days, hypoxia 5 (IQR 3.5–7.5) days, and admission, 6 (IQR 4–8) days.

We compared the data from those recent patients with data from 498 cases of hMPV infection at the hospital during 2005–2020. During that period, 9 (1.8%) cases were detected in November–December and 453 (90.9%) during February–May. Patients in the 2021 season were older than those from the previous 15-year period and had significantly higher rates of hypoxia, pneumonia, antimicrobial drug treatment; they also had longer durations of fever, hypoxia, hospital admission, and PICU admission (Table 1).

In November–December 2021, during the sixth wave of COVID-19 in Spain, the country experienced an extemporaneous hMPV outbreak at the time when RSV epidemic was usually observed. This outbreak of hMPV infections affected children older than were usually affected in previous years; we also observed a more severe clinical course and higher rates of hypoxia, pneumonia, and admission to PICU than historically.

We considered that there may have been competition between respiratory viruses that could justify the delay in the RSV outbreak (5), which occurred in summer (June–July) 2021 in Spain; such competition was not the case for the hMPV outbreak, which coincided with spread of the Omicron variant of SARS-CoV-2. One possible explanation is relaxation of social distancing measures or the extreme contagiousness of Omicron. The increased severity of illness could be partly explained by the absence of hMPV infections in the previous 2 years, resulting in a susceptible population of older children who had not had previous hMPV infections and therefore had no immunity. In previous seasons, children >1 year of age were immunized by previous infections or even through residual maternal protection; this protection did not exist in 2021, and older children were infected. In conclusion, this outbreak illustrates that clinicians should be aware of potential differences in the epidemiology of other viral respiratory infections during and after the COVID-19 pandemic.

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Highly Pathogenic Avian Influenza A(H5N1) Virus in a Harbor Porpoise, Sweden

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We found highly pathogenic avian influenza A(H5N1) virus clade 2.3.4.4b associated with meningoencephalitis in a stranded harbor porpoise (*Phocoena phocoena*). The virus was closely related to strains responsible for a concurrent avian influenza outbreak in wild birds. This case highlights the potential risk for virus spillover to mammalian hosts.

Europe and, more recently, the Americas are experiencing unprecedented mortality in wild and domestic birds because of the highly pathogenic avian influenza virus (HPAI) A(H5N1) virus clade 2.3.4.4b (*1*). Infections in tens of thousands of wild birds representing at least 112 species, including large numbers of seabirds, have been documented (*1,2*). Since December 2021, HPAI H5N1 has dominated infections in wild birds in Sweden, other countries in Europe, and the Americas, and it has spilled over into wild mammals, such as red foxes and mustelids (*1*). Increased mortality in harbor seals (*Phoca vitulina*) and gray seals (*Halichoerus grypus*) in eastern North America has been associated with HPAI H5N1 infection (*3*). Although influenza A virus (IAV) infections in seals have been repeatedly documented, reports in cetaceans are scarce (*4*). We are aware of only 2 cases where IAV in a cetacean might have been associated with disease (*4,5*). We report HPAI H5N1 infection in a harbor porpoise (*Phocoena phocoena*) in Sweden.

In late June 2022, an immature male harbor porpoise became stranded in shallow water off the west coast of Sweden (58.64817 N, 11.28973 E). It swam in circles, was unable to right itself, and drowned shortly after discovery. The carcass was stored frozen until necropsy examination at the National Veterinary Institute (Uppsala, Sweden). Macroscopic findings were minimal and included pulmonary edema consistent with drowning. Microscopically, we detected moderate lymphoplasmacytic meningoencephalitis with neuronal necrosis, gliosis, perivascular cuffing, and vasculitis in the brain (Figure 1, panel A). The lung contained few areas of mild, mononuclear septal thickening and increased numbers of alveolar macrophages.

Stranded porpoises in Sweden are screened routinely for cetacean morbilliviruses, which can be neurotropic in cetaceans, and IAV, which can be neurotropic in other species (*4,6*). We analyzed pooled lung, spleen, and brain samples for cetacean morbilliviruses by using real-time reverse transcription PCR (*6*) with the addition of an in-house designed hydrolysis probe (6FAM-TGG TTC AAC CAG GYA G-MGB) for detection. No morbilliviral RNA was found. We detected IAV genome from lung and bronchial swab specimens (*7*) and subtyped the virus as HPAI H5N1; viral genome sequences were determined directly from tissue samples. Phylogenetic analysis of the complete genome sequences (GISAID accession nos. EPI2150621–8) classified the virus as H5N1 clade 2.3.4.4b. The genome contained no genetic motif of mammalian adaptation besides those already described for H5 clade 2.3.4.4 (HA-H5 172A-Airborne transmission, M1 N30D, Y215A-Virulence// NS1 P42S, L103F and I106M-Virulence) (*8*). Detecting IAV
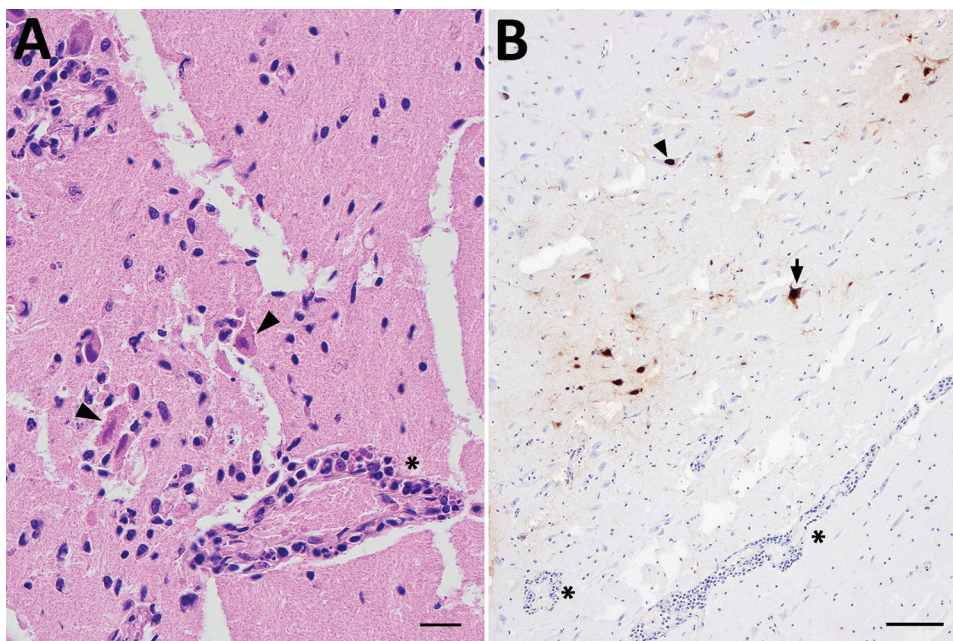

Figure 1. Microscopic analyses of tissue samples from a harbor porpoise (*Phocoena phocoena*) infected with highly pathogenic avian influenza virus H5N1 clade 2.3.4.4b, Sweden. A) Brain tissue showing neuronal necrosis (arrowheads) and perivascular lymphoplasmacytic cuffing of vessels and vasculitis (asterisk). Scale bar represents 20 μm. B) Immunohistochemical labeling of influenza A nucleoprotein in neuronal nuclei (arrowhead) and cytoplasm (arrow), as well as glial cells. Perivascular cuffing (asterisks) is seen in close association to influenza A immunolabeling. Scale bar represents 100 μm.

in respiratory tract swab specimens prompted us to analyze other organs. We detected the highest IAV loads in the brain (cycle threshold [Ct] value 20.57) and smaller loads in the lungs (Ct 30.72), kidney (Ct 31.37), liver (Ct 32.75), and spleen (Ct 33.43). We detected no virus in the intestine, muscle, or blubber.

We performed immunohistochemical analysis using a commercial influenza A nucleoprotein primary monoclonal antibody (EBS-I-238; Biologicals Limited, https://biologicals-ltd.com) as previously described (9) on all organs containing IAV genome to examine viral antigen distribution and the relationship to pathological lesions. We noted multifocal areas of moderate immunolabelling in the brain in nuclei and cytoplasm of neurons (Figure 1, panel B), glial cells, and epithelial cells of the choroid plexus. Scant intranuclear and intracytoplasmic viral antigen was in scattered cells in alveoli (macrophages or sloughed epithelium). We did not observe any viral antigen in other tissues examined.

IAV infection in a harbor porpoise represents expanding viral host range. Infections in cetaceans can result in animal death, as demonstrated by the abnormal behavior and subsequent drowning caused by the meningoencephalitis associated with infection. Virus was found predominantly in the brain, a finding consistent with H5N1 clade 2.3.4.4b infection in other mammals (10). Routine examination of the brain is warranted in cetacean disease surveillance, and IAV infection should be considered in animals demonstrating abnormal behavior or neuropathology. The virus detected in this porpoise was most closely related to viruses circulating in wild birds at the same time and location (Figure 2, https://wwwnc.cdc.gov/EID/article/29/4/22-1426-F2.htm), indicating likely spillover from wild birds. The route of transmission is unknown but includes contact with infected birds or indirect contact through contaminated water, suggesting that infection pressure in the ecosystem was high.

Although the genome of the detected HPAI H5N1 virus did not contain any known genetic marker of mammal adaptation, the clinical manifestations and presence of virus in diverse organs, including the brain, indicate the potential risk of HPAI viruses to mammalian hosts even without adaptation. This risk is a consideration for persons in close contact with infected animals. In addition, extensive circulation of the HPAI H5Nx virus clade 2.3.4.4b in wild and domestic bird populations and sporadic transmission to humans and other mammals enables the virus to evolve, increasing the risk of it becoming more transmissible or pathogenic for mammals.

Understanding the epidemiology and host–pathogen environmental ecology of IAVs among wildlife, coupled with continuous surveillance, developing better tools for risk assessments, and updating public and animal health countermeasures and intervention strategies, are essential for reducing the threats of zoonotic influenza.

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SARS-CoV-2 Omicron Replacement of Delta as Predominant Variant, Puerto Rico

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We reconstructed the SARS-CoV-2 epidemic caused by Omicron variant in Puerto Rico by sampling genomes collected during October 2021–May 2022. Our study revealed that Omicron BA.1 emerged and replaced Delta as the predominant variant in December 2021. Increased transmission rates and a dynamic landscape of Omicron sublineage infections followed.

Since the arrival of SARS-CoV-2 in Puerto Rico in March 2020, epidemic waves of COVID-19 have occurred on the island during the emergence of several variants of concern. Genomic surveillance conducted by partnered public health and academic groups reported an epidemic wave caused by the Alpha variant in April 2021, which coincided with the vaccination campaign for adults (*1*). Despite the detection of other variants of interest or concern, circulation of most of those variants was limited. According to the Puerto Rico Department of Health, the Delta variant has caused >49,000 confirmed cases since June 2021 (Appendix 1, https://wwwnc.cdc.gov/EID/article/29/4/22-1700-App1.pdf). The epidemic wave began to decline in August 2021, reaching its lowest rate since the beginning of the Delta wave in December 2021. This decline was possibly associated with the successful COVID-19 vaccination program, in which 83% of the eligible population of Puerto Rico had received the initial series of COVID-19 vaccines by October 31, 2021 (*2,3*).

The first confirmed case of the Omicron variant in Puerto Rico was reported on November 29, 2021, and within a week, Omicron had replaced Delta to become the dominant circulating variant. The relatively low circulation of Delta, combined with Omicron's high transmissibility and the waning of protective immunity before the vaccine booster campaign, might all have contributed to the rapid spread of this variant (*4*). The first Omicron peak was 9.1 times higher than any previous SARS-CoV-2 epidemic peak documented in Puerto Rico (Appendix Figure 1). By May 31, 2022, epidemic waves of the Omicron variant had caused ≈494,200 cases, peaking appreciably around January and May 2022.

We analyzed the Delta and Omicron sublineage turnover dynamics by using all the SARS-CoV-2