2017

REPORT

Early risk assessment:
What to expect of the 2017/18 influenza season in Norway

Norwegian Institute of Public Health

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Author(s):
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Per Kristian Svendsen and Grete Søimer

Graphic design cover:
Fete Typer

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Scope

This report presents the assessment by the Norwegian Institute of Public Health (NIPH) on the influenza situation early in the 2017/2018 season, and possible characteristics of the upcoming influenza outbreak in Norway. The report is based on data from a late-summer serosurvey, early-season surveillance data, vaccine sales and experience from previous influenza seasons. The report is meant to support capacity planning in the health services, provide background information to infection control and other health-care and public health personnel, as well as to provide in-depth information on influenza outbreaks in general. At this point, as the 2017-2018 outbreak is beginning to unfold, it is of particular interest to assess the different circulating influenza viruses, their eventual spread, and how this will influence the extent of illness, severe illness and mortality in various risk and age groups. The report is this year written in English, since we presume that the pre-season immunity and early surveillance data analysis may be of interest beyond Norway.

Summary prospects for the 2017/18 season

- Influenza activity is currently very low. Virus detections have started to increase early, but is not escalating as fast as last year. Noticeable outbreak activity by Christmas/New Year is likely but the main outbreak may peak later.
- Up to now influenza A(H3N2) detections have been in majority but B/Yamagata lineage viruses at present appear to be increasing more. Influenza A(H1N1)pdm09 and B/Victoria-lineage viruses appear to play a minor role so far.
- Increased population immunity measured in antibodies against influenza A(H1N1)pdm09 and A(H3N2) in blood samples collected in August 2017, particularly in age groups important for virus spread, might limit circulation of these viruses compared to the last two winters. A lower proportion has antibodies against the influenza B viruses.
- The eventual magnitude of the outbreak cannot be forecasted.
- Influenza A(H3N2) and influenza B/Yamagata lineage viruses, of variants similar to viruses circulating last winter, are the most likely candidates for predomination. Both the recent increase in influenza B and the high prevalence of antibodies against A(H3N2) viruses speak in favour of the B/Yamagata virus. However, it is still too early to tell which of the two will be in majority, and this may also come to vary by region.
- Both the A(H3N2) and B/Yamagata lineage viruses tended to infect the elderly last winter but in current-season early data the elderly are not equally prominent among the infected. However, early data on hospitalised influenza cases indicate that these viruses will most likely cause most disease and severe outcomes among the elderly.
- The vaccine effect against the circulating H3 viruses will probably be low to moderate. The vaccine effect against B/Yamagata will also probably be low to moderate, since this virus strain is not included in the vaccine. However, some cross protection between B/Victoria, which is included in the vaccine, and the circulating B/Yamagata is expected.
- Vaccination is the best measure to prevent severe influenza in high-risk groups. Since the effect of influenza vaccines vary, use of antivirals should also be considered, both for vaccinated and unvaccinated people and in particular for risk groups.

Influenza outbreaks are in their very nature unpredictable. The NIPH monitors the situation closely and may add updates to the assessment as needed. Influenza surveillance data are published weekly on www.fhi.no/influensa.

Summary of the previous 2016/2017 season

In 2016/2017, the influenza epidemic started unusually early. The main outbreak peaked around Christmas/New Year (Fig. 1) and was dominated by influenza A(H3N2), genetic group 3C.2a. Influenza B/Yamagata-lineage viruses culminated very late with a minor peak in mid-May, but the total number of detections was low. For the Norwegian population as a whole, last season's influenza epidemic was of medium intensity (Fig. 1). However, for people 65 years or older, the epidemic was of extraordinary intensity. A high proportion of elderly visited general practitioners or emergency clinics and several outbreaks in nursing homes were reported. The incidence of elderly that was hospitalised due to influenza was higher compared to the previous two seasons. The influenza epidemic was estimated to have caused about 1 700 excess deaths attributable to influenza, with most deaths occurring in the elderly.

Last season the approximate number of vaccine doses used was 533 000 doses, of which the majority was used for people over 65 years. The vaccine effect was deemed as moderate against the circulating strains.

The 2017/18 season this far

Influenza-like illness in primary health care

Since week 40/2017, the occurrence of influenza-like illness (ILI) in Norway has been very low (Fig. 1). The proportion of patients diagnosed with ILI at general practitioners and emergency clinics has slowly increased from 0.3% in week 43/2017 to 0.5% in week 48/2017. To determine the start of this season's influenza outbreak, threshold values has been calculated, using the Moving Epidemic Method (MEM) (1). This year's outbreak is defined to start when the ILI % reaches 0.80%. This means that according to this measure, in week 48/2017, this season's influenza outbreak had not yet started. Almost all counties in Norway had very low influenza activity in week 48, but the ILI % varied between the counties. Two counties, Oslo and Finnmark had both crossed the outbreak threshold, with ILI proportions of 0.9%, which indicates low influenza activity.

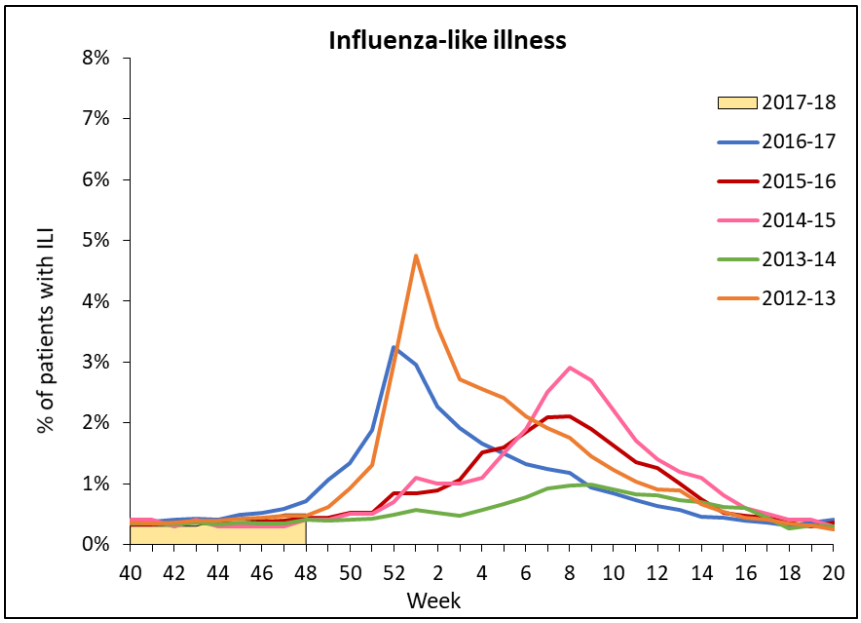

Figure 1. The proportion of patients diagnosed with influenza-like illness (ILI) at general practitioners and emergency clinics in Norway 2012-2017.

Despite of very low ILI activity so far, from week 40/2017 to week 48/2017, two confirmed outbreaks of influenza A-virus were reported from nursing homes, which is early and indicates local influenza virus circulation. Overall, the surveillance results from primary health care suggests that the occurrence of ILI will cross the national outbreak threshold within the next three to four weeks. However, there is still uncertainties about how fast the influenza activity will increase and when the peak of the outbreak is reached. The peak normally culminates either around New Year or after mid-February (Fig. 1).

Severe influenza

From week 40/2017 through week 48/2017 influenza virus has been detected in 84 (1.4%) of the 5 924 hospitalised patients that have been tested. Of these, 47 persons have been 60 years or older. Influenza A virus has been detected in 66 hospitalised patients, whereas influenza B virus has been detected in 18 hospitalised patients. Even though the

number of hospitalised patients is still lower this season when compared with the number of hospitalisations at the same time last season, these results suggest that this year's influenza outbreak may also cause most severe disease in people aged 60 years or older. Influenza A(H3N2) and influenza B-Yamagata viruses are both known to cause most severe disease in this age group. This means that hospitalisations and influenza related excess deaths are expected in this age group, but the extent depends on how widespread A(H3N2) becomes and it may be limited by pre-existing immunity (see section about pre-season population immunity to influenza, August 2017) in this part of the population since a very similar H3 virus circulated widely in this age group during the outbreak last season.

The reporting from the Norwegian Intensive Care Registry of influenza patients in ICU started in week 46/2017. Since week 46, two confirmed (using ICD-10 diagnostic code J10) and nine suspected (J11) influenza cases in ICU were reported. No deaths of influenza patients in ICU has been reported so far.

Since week 40/2017 through week 47/2017, no excess all-cause mortality has been observed.

Laboratory confirmed influenza

The extent of testing for respiratory pathogens has been increasing year by year (Fig. 2), and influenza testing is typically performed as part of a broader testing scheme. During the 2016/17 season, outcomes of more than 160 000 influenza tests were reported to NIPH.

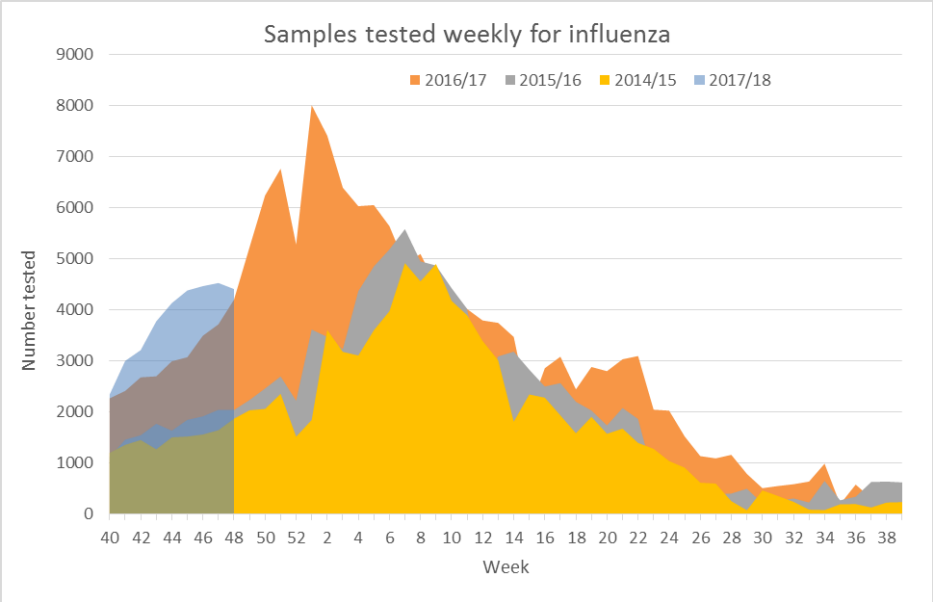

Figure 2. Weekly number of specimens tested for influenza virus, this season and the previous three seasons.

Based on data from a number of other laboratories in Norway, the most frequently detected pathogen this autumn has been rhinovirus with *Mycoplasma pneumoniae* also being widespread and other pathogens, including influenza virus, being considerably less common.

Nevertheless, due to the extent of testing, there were sporadic detections of influenza viruses during every week through the summer, as has also been the case in all recent years. An increase of influenza A virus detections began in mid-October (week 43) and has continued through week 48, but with a slight drop both in number of positives and proportion positive in week 47. Influenza B virus detections did not increase much until it more than doubled in week 46, and by week 48 it has almost caught up with the weekly number of influenza A detections.

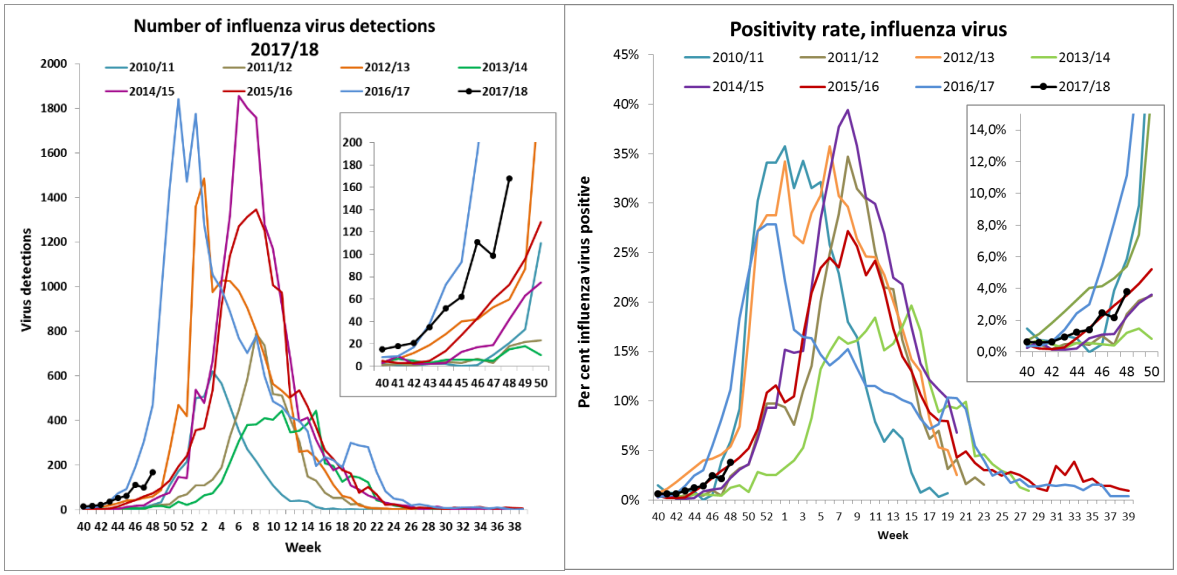

Figure 3. Laboratory detections, Norway, start of 2017-2018 season, compared with preceding seasons. The left-hand panel shows the weekly number of influenza virus detections, the right-hand panel shows the weekly proportion of influenza virus positives among those tested. Whereas the test sensitivity probably has not changed during the years covered by the graph, the number of specimens tested has risen gradually.

When comparing positivity rates with previous seasons (Fig. 3, right-hand panel), the course so far is aligned with seasons that peaked well after New Year. Therefore, although the number of influenza cases are already increasing and higher than most previous seasons at this time of year, it cannot be concluded that activity is heading toward an early main peak. A gradual rise with a slow-down over Christmas/New Year and then a resumed increase toward a later main peak, similar to e.g. the 2015/2016 season, seems just as likely.

The laboratory detections data available at this stage give no information regarding the eventual magnitude of the outbreak.

Whereas there has been twice as many influenza A detections as influenza B since the start of the season, influenza B detections have recently been increasing more and the type A dominance has gradually been lost during the last three weeks (Fig. 4, left panel). If this trend continues, the influenza B viruses may take predominance in the build-up to the main outbreak.

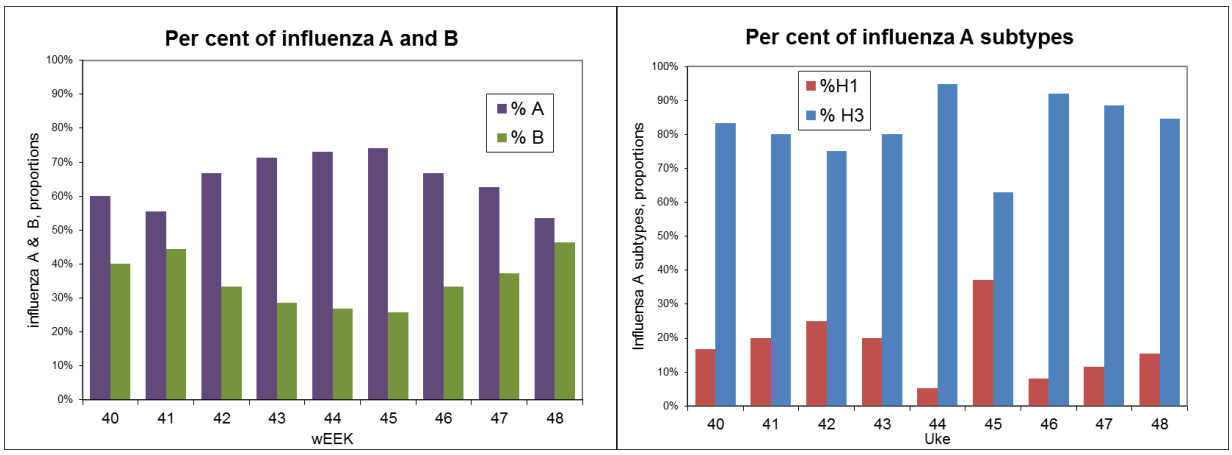

Figure 4. Weekly proportions of influenza type (left panel) and influenza A subtype (right panel), start of 2017-2018 season, Norway.

Among the subtyped influenza A viruses (n=181), the strong predominance of A(H3N2) over A(H1N1) appears to be maintained, and most of the A(H1N1) detections are concentrated in southwestern Norway. Among the influenza B viruses forwarded to the National Influenza Centre (NIC) in NIPH and further identified, 48 (96%) have belonged to the B/Yamagata/16/1988 lineage and only 2 to the B/Victoria/2/1987 lineage.

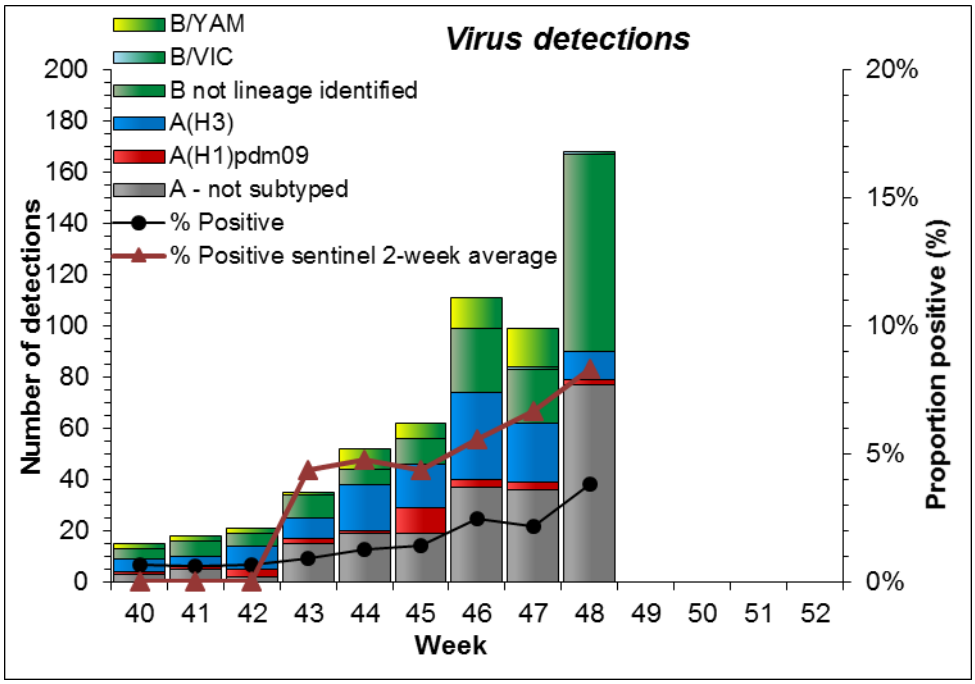

Figure 5. Laboratory detections, Norway, start of 2017-2018 season. Weekly number of the different influenza viruses is displayed as stacked bars, and influenza virus positivity rates of sentinel specimens (two-week averages) and all lab testing, respectively, are shown as line graphs.

Based on the current data, influenza A(H3N2) and B/Yamagata-lineage viruses are the two that are most likely to predominate, with A(H1N1)pdm09 and B/Victoria-lineage viruses likely to play a lesser role in Norway this winter.

To elucidate age profiles for the different viruses, the number of detections per age group was divided by the population size of the age group, thus producing seasonal incidences of detections (Fig. 6).

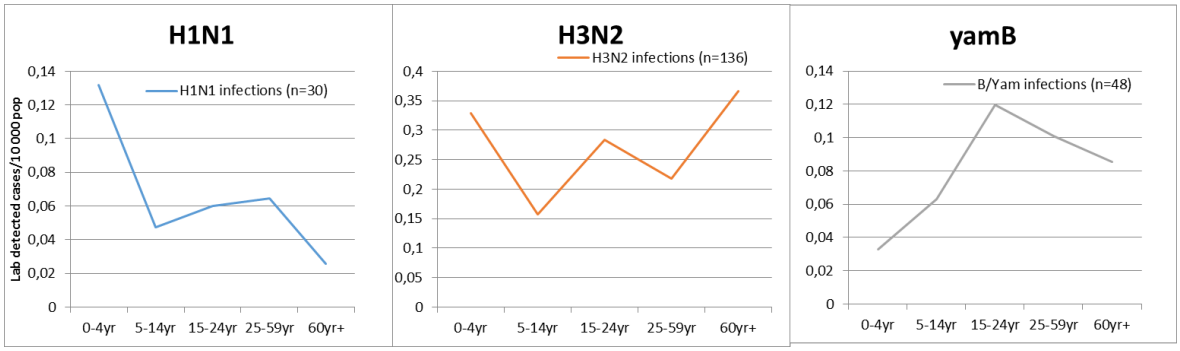

Figure 6. Age group-specific cumulative seasonal incidence of virus detections, for influenza A(H1N1)pdm09, A(H3N2), and B/Yamagata-lineage. Only two B/Victoria-lineage viruses have been detected at this stage, both in the 25-59 years age group. The cumulative number of detections recorded in each age group is divided by the age group population size, to achieve better comparability between age groups. The incidences cannot be compared between the different viruses, since the extent of testing for subtype and lineage varies widely.

Although numbers are small, influenza A(H1N1) cases are skewed more toward infants than observed for the other influenza viruses.

The age profile for influenza A(H3N2) viruses assumes a W-shape like last season, but last winter the incidence for the elderly was much higher than all the other age groups and this is not seen now, at least not yet. The higher incidence in elderly last winter was evident already by week 45/2016, when the cumulative number of H3 cases was lower than the present data collected through week 48/2017.

Similarly, last winter the elderly were more than twice as likely as people in other age groups to be diagnosed with influenza B/Yamagata-lineage viruses, and this is not apparent so far this season.

The early-season age profiles thus may give some hope that the upcoming season might not selectively affect the elderly in the same way as the preceding 2016/2017 season. The age patterns may change as the outbreak progresses.

Genetic characterizations of the viruses in circulation

As of week 46, 43 % (57 viruses) of all influenza viruses received at NIC Norway have been further characterised genetically. A selection of sequences is available in GISAID (www.gisaid.org).

As the previous season, the influenza A(H3N2) viruses are predominating the start of the 2017/18 season (Fig. 7: Cluster analysis of H3). Both H3N2 group 3C.2a and 3C.2a1 viruses are circulating; however, the 3C.2a main group of viruses has so far outnumbered the 3C.2a1 viruses and seem to be more widespread. The same 3C.2a viruses were also dominating the previous season in Norway, in contrast to 3C.2a1 viruses in most of Europe. The H3N2 vaccine component also belongs to the 3C.2a group of virus, but several

3C.2a subclades have emerged, making the genetic picture for H3 viruses very complex (Fig. 7: Cluster analysis of H3).

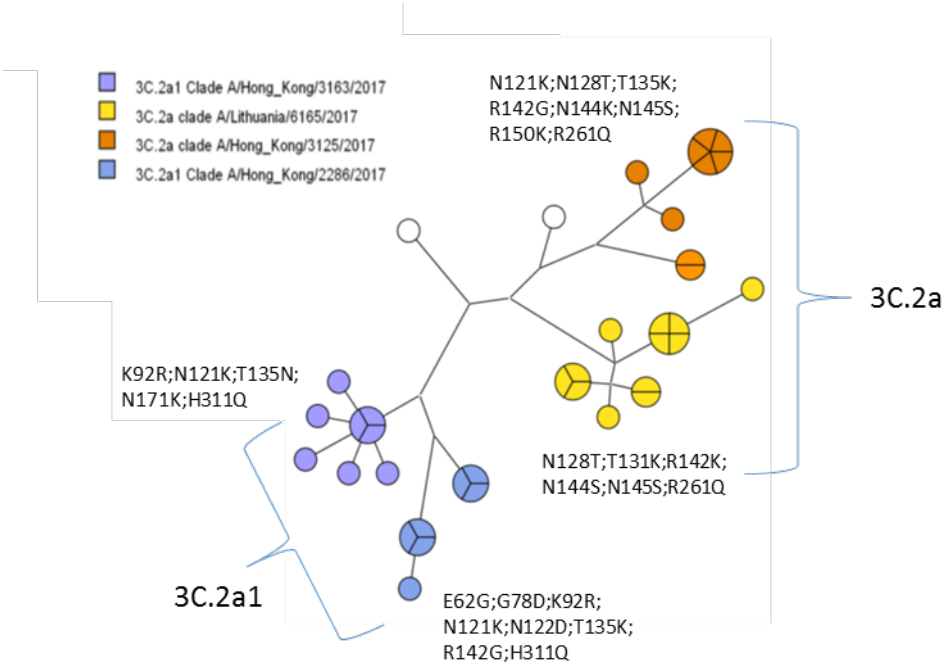

Figure 7. Maximum parsimony cluster analysis of the HA gene of Norwegian influenza H3 virus with northern 3C.2a (2017/18) and southern 3C.2a1 (2018) hemisphere vaccine H3 viruses as references (white circles). Viruses are colour-coded by defined genetic subgroups. HA amino acid substitutions defining the different genetic groups are given (with reference to A/Texas/50/2012).

The NA gene of the 3C.2a viruses group genetically together with the NA genes of the other group of H3 viruses, the 3C.2a1 (not shown). The effect of this reassortment is currently unknown.

The influenza B/Yamagata viruses are similar to the viruses from the previous season in Norway and group genetically with clade 3 B/Yamagata-lineage viruses (Fig. 8). Two single recent viruses from different parts of Norway stand out from the other B/Yamagata this and previous season, possessing two amino acid substitutions in HA compared to the vaccine strain, namely positions Q122K and T181A. The increase in B/Yamagata cases the last weeks cannot be explained by the genetic analysis of the circulating viruses.

Only two influenza B/Victoria viruses have so far been detected, these have not yet been genetically characterised.

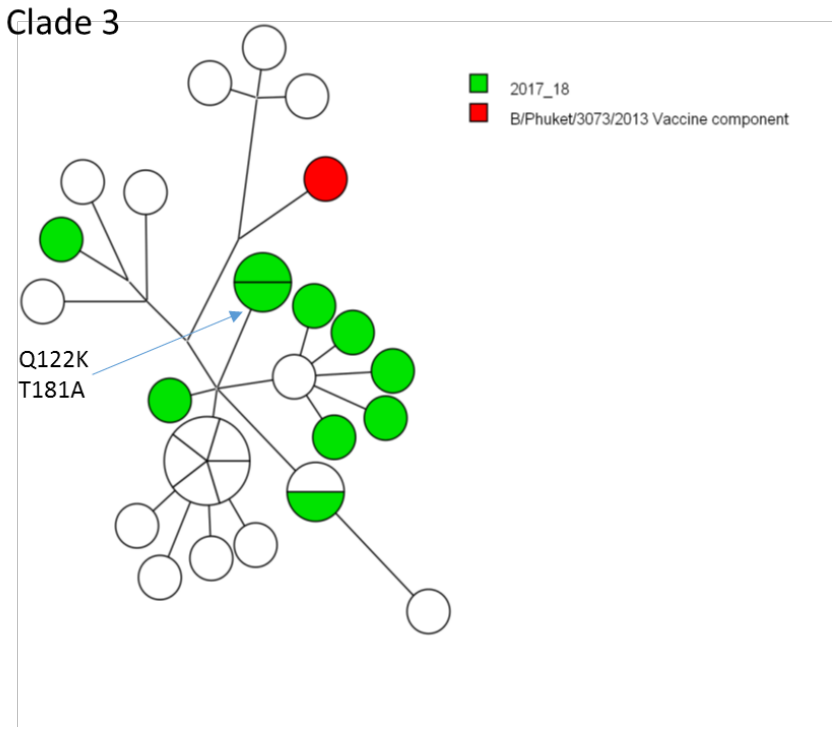

Figure 8. Maximum parsimony cluster analysis of the HA gene of Norwegian influenza B/Yamagata virus with northern hemisphere vaccine B/Yamagata virus (red circle) as reference. Viruses are colour-coded by viruses from the 2017/18 season (green) and from previous 2016/17 season (white). Key HA amino acid substitutions are given.

The H1N1 viruses are all clade 6B.1 and the largest genetic group possesses the following substitutions: T72S, S74R, D97N, P137S, S162N, S164T, K163Q, I216T and I295V.

Antiviral susceptibility

No resistance towards neuraminidase inhibitors like oseltamivir and zanamivir has so far been detected out of 8 H3 viruses, 9 H1 viruses and 1 influenza B virus analysed.

Vaccine

Vaccine match

As the predominant H3 influenza strain in Norway belongs to the same group of H3 viruses as the H3 vaccine component, the vaccine will induce some protection towards the H3 viruses. We expect varying levels of protection as several different subgroups of H3 viruses are in circulation (2). The WHO Collaborating Centre laboratory in London estimates that about 70-80% of the 3C.2a H3 viruses circulating during the last season and the summer months will be well covered by the vaccine, and 60% of the 3C.2a1 H3 viruses (3). The H1 viruses match the H1 vaccine component. However, the B/Victoria virus in the vaccine is not a good match towards the B/Yamagata viruses currently circulating in Norway, still some cross-protection is expected.

The influenza vaccine for the Northern hemisphere 2017/18 contains three different virus components:

- A/Michigan/45/2015 (H1N1)pdm09 6B.1-like virus
- A/Hong Kong/4801/2014 (H3N2 3C.2a) -like virus
- B/Brisbane/60/2008-(B/Victoria)-like virus

Vaccine distribution and coverage

Available vaccines for the vaccination of targets groups (risk groups and health care workers) (4) are split vaccine Vaxigrip® 2017/18 from Sanofi Pasteur, and sub-unit vaccine Influvac® 2017/18 from BGP Products. Both vaccines are available for the private market, in addition to nasal vaccine Fluenz Tetra from Astra Zeneca.

As of seventh of December NIPH has distributed over 525 000 vaccine doses for the target groups. In addition NIPH and other wholesalers have distributed nearly 113 000 doses for the private market. Total volume: almost 638 000 vaccines doses. This is an increase of 90 000 doses compared to last year.

As of sixth of December 337 091 persons have been registered in the national vaccine registry SYSVAK as vaccinated with this season's vaccine. The majority of them – 229 952 - are over 65 years of age. There is a certain lag time in the registration of some vaccinations, so the real number of vaccinated is probably much higher.

The influenza vaccine coverage in Norway remains low. Nevertheless, it has increased the last couple of years, especially in the elderly and among health care workers. Last season the vaccine coverage in the general public was 10 %. The coverage was 38 % among the elderly and 17 % in health care workers. Considering the increase in vaccine sales this season, it seems that the vaccine coverage in general will be at least as high as last year's. This will hopefully contribute to fewer hospital admissions and deaths due to influenza among the risk groups the coming season, since it will add to the prior immunity already present in the population.

Infection control measures

Vaccination is the most effective preventive measure against influenza. Since the effect of influenza vaccines vary, use of antivirals should be considered for patients experiencing influenza illness, particularly for those belonging to risk groups. Antiviral treatment should be initiated as early as possible after onset of symptoms (preferably within 48 hours), both for vaccinated and unvaccinated patients. For patients with severe influenza requiring hospitalisation, treatment with antivirals should also be considered later in the course of the disease.

To prevent transmission of influenza, it is also important to practice good hand and respiratory hygiene.

Pre-season population immunity to influenza, August 2017

The National Seroepidemiological Influenza Programme

The National Seroepidemiological Influenza Programme annually collects in August about 2000 serum samples during the weeks 31-35 from clinical/microbiological laboratories covering the 19 counties of Norway. These anonymised convenience sera are aimed to be representative of the Norwegian population geographically and by age composition. The sera are tested by the hemagglutination-inhibition (HI) test to determine the antibody immunity to relevant circulating influenza viruses. HI titres ≥ 40 against the influenza A strains and ≥80 against ether-treated influenza B strains are considered as protective levels and recorded as seropositive in the analysis.

The 2017 serum panel was tested by HI against the Northern hemisphere 2017/18 seasonal influenza vaccine strains (trivalent/quadrivalent). In addition, two recent viruses were analysed in HI: A/California/07/2009 (H1N1pdm09/X179A), the previous H1N1 vaccine virus, and A/Norway/3806/2016, a recent H3N2 3C.2a.1 variant-like virus belonging to the same group of viruses as A/Singapore/MIFIMH-16-0019/2016, the new H3 vaccine component of the 2018 influenza vaccine for the Southern hemisphere (Table 1).

The results are shown in Fig. 9 and Table 1. HI testing is still ongoing and the present preliminary analysis has been carried out on a subset representing approximately ¾ of the complete annual panel. Protective immunity from subsequent seasonal vaccination will come in addition.

Summary of outcomes

The seroprevalence to both influenza A viruses is relatively high, indicating good protective immunity against the two influenza A vaccine-like viruses. However, new H3N2 HA variants with major antigenic drift may give less protection in the population. Historically, H3N2 viruses have higher frequency of mutations in HA and may thus have a higher potential for new variants as compared to the H1N1pdm09 viruses, which have undergone little antigenic change since the pandemic in 2009. The seroprevalence to influenza B viruses is low to moderate, as it has been the last few years. This may indicate a population vulnerability that has not been exploited during the last seasons, for reasons not well understood. In addition, although it remains rarely detected and that a preliminary analysis suggest cross-immunity from related viruses, the new influenza B/Victoria HA deletion variant may be reason for some concern.

High seroprevalences to influenza A viruses in August 2017

The seroprevalences against both influenza A(H1N1)- and A(H3N2)-viruses are unusually high in August 2017 and had increased significantly from August 2016 (Fig 9). The highest increase in protective antibody immunity, by 22 percentage points, is seen against A(H3N2) with all-ages seroprevalence almost doubled. This reflects the dominant circulation of H3N2 vaccine-like viruses the preceding season. Surprisingly, an increase in seroprevalence to A(H1N1) by 9 percentage points (18% relative increase) is seen even though very few H1N1 viruses were detected the preceding season.

The highest antibody seroprevalences to both H1N1 and H3N2 viruses are seen in 5-14 and 15-24 year olds (Table 1). This holds for both the H1N1 viruses tested, A/California/07/2009 and A/Michigan/45/2015 (clade 6B.1), the previous season and the current season H1 vaccine virus, with about 75 to 80 % seropositive in these two age groups, i.e. increases by 8 and 10 percentage points from previous year. Additionally, the seroprevalences to H1N1 in the age groups 25-59 and 60+ year olds are higher than seen the year before with about 8 to 10 percentage points increase, while for the 0-4 year olds there is a decrease of 8 percentage points.

The highest seroprevalences against the H3N2 vaccine virus A/Hong Kong/4801/2014 (an H3N2 3C.2a variant) are also in those between 5-14 and 15-24 years old with 80% and 67%. This is an increase from the previous year by 20 and 33 percentage points, respectively. The seroprevalences for the other age groups against H3N2 have also increased substantially by between 15 to 19 percentage points from August 2016. Interestingly, the seroprevalences in various age groups against a recent new H3N2 3C.2a.1-group virus, A/Norway/3806/2016, are similar to the prevalences to A/Hong Kong/4801/2014-like virus. An H3 3C.2a.1-variant virus (A/Singapore/INFIMH-16-0019/2016) was recently selected as the H3N2 vaccine component for the Southern hemisphere 2018 season.

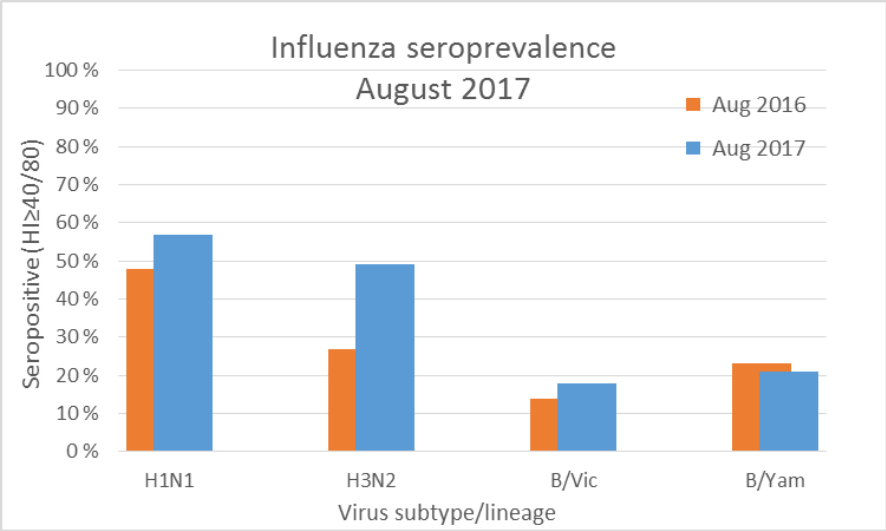

Figure 9. Influenza seroprevalence in August 2017. All-ages percentage of sera with protective HI-titre against the H1N1, H3N2, B-Victoria and B-Yamagata virus components of the 2017/2018 Northern hemisphere trivalent/quadrivalent influenza vaccine (Blue columns, preliminary data). For comparison, the corresponding seroprevalence in August 2016 is also shown (Orange columns).

Table 1. Age group seroprevalences to the 2017/2018 influenza vaccine components and two recent circulating variants (H1N1pdm09 and H3N2 3C.2a.1) of sera collected in August 2017.

Virus	Age group (yrs)				
	0-4	5-14	15-24	25-59	60-99
A(H1N1)pdm09 A/Cal/07/2009 (X179A)	24 %*	78 %	81 %	52 %	46 %
A(H1N1) A/Michigan/45/2015 (6B.1) (**V**)	24 %	74 %	81 %	49 %	41 %
A(H3N2) A/Hong Kong/5738/2014 (3C.2a)** (**V**)	33 %	80 %	67 %	35 %	42 %
A(H3N2) A/Norway/3806/2016 (3C.2a.1)	31 %	80 %	74 %	40 %	46 %
B(Vic) B/Brisbane/60/2008 (**V**)	8 %	30 %	22 %	12 %	22 %
B(Yam) B/Phuket/3073/2013 (**V**4)	4 %	24 %	34 %	21 %	17 %

*Percentage of sera with HI titre ≥40 for influenza A virus and ≥80 for influenza B virus. The results are preliminary data from abt. 2/3 of the 2017 serum panel. Number of samples (n) in each age group are: 113, 199, 222, 503, and 276.
(V): Trivalent vaccine viruses of the Northern hemisphere 2017/2018 vaccine; (V4): the fourth component in 4-valent vaccine.
** A/Hong Kong/5738/2014 is an A/Hong Kong/4801/2014-like virus

Low to moderate seroprevalences to influenza B viruses in August 2017

Less changes from August 2016 in seroprevalences are seen against the two influenza B lineages B/Victoria and B/Yamagata, both for 'All ages' and in the various age groups (Fig 8). For most age groups changes in seroprevalences are less than +/- 5 percentage points, except for those 60 years and older to the B/Victoria-lineage vaccine component B/Brisbane/60/08 with an increase of 11 percentage points.

Immunity against a new influenza B/Victoria-lineage hemagglutinin double deletion variant

During last season, a B/Brisbane/60/08 (B/Victoria-lineage) hemagglutinin (HA) double deletion variant was identified in the US and Norway as well as some other countries, causing serious concern for lack of immunity to this new B/Victoria-lineage variant virus in the human population as indicated by antigenic characterization using monospecific ferret antisera. However, preliminary HI analysis using pre-2016/17 human sera from our annual serum collection may indicate that there is some level of cross-reactive antibodies in the population to this new variant (data not shown). Furthermore, this variant has not been seen in increased numbers during the 2017 Southern Hemisphere season and thus, the risk for major spread of this virus variant in its current form may be lower than initially feared. However, influenza B viruses tend to circulate later in season and if additional mutations in the HA take place, the existing cross-immunity in the human population may give less protection.

Acknowledgements

The NIPH would like to express our gratitude to all medical doctors and other staff at the regular general practitioner offices, emergency clinics and microbiological laboratories that have contributed to the surveillance by sending samples and data. We are also very grateful for the contribution of data from the Norwegian Intensive Care Registry, and to the hospitals and nursing homes for notifying outbreaks in VESUV.

We would also like to thank all the health care workers that have organised the target group vaccination and contributed to the registration of influenza vaccinations in SYSVAK.

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Tel: + 47-21 07 70 00
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Norwegian Institute of Public Health