Key facts

- Influenza activity started in week 47/2017 and returned to baseline levels in week 18/2018.
- Influenza viruses circulated at high levels between weeks 51/2017 and 13/2018. This is longer than in recent seasons and may have contributed to the severity of this season.
- The majority of influenza viruses detected were type B, representing a higher level of circulation of influenza B viruses compared to recent seasons. B/Yamagata lineage viruses, which were not included in the inactivated trivalent influenza vaccine, outnumbered B/Victoria lineage viruses, and more than half of the latter were antigenically distinct from the corresponding 2017–2018 trivalent vaccine component.
- Both influenza A subtypes, A(H3N2) and A(H1N1)pdm09, co-circulated in the region. Different patterns of dominant type and A subtypes were observed between countries.
- Overall, the majority of severe cases reported in 2017–2018 were due to influenza B infection and mostly occurred in persons 40 years of age and older. Over half (53%) of patients in intensive care units were infected by influenza A.
- Excess mortality from all causes was reported by the majority of reporting countries during the influenza season and was mainly observed in people aged 65 years and older.
- The vast majority of influenza viruses tested were susceptible to neuraminidase inhibitors.

Methods

For a detailed description of methods used to produce this report, please refer to the Methods chapter [1].

An overview of the national surveillance systems is available online [2].

Additional data on influenza are accessible from ECDC’s online Surveillance atlas of infectious diseases [3]. ECDC published an early risk assessment for influenza in December 2017 [4] and a rapid communication with the European Influenza Surveillance Network in March 2018 [5].

The surveillance of influenza in EU/EEA countries is carried out by the European Influenza Surveillance Network (EISN), coordinated by the European Centre for Disease Prevention and Control (ECDC).
EU/EEA influenza surveillance is based on weekly data reported to national public health authorities from week 40 to week 20 of the following year. It also draws on sentinel data reported by national influenza reference laboratories and general practitioners and, in some countries, other physicians. Seasonal influenza surveillance in the EU/EEA also takes into account non-sentinel data and severe disease data.

Surveillance data include:

- Qualitative indicators of influenza activity, namely intensity, geographic spread and trend. Intensity, ranging from low activity, i.e. no activity or activity at baseline level, to very high, is an indicator of the level of influenza activity. Geographic spread, ranging from no activity to widespread, refers to the number of affected areas in a given country. Trend – increasing, stable or decreasing – compares the level of influenza-like illness (ILI) and acute respiratory infection (ARI) sentinel consultations with the previous week.
- The aggregate number of ILI and/or ARI cases seen by sentinel physicians\(^1\) [2]. Each country also reports denominator data (population covered by sentinel surveillance) to enable calculation of weekly ILI and ARI consultation rates.
- The aggregate number of sentinel specimens obtained from a systematic sample of ILI/ARI patients that also tested positive for influenza, by type, A subtype, and B lineage [2]. Overall positivity rates of sentinel specimens are used to estimate the start, the duration, and the end of influenza activity; a 10% threshold is used to indicate the start of the seasonal epidemic.
- Antigenic and genetic characterisation and strain-based antiviral susceptibility data for a subset of influenza viruses detected in sentinel and non-sentinel specimens [2].
- Case-based data on patients admitted to intensive care units and/or hospitalised influenza patients; data were reported by a subset of countries\(^2\) and included demographic, clinical and virological data [2].

Since the 2014–2015 season, influenza surveillance in the 53 countries of the WHO European Region has been jointly coordinated by ECDC and the WHO Regional Office for Europe. Results are disseminated through a joint weekly bulletin (www.FlunewsEurope.org) [6]. Archived weekly data from October 2014 onwards are available from: http://www.flunewsurope.org/Archives [6].

This report presents data from EU/EEA countries and the EuroMOMO project [7], which monitors weekly all-cause excess mortality in Europe.

Seasonal data in this report, covering the period from week 40/2017 to 20/2018, were extracted from the database during week 21/2018.

**Sentinel surveillance**

During the 2017–2018 season, 41 315 specimens from sentinel primary care providers were tested for influenza, 22% more than in the previous season; 49% of the specimens were positive for influenza virus (previous season: 40%).

In week 47/2017, the weekly percentage of sentinel specimens positive for influenza crossed the 10% threshold, signalling an earlier than usual beginning of the seasonal epidemic (based on data from the 2013–2016 season, the threshold was exceeded after week 49), but one week later than the 2016–2017 season. Influenza viruses circulated at high levels between weeks 51/2017 and 13/2018 (based on proportions of 40% and above of sentinel specimens testing positive for influenza virus). The percentage of positive specimens peaked at around 60%, higher than in the previous season, and the peak lasted between weeks 52/2017 and 10/2018, longer than in the previous season. Influenza activity returned to baseline levels in week 18/2018, one week later than in the previous season (Figures 1 and 2).

Of 20 322 positive sentinel specimens, 36% were type A, and 64% were type B. A higher level of circulation of influenza B viruses was observed compared to recent seasons. Of 6 030 A viruses subtyped, 38% were A(H3N2) viruses, and 62% were A(H1N1)pdm09 viruses. The majority of A(H1N1)pdm09 detections were reported by France (19%), Germany (17%), Spain (15%) and Italy (11%). Of 6 297 influenza B viruses ascribed to a lineage,

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\(^1\) ILI and a denominator were reported by Austria, Belgium, Croatia, Cyprus, the Czech Republic, Denmark, Estonia, Finland, France, Greece, Hungary, Iceland, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, the Netherlands, Norway, Poland, Portugal, Romania, Slovakia, Slovenia, Spain and the United Kingdom.

\(^2\) Severe Acute Respiratory Infection (SARI) data were reported by the Czech Republic, Denmark, Finland, France, Ireland, the Netherlands, Romania, Slovakia, Spain, Sweden and the United Kingdom.
97% were B/Yamagata, and 3% were B/Victoria viruses. Different patterns of dominant type and A subtype were observed across countries.

**Figure 1.** Weekly proportion of sentinel specimens positive for influenza virus and number of detections by type and subtype, EU/EEA, 2017–2018

**Figure 2.** Influenza intensity by country and week, 2017–2018

Hospitalisations due to influenza

Ten countries reported a total of 9,317 laboratory-confirmed hospitalised influenza cases in intensive care units (ICUs); 17,410 confirmed cases were reported from other wards during the 2017–2018 influenza season. These numbers include data from all hospitals in some of the reporting countries, while other countries reported only data from selected hospitals. Compared with the previous three seasons, there was an increase in the number of reported laboratory-confirmed influenza cases in ICUs and other wards in the countries that provided data in all of the seasons.

Influenza type A viruses were detected in 53% of all laboratory-confirmed influenza cases admitted to ICUs, with type B accounting for 47%. The majority of severe cases occurred in persons aged over 39 years (Figure 3). France and the UK accounted for 31% and 38% of ICU cases, respectively.

For laboratory-confirmed influenza cases reported from wards other than ICUs, type B viruses were detected more frequently (61%) than type A viruses (39%). Among the influenza A detections in patients 65 years of age and older, A(H3N2) accounted for the highest proportion of cases.

Figure 3. Laboratory-confirmed influenza cases admitted to ICU, by age group (year) and (sub)type, 10 EU/EEA countries, season 2017–2018

Virus characterisations

Virus characterisation data were reported from both sentinel and non-sentinel sources. Of 996 influenza A(H3N2) viruses attributed to a clade, 554 (56%) fell in the vaccine virus component clade (3C.2a), 430 (43%) in subclade 3C.2a1 — with viruses defined by N171K, often with N121K, amino acid substitutions in the haemagglutinin — and seven (<1%) fell in clade 3C.3a. Five A(H3N2) viruses were not attributed to any of the predefined clades.

All 560 A(H1N1)pdm09 viruses fell in the A/Michigan/45/2015 vaccine component clade (6B.1).

A reassortant influenza A(H1N2) virus was detected in the Netherlands during the 2017–18 season. The variant virus originated from the reassortment of seasonal influenza A(H3N2) and A(H1N1)pdm09 viruses and was considered to be antigenically similar to the circulating strains [8].

All 1,460 B/Yamagata lineage viruses belonged to clade 3, represented by B/Phuket/3073/2013. Of 123 B/Victoria lineage clade 1A viruses, 66 (54%) belonged to a subgroup represented by B/Norway/2409/2017, which carries HA1 double amino acid deletion, Δ162-163 (deletion of amino acids K162 and N163 in the HA1 subunit), characteristic of a new antigenically distinct subgroup of viruses that has been detected in several countries during the season [9,10].
For more information on virus characterisations for EU/EEA countries, see the February report by the WHO Collaborating Centre for Reference and Research on Influenza in London [11].

All-cause excess mortality

Pooled data from 20 EU/EEA countries reporting to the EuroMOMO project showed an excess mortality from all causes between the beginning of January 2018 and the end of February 2018 [7]. Excess mortality mainly affected people aged 65 years or older.

Antiviral susceptibility and vaccine effectiveness

There was little detection of antiviral resistance to neuraminidase inhibitors (<1% of viruses tested). Interim results on vaccine effectiveness from five European studies indicated that, in all age groups, influenza vaccine effectiveness was 25 to 52% against any type of influenza, 55 to 68% against influenza A(H1N1)pdm09, no effectiveness (~47 to -7) against influenza A(H3N2), and 36 to 54% against influenza B [12], which was consistent with estimates from the United Kingdom [13], Canada [14] and the United States of America [15].

Discussion

The influenza season 2017–2018 was a severe season compared with previous seasons. It lasted longer than previous seasons, which might have contributed to the large number of severe cases. When the overall 10% positivity rate was exceeded, most EU/EEA countries reported low intensity of influenza activity, suggesting a lower sensitivity of intensity as indicator of influenza activity.

For sentinel specimens, the proportion of type B viruses was higher than the proportion of type A viruses, and A(H1N1)pdm09 viruses outnumbered A(H3N2) viruses.

For type B viruses, B/Yamagata lineage viruses greatly outnumbered B/Victoria lineage viruses. B/Yamagata lineage was not included in the 2017–2018 trivalent seasonal influenza vaccine. Nevertheless, the vaccine effectiveness estimates indicated 36–54% vaccine effectiveness against influenza B, a result that likely indicates cross protection from the vaccine against B/Yamagata lineage viruses [12]. Limited circulation of B/Victoria lineage viruses was reported; 54% of these viruses belonged to the newly emerged deletion variant subclade, which is antigenically distinct from the 2017–2018 vaccine virus.

Different patterns of dominant type and A subtype were observed across countries, which may be due to the differences between surveillance systems or natural variation in influenza virus characteristics and epidemiology across Europe. Viruses in the 3C.2a groups are considered to be antigenically similar, but they are evolving rapidly, with the emergence of several virus subclusters defined by additional amino acid substitutions in the haemagglutinin.

ICU cases were mainly persons aged 40 years and older, while excess mortality from all causes was mainly observed in persons aged 65 years and older, which was similar to the previous season.

On 21 February 2018, WHO published recommendations for the influenza vaccine composition for the 2018–2019 season in the northern hemisphere [11]. Compared with the northern hemisphere 2017–2018 trivalent and quadrivalent vaccines, two changes in the vaccine virus components were recommended. The A(H3N2) component was replaced with an A/Singapore/INFIMH-16-0019/2016 (H3N2)-like virus. For trivalent vaccines, the B component was replaced with a B/Colorado/06/2017-like virus, representing the emergent strain of B/Victoria-lineage viruses with Δ162-163 deletion in HA1. The A(H1N1)pdm09 component in both trivalent and quadrivalent vaccines and the B/Yamagata component in quadrivalent vaccines remained the same [11].

Public health implications

Early estimates showed 25–52% vaccine effectiveness against any type of influenza and 36–54% against influenza B, which was mismatched with the B virus trivalent vaccine component. Influenza vaccination should continue to be recommended along the lines of the recommendations of the European Council (2009), as it remains the best preventive measure against influenza [16]. In addition, appropriate use of neuraminidase inhibitors should be continued, in accordance with national and international guidelines.

Regarding the influenza vaccine for 2018–2019, WHO has recommended that the A(H3N2) and the B/Victoria vaccine components should be changed. In addition, as this was the fourth consecutive season in which the circulating B virus was dissimilar from the B strain included in the most widely used trivalent vaccine, close monitoring of circulating B viruses is required. The quadrivalent vaccine, which includes viruses from both B lineages, provides better protection when both lineages are circulating or the circulating strain does not match the trivalent vaccine B virus component.
Surveillance of severe influenza cases should be further improved, with participation of more reporting countries. Antigenic and genetic characterisation of circulating influenza viruses should be continued.

References


