lung, although no specific lesions compatible with this infectious agent were observed.

A pool containing all morbillivirus-positive PCR amplicons for animals 1 and 2 (GenBank accession nos. KT006289 and KT006290), a PCR amplicon for the brain sample from animal 2 (GenBank accession no. KT006291), and a PCR amplicon for the larynx from animal 3 were sequenced. A BLAST search (http://www.ncbi.nlm.nih.gov/blast/Blast.cgi) showed that amplified samples were nearly identical to reference PWMV sequences (GenBank accession nos. AF200817 [*3*] and FJ842381 [*8*]). The sequence obtained from animal 3 was too short and degenerated to be accurately classified as CeMV, although it showed high homology with PWMV and porpoise morbillivirus.

It has been proposed that pilot whales might be enzootically infected with CeMV (*10*). These whales might be responsible for maintaining and transmitting CeMV over long distances or to other odontocetes. No die-offs have been observed in these species. However, an outbreak of a lethal morbillivirus infection in long-finned pilot whales caused by a dolphin morbillivirus strain occurred in the Mediterranean Sea during the end of October 2006–April 2007 (*7*).

Results of this study support the previous hypothesis that pilot whales have a species-adapted morbillivirus but indicate that lethal infections are not as rare as previously believed (*3*). The tropism of the virus in these cases, the high number of multinucleated syncytial cells, and the severity of the lesions resemble the acute systemic symptoms observed in dolphins infected with morbillivirus (*2*). Thus, pilot whales in the northeastern Atlantic Ocean could be at risk for infection, especially in one of the main pilot whale–watching regions between La Gomera and Southern Tenerife Islands in the Canary Islands, which has >700,000 visitors each year.

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Serogroup-specific Seasonality of Verotoxigenic *Escherichia coli*, Ireland

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To the Editor: Globally, an increasing number of serogroups of verotoxigenic *Escherichia coli* (VTEC) have been reportedly associated with human illness. The best known is serogroup O157; the World Health Organization also recognizes VTEC O103, O111, O145, and O26 as having the potential to cause severe disease (*1*). The increasing number of non-O157 VTEC infections is cause for concern. In general, human infections with VTEC are reportedly more common in late summer; the European Centre for Disease Control and Prevention reported that the number of cases across the European Union peaks each year during July–September (*2*). Similarly, the United States reported that the number of VTEC O157 cases peaks in late summer (*3*).

Ireland is now one of the countries with the highest incidence of VTEC infection (*2*). In Ireland, statutory notification of VTEC infection became mandatory in 2004. In common with surveillance internationally, the focus was initially on VTEC O157; since then, testing and surveillance for non-O157 VTEC have improved substantially as a result of increased awareness and availability of diagnostic methods for non-O157 detection. Non-O157 VTEC were first reported in Ireland in 1999 (*4*), and surveillance data indicated that only 14% of VTEC notifications in 2004 compared with 75% in 2014 were caused by non-O157 VTEC.

In the notification dataset for Ireland, the 2 primary VTEC serogroups (O26 and O157) over many years have seemed to differ in their seasonality; VTEC O26 notifications generally peaked ≈2 months earlier than VTEC O157 notifications (Figure, panel A). This earlier incidence peak for VTEC O26 has become progressively more consistent as the number of reported VTEC O26 notifications has risen. A study by Rivero et al. also suggested that non-O157 human infections may not exhibit the same seasonal variation observed for VTEC O157 (*5*).

In this study, we compared the seasonality of the 2 strains by using national notification data for 2004–2014 (n = 2,569 notifications for O157 and O26). We estimated the timing of the seasonal peaks (phase of seasonality) for each of the serogroups, and the difference between the 2 phases, by using times series quasi-Poisson regression, fitting terms for temporal trend, and a sine wave with a period of 12 months for seasonality and for interaction by serogroup. We compared the phase shifts of the 2 serogroups by using the Wald test. To rule out the possibility that the observed distributions were influenced by the occurrence of a limited number of outbreaks, we reanalyzed the data for sporadic cases alone and, because risk factors for VTEC infection have been shown to vary by age (*6*), separately for patients <5 years of age and for older child and adult patients.

The number of predicted cases peaked in July for VTEC O26 and in September for VTEC O157; the 2-month difference in phase (seasonality) by serogroup was significant ($p<0.0001$) (Figure, panel B). The difference in seasonality remained significant ($p<0.0001$) for sporadic cases alone; the predicted 2-month difference in seasonality was the same. The serogroup-dependent seasonality also remained when the data were analyzed separately for patients <5 years of age (predicted difference in phase 2 months, $p<0.0001$) and ≥5 years of age (predicted difference in phase 1 month, $p<0.0001$).

A significant increasing annual trend was also observed, in particular for VTEC O26. However, this increase is probably, at least in part, artifactual because of increased availability and more widespread use of clinical diagnostic tests for non-O157 VTEC in later years.

One possible explanation for the difference in seasonality is that the primary animal reservoirs for the 2 serogroups could differ. Cattle and sheep have been identified

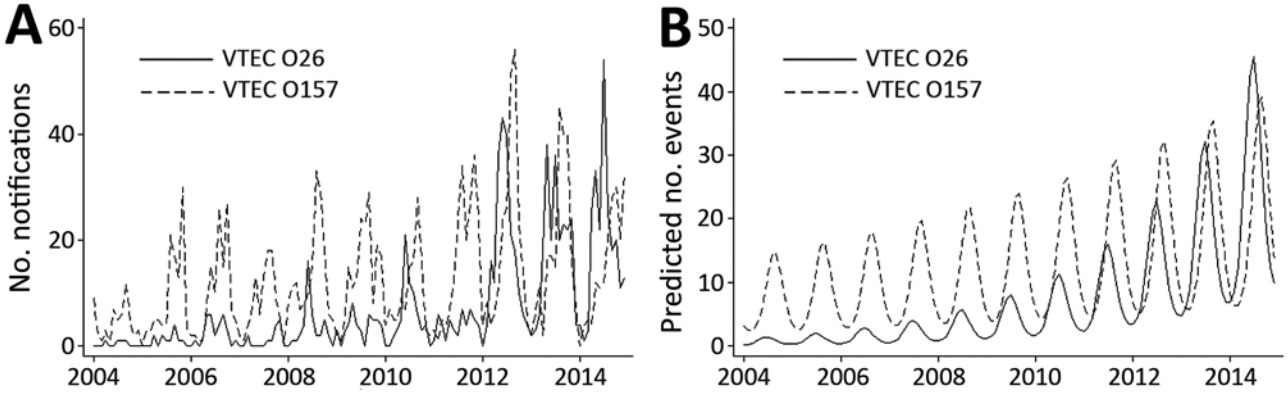

Figure. Verotoxigenic *Escherichia coli* (VTEC) O157 and VTEC O26, Ireland, 2004–2014. A) Seasonal distribution of notifications. B) Predicted seasonal distribution. Data source: Computerised Infectious Disease Reporting System (https://www.hpsc.ie/Notifiable Diseases) in Ireland, as of June 24, 2015. Predicted number of cases by month were derived from a cyclical quasi-Poisson model after trend and seasonality and interaction by serogroup were accounted for.

as carriers of O157 and O26 strains in Ireland (*7*,*8*). In Germany, cattle density has been shown to be significantly associated with human VTEC O157 incidence but only marginally associated with O26 incidence (*9*); the same study showed no association between cattle density and VTEC O91 infection, indicating that not all serogroups necessarily share the same reservoirs. Alternatively, animals of the same species may be preferentially colonized with different serogroups at different times of the year or at different developmental ages. Other possible explanations could be variation in survival characteristics between the 2 strains, which results in a different seasonal distribution in the environment, or specific human behavior (e.g., seasonal food) resulting in more frequent exposure to sources of VTEC O157 and VTEC O26 at different times of the year.

The consistent differences in seasonality identified here between the 2 most common VTEC serogroups suggest the existence of noteworthy underlying differences in disease etiology between the strains. Further exploration is recommended.

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New Delhi Metallo-β-Lactamase-1–Producing *Klebsiella pneumoniae*, Florida, USA[1]

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To the Editor: New Delhi metallo-β-lactamase (NDM)–producing *Enterobacteriaceae* have swiftly spread worldwide since an initial report in 2008 from a patient who had been transferred from India back home to Sweden (*1*). Epidemiologically, the global diffusion of NDM-1 producers has been associated with the Indian subcontinent and the Balkan region, which are considered the primary and secondary reservoirs of these pathogens, respectively (*1*). However, recent reports suggest that countries in the Middle East may constitute another potential reservoir for NDM-1 producers (*1*). More than 100 NDM-producing isolates have been reported in the United States, most of

[1]Preliminary results from this study were presented at the 54th Interscience Conference on Antimicrobial Agents and Chemotherapy, September 5–9, 2014, Washington, DC, USA.