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SURVEILLANCE REPORT

Influenza Genomic Surveillance in Ontario: 2026–27 Early Season

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Introduction

This report summarizes the results of influenza whole genome sequencing completed by Public Health Ontario (PHO) and the Ontario COVID-19 Genomics Network (OCGN) for the beginning of the 2026–27 influenza season. The 2025–26 season report can be found on PHO's website.

Highlights

- A total of 229 specimens were included for the current season from August 30 to October 10, 2026 (surveillance weeks 35 to 40), representing 21.1% of specimens that tested positive for influenza within Ontario. All specimens sequenced were influenza A, there were no eligible influenza B specimens.
- Of the 229 influenza A specimens sequenced from the current season, 226 (98.7%) were H1N1pdm09 and three (1.3%) were seasonal H3N2.
    - All 226 H1N1pdm09 specimens (98.7%) were genetic subclade D.3.1.1 (clade 5a.2a.1). The H1N1pdm09 component of the 2026–2027 Northern Hemisphere influenza vaccine belongs to the genetic subclade D.3.1 (clade 5a.2a.1). The genetic subclade D.3.1.1 is genetically related to D.3.1. (see Figure 2a).
    - All three H3N2 specimens (1.3%) were genetic subclade K (clade 2a.3a.1). The H3N2 component of the 2026–2027 Northern Hemisphere influenza vaccine belongs to the genetic subclade K (clade 2a.3a.1).
- One specimen (0.4%) of the influenza A H1N1pdm09 specimens sequenced had the H275Y amino acid substitution in the neuraminidase (NA) gene, which is associated with resistance to oseltamivir (see Table 8).
- Of the 183 influenza A specimens sequenced that met quality control criteria for the polymerase acidic (PA) gene, none had the I38T amino acid substitution in the PA gene associated with resistance to baloxavir.
- Of the 226 influenza A H1N1pdm09 specimens sequenced, 213 (94.2%) had a G155E amino acid substitution in the hemagglutinin (HA) protein. Preliminary studies have suggested that this substitution may result in reduced neutralizing antibody response, though the Public Health Agency of Canada thus far has not reported differences between circulating H1N1pdm09 viruses and the current vaccine strain based on antigenic characterization as of October 30, 2026 (see Appendix Table B).[1-3]

Background

Two types of influenza virus are responsible for most human cases during the influenza season, influenza A and B. Influenza A can be further classified into subtypes (e.g., H1N1pdm09, seasonal H3N2) while influenza B can be further classified into lineages (i.e., Victoria). As influenza spreads through populations, changes can occur to the virus' genome. The accumulation of these changes or mutations can result in new subdivisions beyond subtypes or lineages called clades and subclades. Although many subclades will have no differences in the ability to cause disease, some may have mutations that affect virulence, transmissibility, antiviral susceptibility, or allow the virus to escape natural or vaccine-induced immunity. Genomic surveillance uses whole genome sequencing to monitor these changes in the genome as a virus evolves over time. This allows public health professionals to provide context to the current season, assess whether antivirals are working against the currently circulating viruses, and advise on vaccine strains for the upcoming seasons.[4] For the 2026–2027 influenza season, publicly funded vaccines available in Ontario are trivalent including influenza A H1N1pdm09 subclade D.3.1, influenza A H3N2 subclade K, and influenza B Victoria subclade C.3.1.[3,5]

To understand the diversity of influenza viruses circulating early in the 2026–27 season, whole genome sequencing (WGS) was performed on all eligible positive influenza specimens from Public Health Ontario (PHO), the Ontario COVID-19 Genomics Network (OCGN), and the Sentinel Practitioner Surveillance Network (SPSN).[6]

Sequences were processed using bioinformatics analyses to assign clades, subclades, and to identify mutations (See Technical Notes for details).

For the 2026–27 influenza early season, genomic surveillance was expanded beyond routine PHO specimens to include Ontario specimens from the Sentinel Practitioner Surveillance Network (SPSN) and specimens sequenced by the Ontario COVID-19 Genomics Network (OCGN).

Results

Table 1: Number of Positive Influenza Specimens, Number and Percentage Sequenced, Ontario, August 30 to October 10, 2026

Month	Number of Positive Specimens	Number Sequenced	Percentage Sequenced
August*	19	7	36.8%
September	708	169	23.9%
October*	358	53	14.8%
Total	**1,085**	**229**	**21.1%**

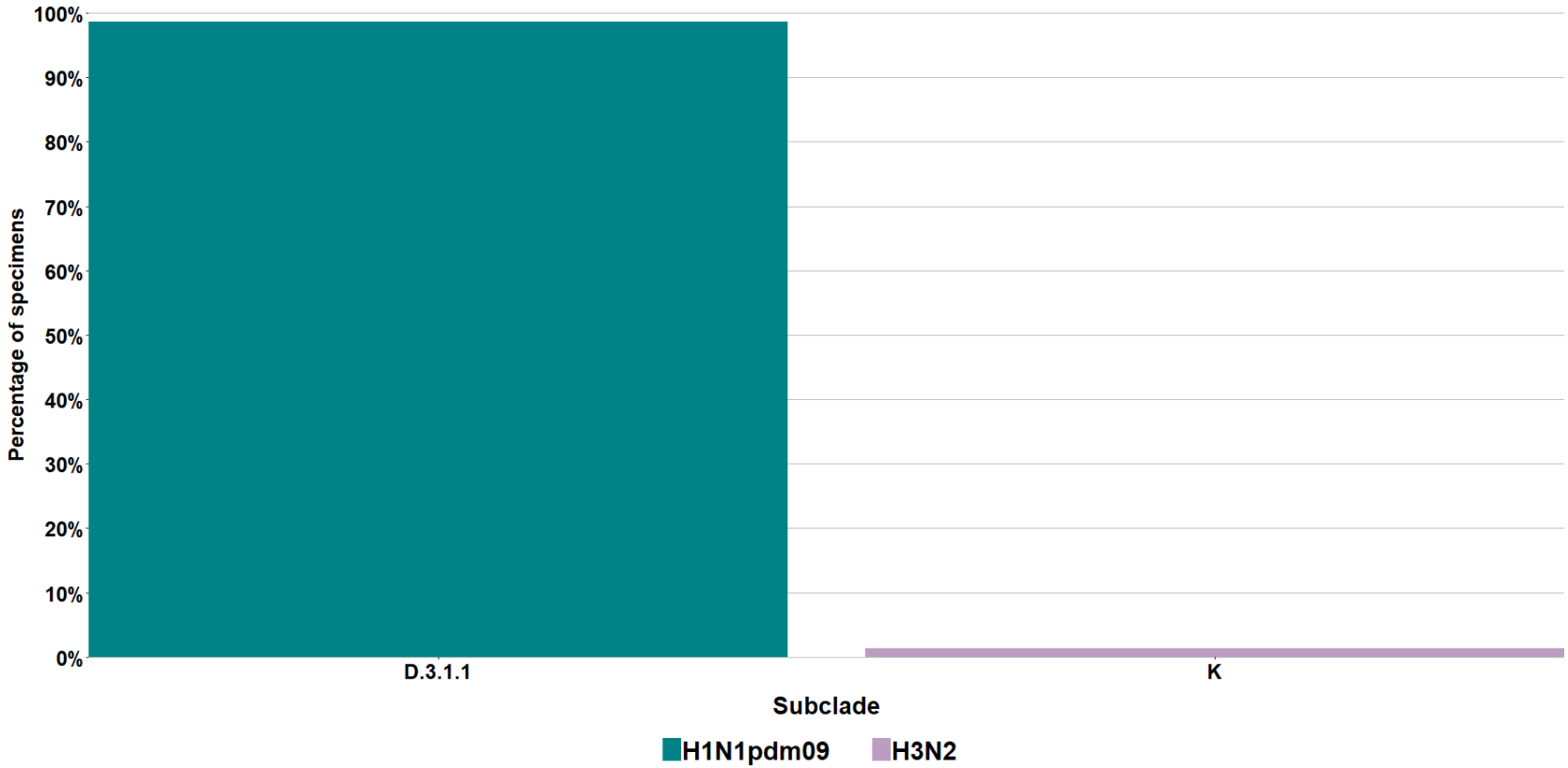
Note: *August and October are partial months. 'Number of Positive Specimens' includes specimens that tested positive for influenza in Ontario as reported in the Ontario Respiratory Virus Tool.[7] 'Number Sequenced' includes all specimens from PHO, the OCGN, and the SPSN that tested positive for influenza and were eligible for sequencing. See Technical Notes for WGS eligibility criteria.

Table 2: Number and Percentage of Positive Influenza A Specimens Sequenced by Genetic Characterization, Ontario, August 30 to October 10, 2026

Genetic Characterization	Routine PHO Specimens	OCGN Specimens	SPSN Specimens	Total
H1N1pdm09	**139 (99.3%)**	**67 (98.5%)**	**20 (95.2%)**	**226 (98.7%)**
D.3.1.1 (5a.2a.1)	139 (99.3%)	67 (98.5%)	20 (95.2%)	226 (98.7%)
H3N2	**1 (0.7%)**	**1 (1.5%)**	**1 (4.8%)**	**3 (1.3%)**
K (2a.3a.1)	1 (0.7%)	1 (1.5%)	1 (4.8%)	3 (1.3%)
Total Sequenced	**140 (100%)**	**68 (100%)**	**21 (100%)**	**229 (100%)**

Note: 'Routine PHO Specimens' include specimens tested according to Public Health Ontario's influenza respiratory testing algorithm and sequenced by PHO. 'OCGN Specimens' include specimens tested and sequenced by the OCGN laboratories. 'SPSN Specimens' include specimens collected by the SPSN and sequenced at PHO. The influenza A genetic subclades included in the 2026–27 season's influenza vaccine are H1N1pdm09 subclade D.3.1 and influenza A H3N2 subclade K.[3]

Figure 1: Percentage of Positive Influenza A Specimens Sequenced, by Genetic Characterization, Ontario, August 30 to October 10, 2026



Note: The influenza A genetic subclades included in the 2026–27 season's influenza vaccine are H1N1pdm09 subclade D.3.1 and influenza A H3N2 subclade K.[3]

Table 3: Number and Percentage of Positive Influenza A Specimens Sequenced, by Genetic Characterization and Month, Ontario, August 30 to October 10, 2026

Genetic Characterization	August*	September	October*	Total
H1N1pdm09	**7 (100%)**	**166 (98.2%)**	**53 (100%)**	**226 (98.7%)**
D.3.1.1	7 (100%)	166 (98.2%)	53 (100%)	226 (98.7%)
H3N2	**0 (0.0%)**	**3 (1.8%)**	**0 (0.0%)**	**3 (1.3%)**
K	0 (0.0%)	3 (1.8%)	0 (0.0%)	3 (1.3%)
Total Sequenced	**7 (100%)**	**169 (100%)**	**53 (100%)**	**229 (100%)**

Note: *August and October are partial months. The influenza A genetic subclades included in the 2026–27 season's influenza vaccine are H1N1pdm09 subclade D.3.1 and influenza A H3N2 subclade K.[3]

Table 4: Number and Percentage of Positive Influenza A Specimens Sequenced, by Genetic Characterization and Age Group, Ontario, August 30 to October 10, 2026

Genetic Characterization	0–4 Years	5–17 Years	18–64 Years	65 Years and Over	Total
H1N1pdm09	**28 (100%)**	**15 (100%)**	**44 (93.6%)**	**139 (100%)**	**226 (98.7%)**
D.3.1.1	28 (100%)	15 (100%)	44 (93.6%)	139 (100%)	226 (98.7%)
H3N2	**0 (0.0%)**	**0 (0.0%)**	**3 (6.4%)**	**0 (0.0%)**	**3 (1.3%)**
K	0 (0.0%)	0 (0.0%)	3 (6.4%)	0 (0.0%)	3 (1.3%)
Total Sequenced	**28 (100%)**	**15 (100%)**	**47 (100%)**	**139 (100%)**	**229 (100%)**

Note: Age was assigned based on the date of birth provided on the test requisition; specimens with a missing date of birth were excluded. Age groups were updated to align with the Ontario Respiratory Virus Tool. The influenza A genetic subclades included in the 2026–27 season's influenza vaccine are H1N1pdm09 subclade D.3.1 and H3N2 subclade K.[3]

Table 5: Number and Percentage of Positive Influenza A Specimens Sequenced, by Genetic Characterization and Setting, Ontario, August 30 to October 10, 2026

Genetic Characterization	Intensive Care Unit	Hospital/Emergency Department	Congregate Living	Other/ No Setting Reported	Primary Care (SPSN)	Total
H1N1pdm09	**5 (100%)**	**104 (98.1%)**	**64 (100%)**	**33 (100%)**	**20 (95.2%)**	**226 (98.7%)**
D.3.1.1	5 (100%)	104 (98.1%)	64 (100%)	33 (100%)	20 (95.2%)	226 (98.7%)
H3N2	**0 (0.0%)**	**2 (1.9%)**	**0 (0.0%)**	**0 (0.0%)**	**1 (4.8%)**	**3 (1.3%)**
K	0 (0.0%)	2 (1.9%)	0 (0.0%)	0 (0.0%)	1 (4.8%)	3 (1.3%)
Total Sequenced	**5 (100%)**	**106 (100%)**	**64 (100%)**	**33 (100%)**	**21 (100%)**	**229 (100%)**

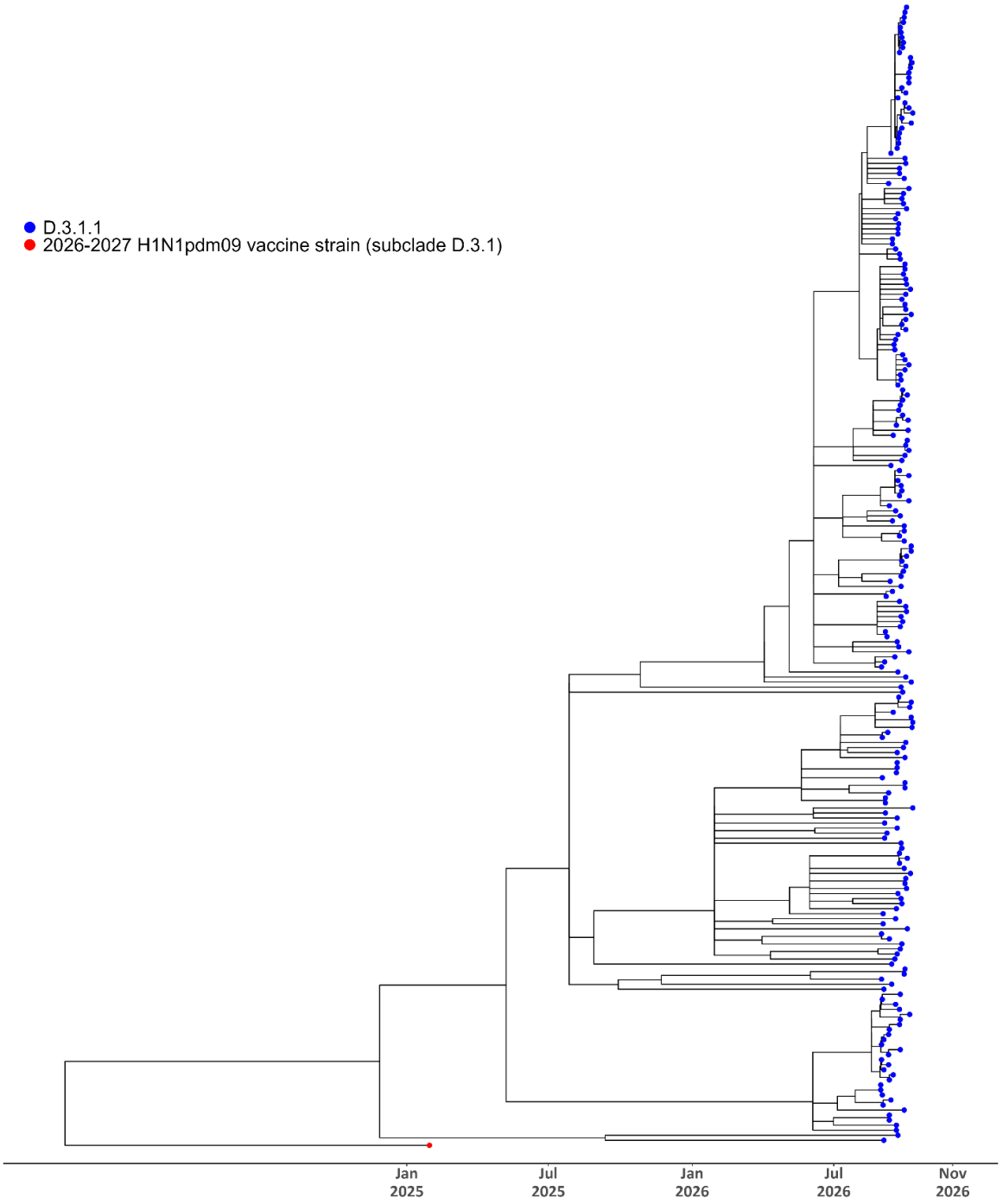
Note: Setting represents the health care facility at which an individual received care. PHO and OCGN specimens were categorized into four settings: 'Intensive Care Unit', 'Hospital/Emergency Department', 'Congregate Living', or 'Other/No Setting Reported'. Congregate living includes long-term care homes, retirement homes, correctional facilities, and undefined institutions (excluding hospitals). 'Primary Care (SPSN)' were restricted to specimens tested as part of the Sentinel Surveillance Practitioner Network (SPSN). The influenza A genetic subclades included in the 2026-27 season's influenza vaccine are H1N1pdm09 subclade D.3.1 and influenza A H3N2 subclade K.[3]

Table 6: Number and Percentage of Positive Influenza A Specimens Sequenced, by Genetic Characterization and Region, Ontario, August 30 to October 10, 2026

Genetic Characterization	Northern	Eastern	Central East	Toronto	South West	Central West	Total
H1N1pdm09	**0 (0.0%)**	**23 (100%)**	**68 (100%)**	**89 (98.9%)**	**25 (96.2%)**	**21 (95.5%)**	**226 (98.7%)**
D.3.1.1	0 (0.0%)	23 (100%)	68 (100%)	89 (98.9%)	25 (96.2%)	21 (95.5%)	226 (98.7%)
H3N2	**0 (0.0%)**	**0 (0.0%)**	**0 (0.0%)**	**1 (1.1%)**	**1 (3.8%)**	**1 (4.5%)**	**3 (1.3%)**
K	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (1.1%)	1 (3.8%)	1 (4.5%)	3 (1.3%)
Total Sequenced	**0 (0.0%)**	**23 (100%)**	**68 (100%)**	**90 (100%)**	**26 (100%)**	**22 (100%)**	**229 (100%)**

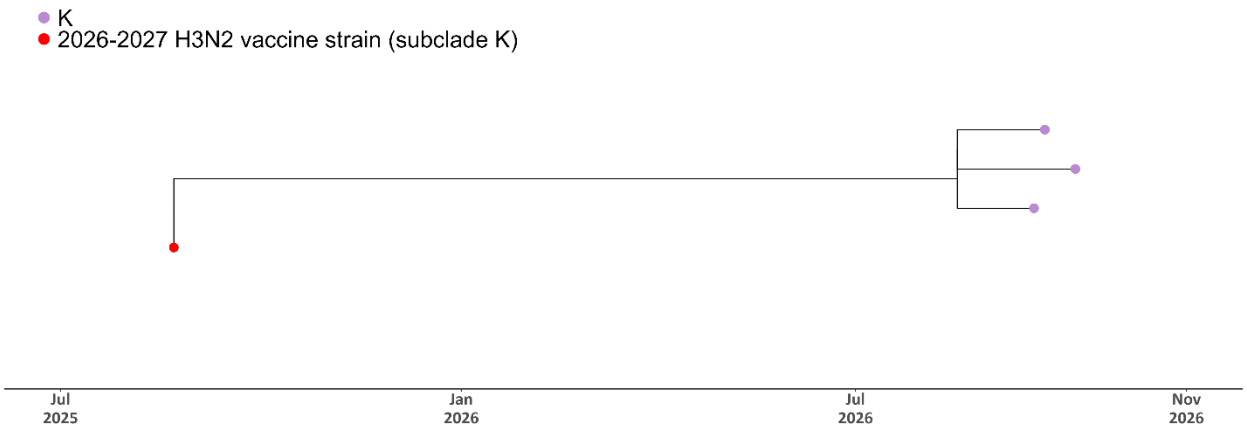
Note: Region was assigned using patient address when available. If missing, region was assigned using submitter address. For additional information on which public health units are included in each region, see Technical Notes. The influenza A genetic subclades included in the 2026–27 season's influenza vaccine are H1N1pdm09 subclade D.3.1 and influenza A H3N2 subclade K.[3]

Figure 2a: Time-Scaled Phylogenetic Tree of Positive Influenza A H1N1pdm09 Specimens Sequenced, Ontario, August 30 to October 10, 2026



Note: Sequences of the influenza HA gene (which encodes the virus' hemagglutinin surface protein) were analyzed and shown in the time-scaled phylogenetic tree. Each circle (tip) represents one specimen, and the colour indicates its influenza A H1N1pdm09 subclade. The branches (horizontal lines) are proportional to time and represent the inferred evolutionary relationships among viruses, with internal nodes showing the estimated time to a common ancestor, with the tips representing descendants. The x-axis represents time and approximates the specimen collection date. The 2026–27 vaccine reference strain, A/Missouri/11/2025 (H1N1)pdm09-like virus (EPI_ISL_20066835) belonging to subclade D.3.1, is shown in red. The specimens circulating in Ontario with subclade D.3.1.1 are genetically related to D.3.1.

Figure 2b: Time-Scaled Phylogenetic Tree of Positive Influenza A H3N2 Specimens Sequenced, Ontario, August 30 to October 10, 2026



Note: Sequences of the Influenza HA gene (which encodes the virus' hemagglutinin surface protein) were analyzed and shown in the phylogenetic tree. Each circle (tip) represents one specimen, and the colour indicates its influenza A H3N2 subclade. The branches (horizontal lines) are proportional to time and represent the inferred evolutionary relationships among viruses, with internal nodes showing the estimated time to a common ancestor, with the tips representing descendants. The 2026-27 vaccine reference strain**,** A/Darwin/1454/2025 (H3N2)-like virus (EPI_ISL_20260622) belonging to subclade K, is shown in red.

Table 7a: Number and Percentage of Positive Influenza A H1N1pdm09 Specimens Sequenced with Any Antigenic Site Amino Acid Substitutions, by Genetic Characterization, Ontario, August 30 to October 10, 2026

Genetic Characterization	HA Antigenic Site Ca	HA Antigenic Site Cb	HA Antigenic Site Sa	HA Antigenic Site Sb	Total
H1N1pdm09	**99.6% (225/226)**	**1.8% (4/226)**	**97.3% (220/226)**	**1.3% (3/226)**	**99.6% (225/226)**
D.3.1.1	99.6% (225/226)	1.8% (4/226)	97.3% (220/226)	1.3% (3/226)	99.6% (225/226)
Total Sequenced	**99.6% (225/226)**	**1.8% (4/226)**	**97.3% (220/226)**	**1.3% (3/226)**	**99.6% (225/226)**

Note: The effect of the identified antigenic site substitutions on vaccine-induced immunity is unknown. This data is exploratory in nature and should be interpreted with caution. This data should not be used to directly inform clinical decisions or infer impacts on vaccine-induced immunity. See Technical Notes for details. Antigenic site amino acid substitutions were identified relative to the strain included in the 2026–27 season's influenza vaccine (influenza A H1N1pdm09 subclade D.3.1).[3] Sequenced viruses may have substitutions at more than one position within the antigenic site. Antigenic site Ca includes any substitutions at positions 139, or 204 of the hemagglutinin (HA) protein. Antigenic site Cb includes any substitutions at positions 68, 71, or 72 of the HA protein. Antigenic site Sa includes any substitutions at positions 64, 121, 125, 154, 155, or 157 of the HA protein. Antigenic site Sb includes substitutions at position 190 of the HA protein. Of the influenza A H1N1pdm09 specimens, 213/226 (94.2%) had a G155E substitution.

Table 7b: Number and Percentage of Positive Influenza A H3N2 Specimens Sequenced with Any Antigenic Site Amino Acid Substitutions, by Genetic Characterization, Ontario, August 30 to October 10, 2026

Genetic Characterization	HA Antigenic Site A	HA Antigenic Site B	HA Antigenic Site C	HA Antigenic Site D	HA Antigenic Site E	Total
H3N2	**0.0% (0/3)**	**100% (3/3)**	**0.0% (0/3)**	**0.0% (0/3)**	**0.0% (0/3)**	**100% (3/3)**
K	0.0% (0/3)	100% (3/3)	0.0% (0/3)	0.0% (0/3)	0.0% (0/3)	100% (3/3)
Total Sequenced	**0.0% (0/3)**	**100% (3/3)**	**0.0% (0/3)**	**0.0% (0/3)**	**0.0% (0/3)**	**100% (3/3)**

Note: The effect of the identified antigenic site substitutions on vaccine-induced immunity is unknown. This data is exploratory in nature and should be interpreted with caution. This data should not be used to directly inform clinical decisions. See Technical Notes for details. Antigenic site amino acid substitutions were identified relative to the strain included in the 2026–27 season's influenza vaccine (influenza A H3N2 subclade K).[3] Sequenced viruses may have substitutions at more than one position within the antigenic site. Antigenic site B includes substitutions at position 195 of the HA protein.

Table 8: Number and Percentage of Positive Influenza A H1N1pdm09 Specimens Sequenced with Amino Acid Substitution H275Y Associated with Oseltamivir Resistance, by Genetic Characterization, Ontario, August 30 to October 10, 2026

Genetic Characterization	Amino Acid Substitution H275Y
H1N1pdm09	**0.4% (1/226)**
D.3.1.1	0.4% (1/226)*
Total Sequenced	**0.4% (1/226)**

Note: *This specimen is from a non-outbreak congregate living setting, the clinical and anti-viral therapeutic context are missing for this patient. H275Y substitution in the NA gene of the influenza genome has been associated with oseltamivir resistance in influenza A H1N1pdm09 viruses.[8] This data is exploratory in nature and should be interpreted with caution. Antiviral resistance is determined by investigation at specific sites previously identified to confer resistance and does not account for all potential mechanisms of resistance. This data should not be used to directly inform clinical decisions. See Technical Notes for details.

Table 9: Number and Percentage of Positive Influenza A Specimens Sequenced with Amino Acid Substitution I38T Associated with Baloxavir Resistance, by Genetic Characterization, Ontario, August 30 to October 10, 2026

Genetic Characterization	Amino Acid Substitution I38T
H1N1pdm09	**0.0% (0/180)**
D.3.1.1	0.0% (0/180)
H3N2	**0.0% (0/3)**
K	0.0% (0/3)
Total Sequenced	**0.0% (0/183)**

Note: I38T substitution in the PA gene of the influenza A genome has been associated with baloxavir resistance.[9] Not all influenza sequences had sufficient sequence quality at the PA gene to be included within the I38T analysis. As such, the total number of sequences included differs from other tables and figures. This data is exploratory in nature and should be interpreted with caution. Antiviral resistance is determined by investigation at specific sites previously identified to confer resistance and does not account for all potential mechanisms of resistance. This data should not be used to directly inform clinical decisions. See Technical Notes for details.

Technical Notes

Data Sources

Public Health Ontario (PHO)

- Data were extracted from the PHO Laboratory Information Management System on October 22, 2026.
- Bioinformatics processing of data by the Biocomputing Centre were completed on October 22, 2026.

Kingston Health Sciences Centre (KHSC)

- Data were submitted to PHO on October 14, 2026.

Shared Hospital Laboratory (SHL)

- Data were submitted to PHO on October 14, 2026.

The Hospital for Sick Children (HSC)

- Data were submitted to PHO on October 13, 2026.

Ontario Laboratories Information System (OLIS) data – Ontario Respiratory Virus Tool (ORVT)

- OLIS data were extracted from the ORVT on October 20, 2026.

Respiratory Testing and Whole Genome Sequencing Eligibility

- The 2026–27 respiratory season began on August 30, 2026.
- PHO and the OCGN respiratory virus testing (including for influenza) is based on patient setting and follows provincial ministry guidance. Testing is offered to individuals in long-term care and hospital settings, or individuals with increased risk of severe disease or complications.[10]
- The SPSN is a network of primary care practitioners that collect specimens from patients presenting with acute respiratory illness or influenza-like illness to assess vaccine effectiveness.[6] SPSN specimens are sent to PHO for testing and sequencing.
- Specimens were eligible for whole genome sequencing (WGS) by PHO and the OCGN if they were upper respiratory specimens, had Ct value ≤ 27 for influenza, were PCR positive only for influenza A (no co-infection), and had sufficient volume remaining. Additionally, only the first specimen from an outbreak was eligible for WGS.
- Specimens from the Sentinel Practitioner Surveillance Network (SPSN) were eligible for WGS if they were upper respiratory specimens and had Ct value ≤ 27 for influenza and had sufficient volume remaining.
- During the early season, all eligible specimens were sequenced.

Bioinformatic Analysis

- Genetic characterization was completed using whole genome sequencing and analyzed by a bioinformatics pipeline using Fastp (0.23.2), CFIA-NCFAD/nf-flu (3.10.1), bwa (0.7.17), bedtools (2.31.1), bcftools (1.22), ivar (1.4.3), emboss (6.6.0), and BLAST (2.17.0).[11-19] Clade and subclade were assigned with Nextclade (3.23.0) and Nextclade dataset (2026-08-07--07-54-41Z).[20-21]

- Time-scaled phylogenetic trees were created using IQ-TREE (3.0.1) and TreeTime (0.12.1).[22-23] The maximum likelihood phylogenetic tree was generated based on influenza HA-gene sequences using IQ-TREE with the GTR model (General time reversible model with unequal rates and unequal base freq), and 100 bootstrap replicates.[24] The tree was rooted on the vaccine strain and time-scaled using a clock rate of 3.0×10^{-3} substitutions/site/year for influenza A(H1N1pdm09), and 5.88×10^{-3} substitutions/site/year for influenza A(H3N2), with clock filtering disabled.
- An internally developed influenza mutational analysis pipeline was used to identify amino acid substitutions from HA gene sequences relative to the corresponding vaccine strain and map these substitutions to known HA antigenic sites.

Antigenic Characterization

- Antigenic characterization of influenza viruses involves an investigation of key proteins present on the outer surface of the influenza virus that can stimulate an immune response in the infected host. The main antigenic sites are contained within proteins which are involved in the entry and release of viral particles in host cells (the hemagglutinin (HA) and neuraminidase (NA) proteins). Antibodies that bind to specific regions of these proteins can initiate recognition of the virus by the infected host cells.[25]
- Within a respiratory season, antigenic characterization (typing/matching) of circulating influenza viruses can be assessed by in vitro laboratory experiments that measure the strength of antibody responses and by sequence-based analysis of the viral genome. The similarity in genetic sequence can be used to determine the degree of relatedness between currently circulating influenza strains and those included in the recommended annual influenza vaccine.
- The data presented provides a summary of the mutations identified in the main antigenic sites relative to the influenza viruses in circulation at the time of this report. This data is exploratory in nature and should be interpreted with caution. The potential outcomes of the identified mutations on vaccine-induced immunity or antiviral response are unknown. This data should not be used to directly inform clinical decisions.

Antiviral Resistance

- Antiviral resistance was based on screening of genomic data for molecular markers of resistance as opposed to susceptibility testing.
- H275Y in the neuraminidase gene is considered a clinically relevant amino acid substitution associated with oseltamivir resistance in influenza A H1N1pdm09 viruses.[8] The effect of other substitutions (including those in H3N2 viruses) within this gene on oseltamivir resistance are not well described.
- I38T in the polymerase acidic (PA) gene is considered a clinically relevant amino acid substitution associated with baloxavir resistance in influenza A and influenza B viruses.[9] The effect of other substitutions within this gene on baloxavir resistance are not well described.

Data Caveats

- Specimens sequenced for this report represented 21.1% of all influenza positive specimens in Ontario during this time period.
- Data may not be representative of Ontario overall, as other hospitals and private laboratories also provide respiratory pathogen testing services.

- The population tested by PHO and OCGN labs is more likely to include older adults (≥65 years) and individuals at higher risk of severe influenza outcomes. The population tested by SPSN reflects a community-based setting and may differ from the PHO- and OCGN lab- tested population in age distribution, risk factors, and likelihood of severe disease.
- Additional biases may be introduced due to eligibility criteria for the volume of specimen available, whole genome sequencing specimen selection criteria, and whether a specimen can be successfully sequenced. As a result, H1N1pdm09 and H3N2 subtype proportions sequenced may not align with the Ontario Respiratory Virus Tool.
- Counts based on specimens do not represent unique individuals, as some individuals may have more than one specimen tested.
- The specimen date was assigned using the sample collection date when available, or the date of entry into PHO's or OCGN's laboratory information system if the collection date was missing.
- Age was assigned based on date of birth provided and the specimen date (sample collection date when available, or the date of entry into PHO's or OCGN's laboratory information system if the collection date was missing).
- Patient setting is missing for approximately 10% of influenza specimens.
- Region was assigned based on patient address when available and submitter address when missing. As such, a small proportion of individuals with missing patient address on the requisition may be misclassified.

  The regional groupings include the following public health units:

  - Northern region includes Algoma Public Health, Northeastern Public Health (formerly Porcupine Health Unit and Timiskaming Health Unit), Northwestern Health Unit, North Bay Parry Sound District Health Unit, Public Health Sudbury & Districts, and Thunder Bay District Health Unit.
  - Eastern includes Eastern Ontario Health Unit, Ottawa Public Health, Renfrew County and District Health Unit, and Southeast Public Health (formerly Hastings and Prince Edward Counties Health Unit, Kingston, Frontenac and Lennox and Addington Health Unit, and Leeds, Grenville and Lanark District Health Unit).
  - Central East includes Durham Region Health Department, Lakelands Public Health (formerly Haliburton, Kawartha, Pine Ridge District Health Unit and Peterborough County-City Health Unit), Peel Public Health, Simcoe Muskoka District Health Unit, and York Region Public Health.
  - Toronto includes Toronto Public Health.
  - South West includes Chatham-Kent Public Health, Grey Bruce Health Unit, Huron Perth Public Health, Lambton Public Health, Middlesex-London Health Unit, Southwestern Public Health, and Windsor-Essex County Health Unit.
  - Central West includes City of Hamilton Public Health Services, Grand Erie Public Health (formerly Brant County Health Unit and Haldimand-Norfolk Health Unit), Halton Region Public Health, Niagara Region Public Health, Region of Waterloo Public Health and Emergency Services, and Wellington-Dufferin-Guelph Public Health.

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Appendix A: Jurisdictional Comparison of Influenza Specimens by Genetic Characterization

Table A1: Number and Percentage of Positive Influenza A Specimens Sequenced, by Genetic Characterization and Jurisdiction, May 24 to October 24, 2026

Genetic Characterization	Ontario (August 30 – October 10)	Canada (September 1 – October 24)	United States of America (May 24 – October 10)	United Kingdom (August 24 – October 11)
H1N1pdm09	**226 (98.7%)**	**68 (97.1%)**	**252 (76.4%)**	**111 (65.3%)**
D.3.1	0 (0.0%)	0 (0.0%)	1 (0.3%)	0 (0.0%)
D.3.1.1	226 (98.7%)	68 (97.1%)	251 (76.1%)	111 (65.3%)
H3N2	**3 (1.3%)**	**2 (2.9%)**	**78 (23.6%)**	**59 (34.7%)**
J.2.4.2	0 (0.0%)	0 (0.0%)	5 (1.5%)	0 (0.0%)
K	3 (1.3%)	2 (2.9%)	73 (22.1%)	59 (34.7%)
Total sequenced	**229 (100%)**	**70 (100%)**	**330 (100%)**	**170 (100%)**

Note: Prevalence may not be directly comparable across jurisdictions due to varying time periods and sampling strategies.
Data Sources: Public Health Ontario (Table 2), Public Health Agency of Canada[3], Centres for Disease Control and Prevention[26], UK Health Security Agency[27]

Appendix B: Antigenic Characterization by the Public Health Agency of Canada

Table B1: Results of Antigenic Characterization by Hemagglutination Inhibition Assay Comparing Circulating Influenza A Isolates to Their Respective Vaccine Reference Viruses in Canada, 2026–2027 Season

Genetic Characterization	Similar Reactivity	Percent Similar	Reduced Reactivity	Percent Reduced
Influenza A(H1N1)	114	100	0	0
Influenza A(H3N2)	2	100	0	0

Data Source: Public Health Agency of Canada[3], data was updated on October 30, 2026 with data up to and including October 24, 2026 (surveillance week 42).

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