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Letter to the editor

Climate and influenza in Oslo: A case for vapour pressure

Globally, influenza epidemics constitute a major healthcare challenge. A better understanding of the different driving factors behind their seasonality is important for optimizing interventions, which may reduce the associated impact on health and healthcare resources [1].

In temperate regions, annual outbreaks of influenza are largely confined to the cold winter months [1]. This indicates an interplay between climate factors and these seasonal epidemics, although the *size* and *impact* of the outbreaks also may be affected by antigenic drift of the virus and the immune status of the population [2].

A previous study by Sundell et al found a strong association between low outdoor temperature, low absolute humidity and the incidence of influenza [3]. To further explore the relationship between climate factors and influenza, we conducted a simple retrospective study in the city of Oslo, Norway, using meteorological data and influenza incidence.

Data on the weekly incidence of influenza-like illness (ILI) in Oslo for the period 2010 to 2018 were obtained from the Norwegian Directorate of Health. The study population included all persons with a registered address in Oslo where a general practitioner (GP) had filed for reimbursement for the diagnosis of ILI, defined as R80 within the ICPC2 system [4]. In addition, data on samples positive for influenza analysed at the department of microbiology, Oslo University Hospital, in the years 2010 to 2018, were obtained from the Norwegian Institute for Public Health.

Averaged weekly data on outdoor temperature (T) and vapour pressure (VP) for Oslo were collected from the eKlima database, operated by the Norwegian Meteorological Institute. VP was used as a metric for absolute humidity.

Weekly incidence of ILI, virus positive samples and meteorological data during the study period are depicted in Fig. 1. The data were visually inspected to check for temporal relationships between meteorological factors and disease. We found a recurrent pattern throughout the study period, where both the incidence of R80 and virus positive samples increased sharply approximately 1 week after weekly average VP for the first time subsided 4 hPa (Fig. 1). This is consistent with the threshold for VP found in the study by Sundell et al. [3]. It is noteworthy that this was also the case for the relatively mild epidemic of 2013/2014 [5].

Our data suggests that low VP is an important factor for the initial spread of the virus but may be of less importance once the epidemic is established (Fig. 1). On a hypothetical note, this could reflect the predominance of aerosol transmission in the early phases of the epidemic, where low absolute humidity prolongs the survival of infective aerosols [2,6]

Our study is limited by the use of aggregate statistics and the R80 diagnostic code in primary care as a surrogate metric for influenza incidence. First, national holidays may introduce gaps in the data since GP offices then are generally closed. Secondly, although there are indications R80 can serve as an alternative source for influenza incidence [7], it encompasses both ILI and influenza without the need for laboratory confirmation. We have therefore added virus positive samples from Oslo University Hospital to the data. These samples are mainly referred from hospital inpatients, but could mirror the incidence of influenza in the general population, since the hospital serves a large part of the population of Oslo.

In conclusion, our study suggests that low vapour pressure, possibly

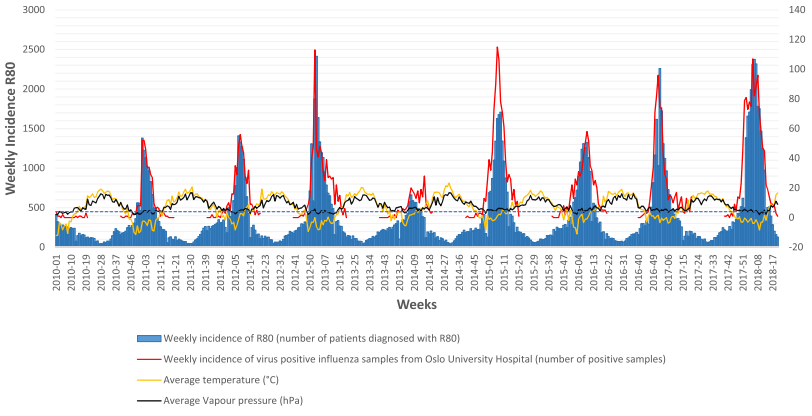

Fig. 1. Weekly incidence (number of cases/week) of R80 diagnoses and virus positive samples from Oslo University Hospital according to average weekly outdoor temperature and vapour pressure across the entire study period (January 2010 – May 2018). The dotted line indicates the 4 hPa threshold for vapour pressure.

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a weekly average beneath 4 hPa, is implicated in the triggering of seasonal influenza. Monitoring this parameter could therefore provide an early signal of an emerging epidemic.

Credit author statement

ES Kolberg designed the study. The data was collected by ES Kolberg. All of the authors participated in the analyzing and presentation of the data, discussed the results and contributed to the final manuscript.

Original publication

This work contains previously unpublished material and is not being considered for publication elsewhere.

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Compliance with ethical standards

Approval from the regional committees for medical and health research ethics in Norway was not required according to the Norwegian Health Registry Act. As the data contained in the study are aggregated, secondary data, no consent from individual patients is by law required.

Declaration of competing interest

The authors declare no conflict of interest.

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