 2001) with associated relatively low hospitalisations. It has not been detected in New Zealand since 2010.

Figure 15. Influenza A viruses by subtypes 1997–2026

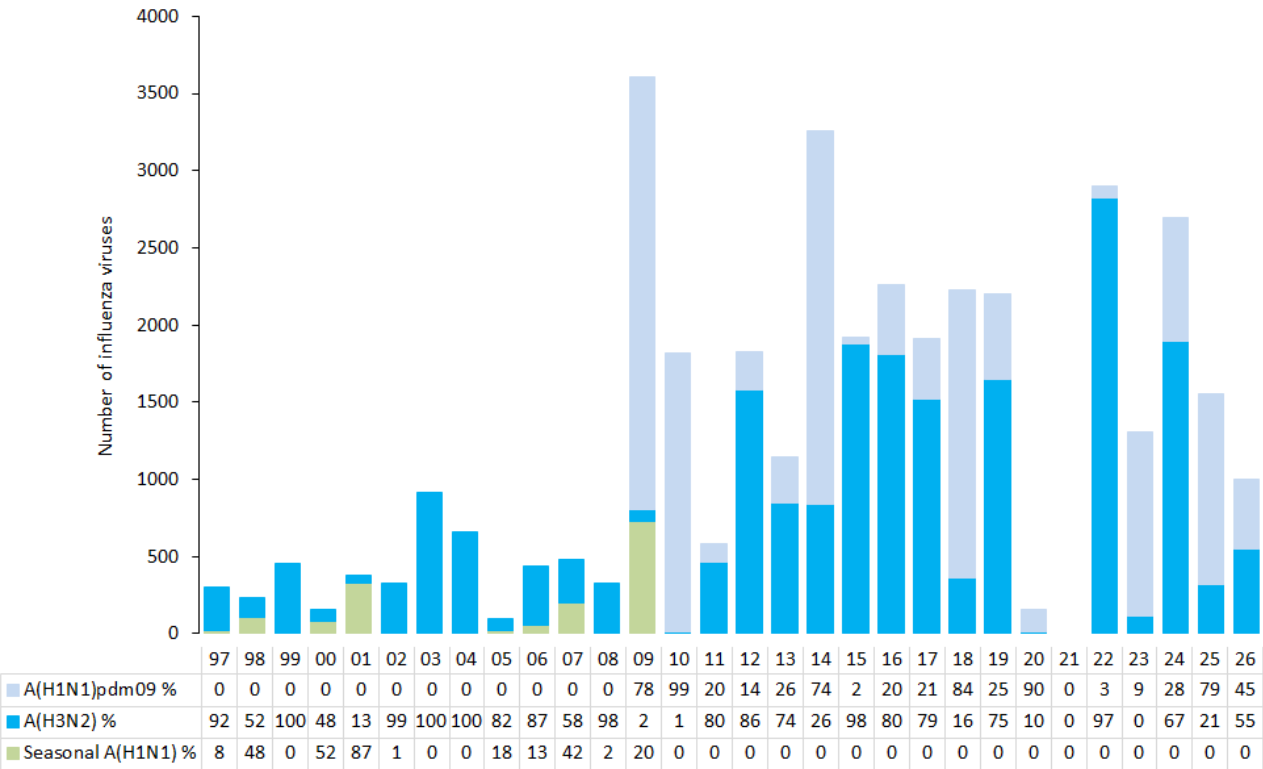
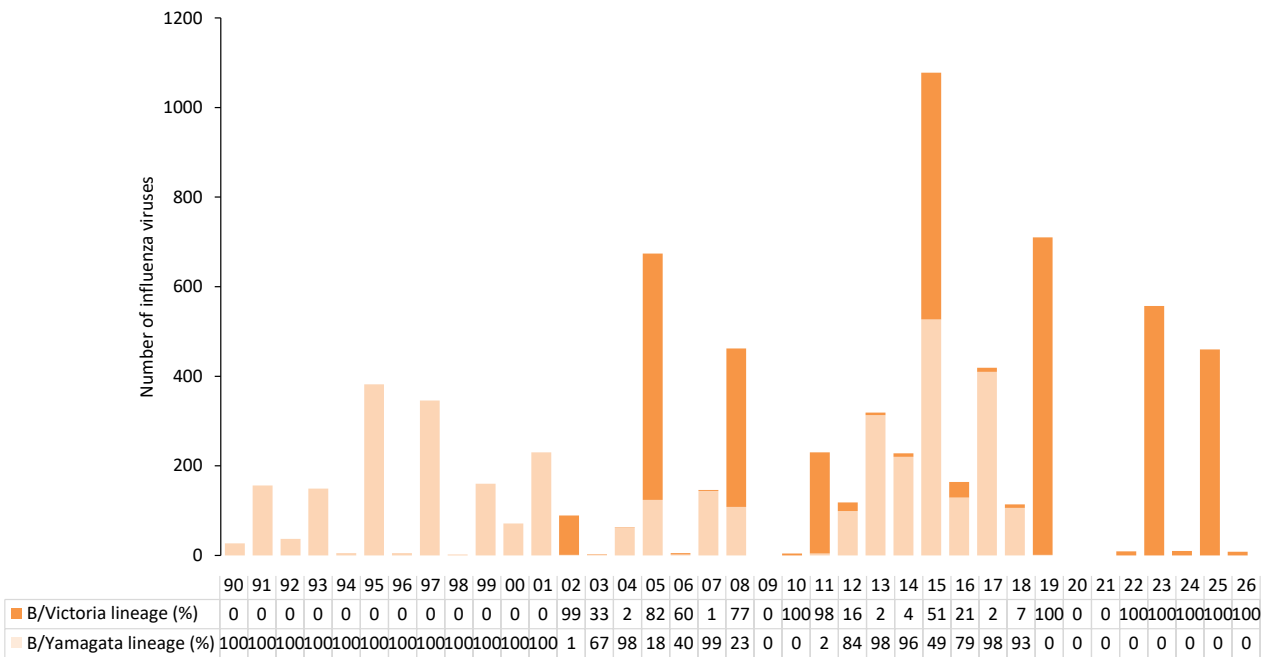


Figure 16 shows the number and percentage of all B viruses from 1990 to 2026 (excluding influenza B not lineage-typed). Overall, the patterns of the predominant influenza B among all lineage-typed B viruses are described below:

- Influenza B/Yamagata lineage: During 1990–2001, Influenza B/Yamagata lineage was the only lineage circulating in New Zealand. Relatively high number of influenza B viruses were recorded in 1995 and 1997. Among 18 influenza seasons during 2002–2019, B/Yamagata lineage viruses predominated over B/Victoria lineage virus for 9 seasons during 2003–2004 (2), 2007 (1), 2012–2014 (3), and 2016–2018 (3). Since 2019, no B/Yamagata lineage virus has been detected in New Zealand.
- B/Victoria lineage: In 2002, B/Victoria lineage viruses were introduced into New Zealand. Since then, 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. During 2012–2019, B/Victoria lineage viruses predominated over the B/Yamagata lineage viruses in every four years (2015 and 2019).

Figure 16. Influenza B viruses by lineages, 1990–2026



4.2 ANTIGENIC AND GENETIC CHARACTERIZATION

4.2.1 Influenza A(H1N1)pdm09

Representative of influenza A(H1N1)pdm09 isolates were antigenically typed at the WHO National Influenza Centre at PHF Science using ferret antisera supplied by the WHO Collaborating Centre (WHOCC). Some of these isolates were also sent to WHOCC-Melbourne. During 1 January to 30 August 2026, a total of 26 influenza A(H1N1)pdm09 isolates were antigenically typed with hemagglutination inhibition assay using antisera raised against A/Missouri/11/2025(H1N1)-like virus. All of them were inhibited well by the antisera against the vaccine strain A/Missouri/11/2025(H1N1). Genetically, most of influenza A(H1N1) viruses fell into subclade D.3.1.1 (Figure 17).

Since February 2026, A(H1N1)pdm09 viruses circulated globally but did not predominate in any regions except for Western Asia.

The haemagglutinin (HA) genes of viruses that were genetically characterized belonged to subclades D.3.1, D.3.1.1, C.1.9 and C.1.9.3. Viruses from subclade D.3.1.1 predominated worldwide, with cocirculation of subclade D.3.1 and very small proportions of subclades C.1.9 and C.1.9.3 being detected in a few countries in the Americas, Eastern Mediterranean and Africa.

During this reporting period, the majority of A(H1N1)pdm09 viruses sequenced globally belonged to subclade D.3.1.1, characterized by R113K, A139D, E283K and K302E. Genetic diversity of the HA in subclade D.3.1.1 viruses increased during this reporting period. Emerging viruses within subclade D.3.1.1 with an additional substitution of G155E increased in recent weeks and belonged to several genetic groups characterized by different combinations of additional amino acid substitutions. Viruses from subclade D.3.1 with substitutions T120A, I372V, I460T and V520A still predominated in South Africa and circulated in minor proportions in some countries in Central America, Africa, Asia and Oceania.

The antigenic properties of A(H1N1)pdm09 viruses were assessed in haemagglutination inhibition (HI) assays with post-infection ferret antisera. HI results for viruses with collection dates since February 2026, showed that post-infection ferret antisera raised against viruses from subclade D.3.1 (e.g., A/Missouri/11/2025) recognized most recently circulating viruses well. However, post-infection ferret antisera raised against viruses from subclades C.1.9.3, D.3.1 and D.3.1.1 showed reductions in recognition of some viruses from subclade D.3.1.1 with additional substitutions including G155E.

Human serology studies used seven serum panels from children (<10 years), adults (18 to 64 years) and older adults (≥65 years) who had received egg-propagated inactivated (standard or adjuvanted) or cell culture-propagated inactivated vaccines with SH 2026 influenza vaccine formulations. SH 2026 egg-based vaccines contained A/Missouri/11/2025 (H1N1)pdm09-like, A/Singapore/GP20238/2024 (H3N2)-like and B/Austria/1359417/2021-like virus antigens. Cell culture-propagated vaccines contained A/Missouri/11/2025 (H1N1)pdm09-like, A/Sydney/1359/2024 (H3N2)-like and B/Austria/1359417/2021-like virus antigens.

Recent A(H1N1)pdm09 viruses with HA genes from subclade D.3.1.1 were analysed in HI assays using these human serum panels. When compared to the post-vaccination geometric mean titers (GMTs) to egg- or cell culture-propagated A/Missouri/11/2025 (H1N1)pdm09-like vaccine reference viruses, post-vaccination GMTs were significantly reduced for some recently circulating viruses from subclade D.3.1.1 with amino acid substitutions Q54H, D139N, G155E, or D139N, V152I, G155E, or D139N, V152I, S157L, S190G in several serum panels.

In summary, since February 2026, A(H1N1)pdm09 viruses circulated globally. The majority of viruses had HA genes belonging to subclades D.3.1 and D.3.1.1, with D.3.1.1 viruses

predominating. Post-infection ferret antisera raised against the southern hemisphere (SH) 2026 and the northern hemisphere (NH) 2026-27 A/Missouri/11/2025-like A(H1N1)pdm09 vaccine viruses recognized most D.3.1 and D.3.1.1 viruses well. In human serology studies, post-vaccination haemagglutination inhibition (HI) geometric mean titres (GMTs) were significantly reduced for some recently circulating A(H1N1)pdm09 viruses when compared to the responses to egg- or cell culture-propagated A/Missouri/11/2025-like vaccine reference viruses.

Based on all of the available data, the WHO consultation recommended to use an egg-propagated or cell culture-propagated A/Missouri/11/2025 (H1N1)pdm09-like as the vaccine strains for use in 2027. The AIVC accepted this recommendation.

(Abridged from WHO website: [Recommended composition of influenza virus vaccines for use in the 2027 southern hemisphere influenza season](#) and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne).

4.2.2 Influenza A(H3N2)

Representative seasonal influenza A(H3N2) isolates were antigenically typed at the WHO National Influenza Centre at PHF Science using HAI typing kit supplied by the WHO Collaborating Centre (WHOCC) in Melbourne. Some of these isolates were also sent to WHOCC-Melbourne. During 1 January to 30 August 2026, a total of 60 influenza A(H3N2) isolates were antigenically typed with hemagglutination inhibition assay using antisera raised against A/Sydney/1359/2024 (H3N2)-like virus. All H3N2 isolates (except one low-reactor) were inhibited well by the antisera against the vaccine strain A/Sydney/1359/2024 (H3N2)-like. Genetically, most of influenza A(H3N2) viruses fell into subclade K (Figure 18).

Since February 2026, A(H3N2) viruses circulated globally and predominated in most transmission zones, including Oceania, South-east, Southern and Western Asia.

Phylogenetic analysis of the HA gene sequences of A(H3N2) viruses collected since February 2026 showed that the majority of circulating viruses globally belonged to subclade K, (characterized by K2N, T135K, S144N [a potential addition of an N-glycosylation site], N158D, I160K, Q173R, T328A and S378N). There were still low proportions of viruses circulating from J.2 subclades, notably J.2.2 (characterized by S124N) in Asia, J.2.3 (characterized by N158K, K189R and S378N) in the Americas, J.2.4 (characterized by T135K [a potential loss of an N-glycosylation site] and K189R) in Asia and J.2.4.2 (characterized by F79V, T135K, S144N [a potential addition of an N-glycosylation site], N158D, I160K and T328A) in most regions.

Post-infection ferret antisera raised against cell culture-propagated A/Sydney/1359/2024-like and egg-propagated A/Singapore/GP20238/2024-like J.2.4 viruses, representing the A(H3N2) component for the SH 2026 influenza vaccines, recognized most J.2.4 viruses and many subclade J.2.4.2 and K viruses well. However, subclade J.2.4.2 and K viruses were better recognized by post-infection ferret antisera raised against cell culture-propagated A/Darwin/1415/2025-like and egg-propagated A/Darwin/1454/2025-like (subclade K) viruses.

Human serology studies were conducted using the serum panels as described above by HI and virus neutralization (VN) assays with recently circulating A(H3N2) viruses with HA genes from subclades J.2.3, J.2.4.2 and K. When compared to titres against cell-propagated A/Sydney/1359/2024-like and egg-propagated A/Singapore/GP20238/2024-like vaccine reference viruses, post-vaccination HI GMTs or VN GMTs against many of the recent viruses in J.2.3, J.2.4.2 and K subclades tested were significantly reduced in many serum panels.

In summary, since February 2026, A(H3N2) viruses circulated and predominated in many regions. The vast majority of A(H3N2) viruses analysed had HA genes from subclade K. Post-infection ferret antisera raised against SH 2026 influenza season A(H3N2) vaccine viruses (cell culture-

propagated A/Sydney/1359/2024-like and egg-propagated A/Singapore/GP20238/2024-like J.2.4 viruses) recognized subclade J.2.4 and many subclade J.2.4.2 and K viruses well; however, post-infection ferret antisera raised against NH 2026-27 A(H3N2) subclade K vaccine viruses (cell culture-propagated A/Darwin/1415/2025-like and egg-propagated A/Darwin/1454/2025-like viruses) showed improved recognition of subclade J.2.4.2 and K viruses. When compared to titres against cell culture-propagated A/Sydney/1359/2024-like and egg-propagated A/Singapore/GP20238/2024-like vaccine reference viruses, human post-vaccination HI GMTs or virus neutralization (VN) GMTs against many of the recent viruses in subclades J.2.3, J.2.4.2 and K were significantly reduced.

Based on all available data, the WHO Consultative Group recommended to use an egg- or cell culture-propagated A/Darwin/1454/2025 (H3N2)-like strain as the vaccine strains for use in 2027. AIVC accepted this recommendation.

(Abridged from WHO website: [Recommended composition of influenza virus vaccines for use in the 2027 southern hemisphere influenza season](#) and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne).

4.2.3 Influenza B

Representative seasonal influenza B isolates were antigenically typed at the WHO National Influenza Centre at PHF Science using HAI typing kit supplied by the WHO Collaborating Centre (WHOCC). Some of these isolates were also sent to WHOCC-Melbourne. During 1 January to 30 August 2026, a total of 3 influenza B/Victoria-lineage isolates were antigenically typed with hemagglutination inhibition assay using antisera raised against B/Austria/1359417/2021-like virus. All B/Victoria-lineage isolates were inhibited well by the antisera against the vaccine strain B/Austria/1359417/2021. No sequences of B/Victoria lineage viruses were available for reporting.

Since February 2026, influenza B viruses were detected in all WHO regions, and all those characterized belonged to the B/Victoria/2/87 lineage. There have been no confirmed detections of circulating B/Yamagata lineage viruses after March 2020.

All influenza B/Victoria lineage viruses collected since February 2026 with available HA sequences belonged to subclades C.3 and C.5. Viruses which shared a D197E substitution are classified as subclade C.5 which has undergone further diversification of HA genes resulting in subclades C.5.1 (characterized by E183K), C.5.6 (characterized by D129N), C.5.6.1 (characterized by T37I, E128D, and T199A in addition to D129N), and C.5.7 (characterized by E183K and E128G). Circulating viruses derived from subclade C.3 (characterized by E128K and A154E) include subclades C.3.1 (characterized by D197N with potential gain of N-glycosylation) and C.3.3 (characterized by substitutions S208P, S255P, I267V, and D197N with potential gain of N-glycosylation). There were differences in subclade circulation and predominance between and within regions based on currently available data. Subclade C.3.1 viruses predominated in the Americas, subclade C.3.3 viruses predominated in East Asia and subclade C.5.6 predominated in Africa, Europe, South East Asia and Oceania.

Antigenic analysis showed that post-infection ferret antisera raised against B/Austria/1359417/2021-like viruses (subclade C), representing the B component for the SH 2026 influenza vaccines, recognized viruses within the C.5 subclades well. In contrast, viruses with the HA substitution D197N from C.3 subclades were recognized poorly. Post-infection ferret antisera raised against cell culture-propagated B/Pennsylvania/14/2025-like viruses (C.3.1), representing cell-based vaccine viruses for the NH 2026-27 influenza season, recognized recently circulating viruses from C.3 and C.5 subclades well. Post-infection ferret antisera raised against egg-propagated B/Tokyo/EIS13-175/2025-like viruses (C.3.1), representing egg-based vaccine viruses for the NH 2026-27 influenza season, recognized most viruses from the C.5 subclades well but

showed reduced recognition of C.3 subclades compared to that of the cell equivalent.

In human serology studies, recently circulating B/Victoria lineage viruses with HA genes from subclades C.3.1, C.3.3, C.5.6 and C.5.6.1 were tested using the serum panels described above. When compared to titres against egg- or cell culture-propagated B/Austria/1359417/2021-like vaccine reference viruses, titres against many viruses with HA genes from subclades C.5.6 and C.5.6.1 were not significantly reduced in most serum panels; however, titres against subclade C.5.6.1 viruses with the amino acid substitution P241Q were significantly reduced in some panels. In contrast, titres against many viruses with HA genes from subclades C.3.1 and C.3.3 were significantly reduced in most serum panels.

In summary, since February 2026, influenza B viruses co-circulated globally but at different levels among regions. All influenza B viruses characterized belonged to the B/Victoria/2/87 lineage and had HA genes belonging to subclades C.3 and C.5. Post-infection ferret antisera raised against B/Austria/1359417/2021-like subclade C viruses, representing the B component of vaccine viruses for the SH 2026 influenza season, recognized viruses within the C.5 subclades (C.5.6, C.5.6.1, C.5.7) well, but C.3 subclade viruses (C.3.1, C.3.3) with HA substitution D197N were recognized poorly. Post-infection ferret antisera raised against cell culture-propagated NH 2026-27 vaccine viruses (B/Pennsylvania/14/2025-like C.3.1 viruses) recognized recently circulating C.3 and C.5 subclades well. Egg-propagated NH vaccine viruses (B/Tokyo/EIS13-175/2025-like C.3.1 viruses) acquired egg-adaptive mutations that removed the potential N-glycosylation site at HA 197 to 199, and post-infection ferret antisera raised against these viruses showed reduced recognition of viruses from C.3 subclades compared to that of the cell equivalent. Human serology assays showed that post-vaccination titres against most recent B/Victoria lineage viruses with HA genes from C.5 subclades were not significantly reduced when compared to titres against egg- or cell culture-propagated B/Austria/1359417/2021-like vaccine reference viruses. Titres against viruses with HA genes from C.3 subclades were significantly reduced in most serum panels.

Based on all available data, the WHO Consultative Group recommended to use an egg-propagated B/Tokyo/EIS13-175/2025 and cell culture-propagated B/Pennsylvania/14/2025-like strain for use in 2027. AIVC accepted this recommendation.

(Abridged from WHO website: [Recommended composition of influenza virus vaccines for use in the 2027 southern hemisphere influenza season](#) and a report to AIVC by Dr Ian Barr, WHO Collaborating Centre for Influenza, Melbourne).

Figure 17. Phylogenetic relationships among influenza A(H1N1)pdm09 virus haemagglutinin gene

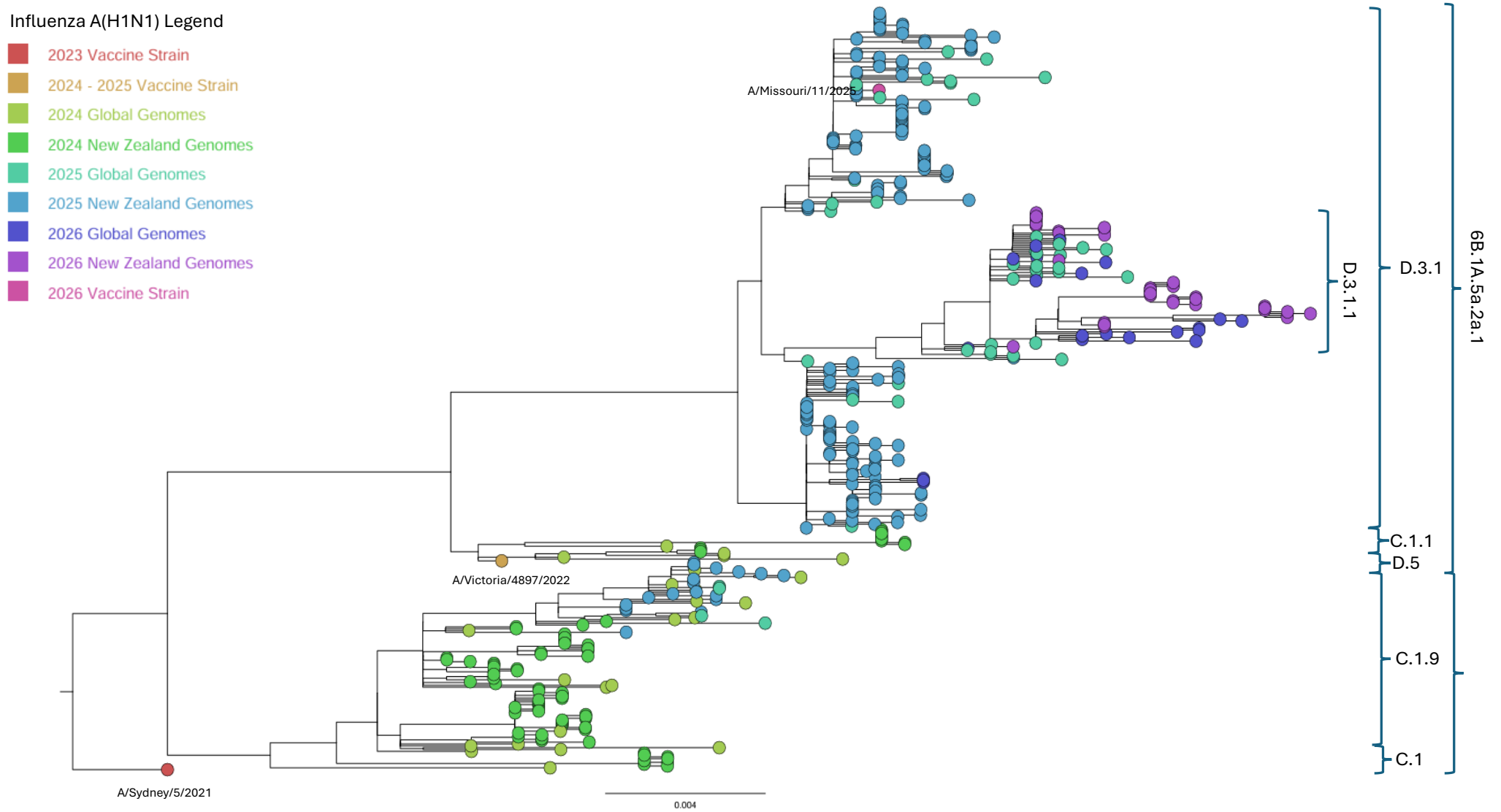
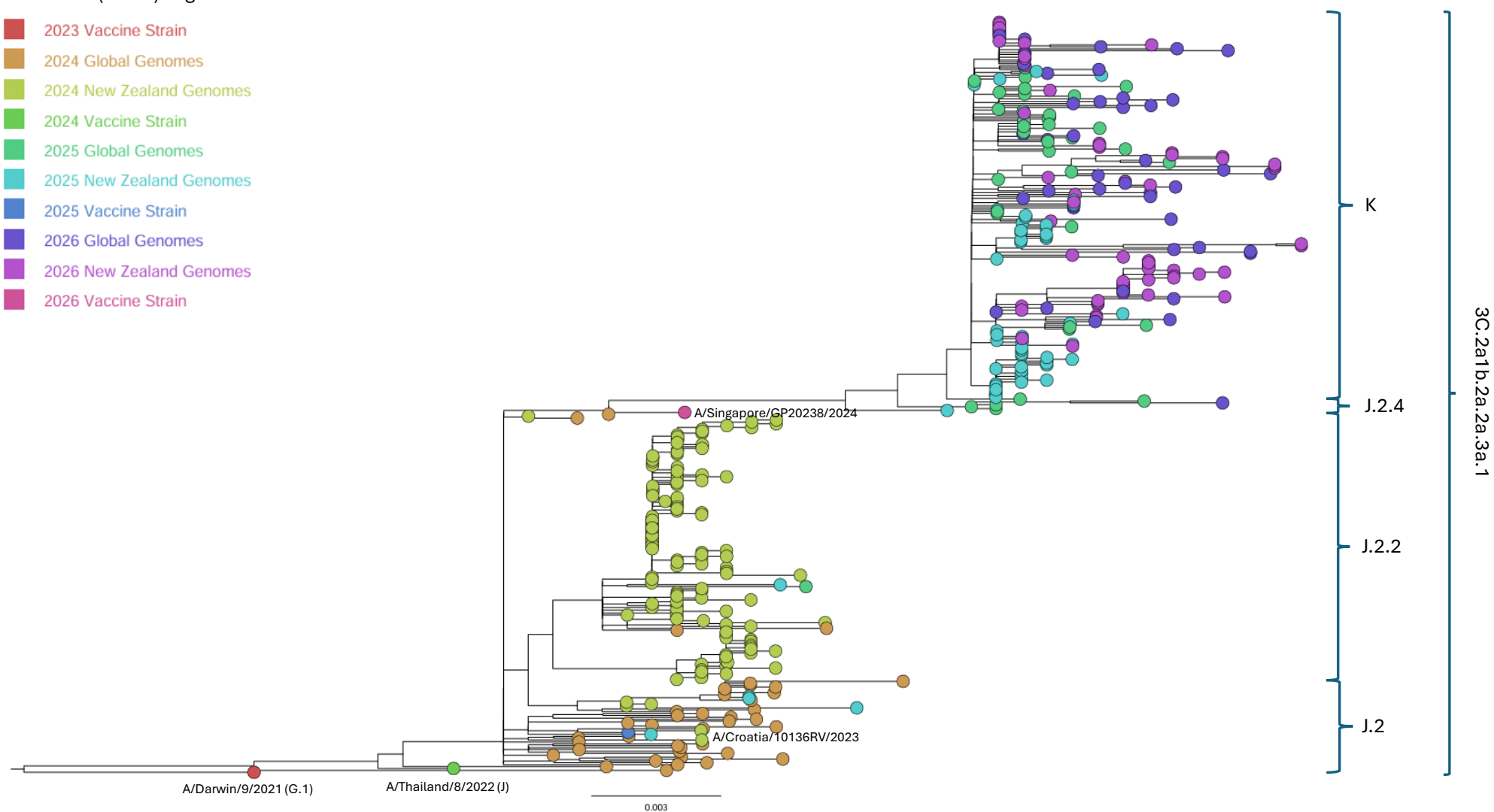


Figure 18. Phylogenetic relationships among influenza A(H3N2) virus haemagglutinin gene

Influenza A(H3N2) Legend

- 2023 Vaccine Strain
- 2024 Global Genomes
- 2024 New Zealand Genomes
- 2024 Vaccine Strain
- 2025 Global Genomes
- 2025 New Zealand Genomes
- 2025 Vaccine Strain
- 2026 Global Genomes
- 2026 New Zealand Genomes
- 2026 Vaccine Strain



4.3 ANTIVIRAL RESISTANCE

The WHO National Influenza Centre at PHF employed a phenotypic method (fluorometric neuraminidase inhibition assay) for the surveillance of anti-viral drug resistance in influenza viruses. In addition, NIC at PHF 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 2026, fluorometric neuraminidase inhibition assay was used to test 88 influenza viruses against oseltamivir and 86 influenza viruses against zanamivir. The preliminary results showed that all were sensitive to both oseltamivir and zanamivir (Tables 7 & 8).

Table 7. Antiviral susceptibility to oseltamivir for influenza viruses, 2019–2026[^]

Influenza	NA inhibition to Oseltamivir*	Fold change in IC50 of test viruses (No. of viruses)**							
		2019	2020	2021	2022	2023	2024	2025	2026
A(H1N1)pdm09	Normal	0-3 (20)	5	1	0-3 (16)	0-6 (360)	0-3 (112)	0-3 (45)	0-3 (26)
	Reduced	-	-	-	-	-	-	-	-
	Highly reduced	-	-	-	-	-	-	-	-
A(H3N2)	Normal	0-4 (75)		3	0-4 (442)	0-2 (54)	0-2 (107)	-	0-1 (59)
	Reduced	-	-	-	-	-	-	-	-
	Highly reduced	-	-	-	-	-	-	-	-
Influenza B	Normal	0-5 (226)		2	0-1 (2)	0-2 (280)	0-2 (9)	0-2 (127)	1-3 (3)
	Reduced	28-29 (1)	-	-	-	10-10 (1)	-	-	-
	Highly reduced	-	-	-	-	-	-	-	-

Table 8. Antiviral susceptibility to zanamivir for influenza viruses, 2019–2026[^]

Influenza	NA inhibition to Zanamivir*	Fold change in IC50 of test viruses (No. of viruses)**							
		2019	2020	2021	2022	2023	2024	2025	2026
A(H1N1)pdm09	Normal	0-5 (22)	5	1	0-2 (15)	0-3 (361)	0-6 (112)	0-2 (45)	0-3 (26)
	Reduced	15-16 (1)	-	-	-	-	-	-	-
	Highly reduced	-	-	-	-	-	-	-	-
A(H3N2)	Normal	0-6 (75)		3	0-3 (442)	0-2 (54)	0-3 (107)	-	0-4 (57)
	Reduced	-	-	-	-	-	-	-	-
	Highly reduced	-	-	-	-	-	-	-	-
Influenza B	Normal	0-2 (228)		2	0-2 (2)	0-3 (279)	0-1 (9)	0-3 (127)	0-1 (3)
	Reduced	10-11 (1)	-	-	-	12-13 (1)	-	-	-
	Highly reduced	-	-	-	-	-	-	-	-

Note: For Tables above:

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)

5. INFLUENZA VACCINE EFFECTIVENESS

In New Zealand seasonal quadrivalent 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 vaccine effectiveness (VE). VE was estimated from the odds ratio (OR) comparing vaccination among patients with laboratory-confirmed influenza versus patients who test negative by RT-PCR for influenza, where $VE = (1 - OR_{adj}) \times 100\%$. Odds ratios were estimated using logistic regression models where influenza test status was the dependent (i.e. outcome) variable and vaccination status was the main effect variable of interest. Logistic regression allows adjustment for other variables that may influence the estimate, such as age category, and calendar time. VE is interpreted as the relative reduction in odds of medically-attended influenza (either outpatient or hospital) or non-medically-attended influenza (such as cohort participants) among vaccinated vs. unvaccinated individuals. VE of 0% represents no protection and VE of 100% represents complete protection. The method for calculating VE allows the point estimate to be less than 0. A negative estimate suggests a lack of protection. Estimates are reported with a 95% confidence interval. Confidence intervals will generally be wider when the sample size is lower. For an estimate to have narrow confidence intervals, a substantially larger sample may be needed. We have excluded any estimates where the confidence interval spanned more than 150.

We estimated the effectiveness of seasonal inactivated influenza vaccine in preventing laboratory-confirmed influenza among patients hospitalised with severe acute respiratory infections (SARI), among GP-consultation seeking patients with influenza-like illness, and among WellKiwis participants with an acute respiratory illness (ARI) 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 ARI, 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.

For influenza-confirmed SARI cases among acutely admitted hospital patients, the estimated adjusted vaccine effectiveness (VE) was 46% (95% CI: 30, 58) (Table 9). For influenza-confirmed ILI cases among GP consultation patients, the estimated adjusted vaccine effectiveness (VE) was 58% (95% CI: 37,72). For influenza-confirmed ARI among cohort participants, the estimated adjusted vaccine effectiveness (VE) was 45% (95% CI: 29, 57).

Table 9. Estimated influenza vaccine effectiveness against medically and non-medically attended influenza infections, 2026

Outcome Measure	Virus/Age Group		Influenza Positive		Influenza Negative		Crude	Adjusted*
			Vaccinated Yes	Vaccinated No	Vaccinated Yes	Vaccinated No	VE% (95%CI)	VE% (95%CI)
Hospital SARI	Influenza	All ages	110	378	469	1016	37 (20; 51)	46 (30; 58)
		0-17 years	6	183	81	540	78 (49; 92)	NA
		18-64 years	30	100	101	275	18 (-33; 51)	NA
		65+ years	74	95	287	201	45 (21; 62)	NA
	Influenza A	All ages	107	373	476	1029	38 (21; 52)	47 (31; 59)
		0-17 years	6	180	81	547	77 (48; 92)	NA
		18-64 years	29	98	103	278	20 (-30; 52)	NA
		65+ years	72	95	292	204	47 (23; 63)	NA
GP ILI	Influenza	All ages	150	36	522	316	60 (41; 74)	58 (37; 72)
		0-17 years	55	3	146	32	75 (15; 95)	NA
		18-64 years	87	20	334	195	61 (33; 78)	NA
		65+ years	8	13	42	89	NA	NA
	Influenza A	All ages	149	36	524	315	60 (40; 74)	57 (36; 72)
		0-17 years	55	3	146	32	75 (15; 95)	NA
		18-64 years	86	20	336	194	60 (31; 77)	NA
		65+ years	8	13	42	89	NA	NA
WellKiwis cohort	Influenza	All ages	131	199	791	604	50 (36; 61)	45 (29; 57)
		0-17 years	56	142	277	394	44 (21; 60)	N/A
		18-64 years	67	57	463	207	47 (22; 64)	N/A
		65+ years	8	0	51	3	N/A	N/A
	A(H1N1)	All ages	43	48	879	755	23 (-17; 50)	19 (-26; 48)
		0-17 years	15	34	318	502	30 (-30; 63)	N/A
		18-64 years	27	14	503	250	NA	N/A
		65+ years	1	0	58	3	NA	N/A
	A(H3N2)	All ages	51	94	871	709	56 (37; 69)	45 (20; 62)
		0-17 years	28	76	305	460	44 (12; 65)	N/A
		18-64 years	20	18	510	246	46 (-3; 72)	N/A
		65+ years	3	0	56	3	N/A	N/A

*Adjusted for age

N/A: not applicable as numbers too low to reach any significance; CI: Confidence interval; ILI: Influenza-like illness; ARI: acute respiratory illness; SARI: severe acute respiratory infections. N/A cells indicate not available because of large confidence intervals.

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