


Another Dimension

Thoughtful essays, short stories, or poems on philosophical issues related to science, medical practice, and human health. Topics may include science and the human condition, the unanticipated side of epidemic investigations, or how people perceive and cope with infection and illness. This section is intended to evoke compassion for human suffering and to expand the science reader’s literary scope. Manuscripts are selected for publication as much for their content (the experiences they describe) as for their literary merit.
LETTERS

adsorbed with purified pH1N1 and dk/NY/96 virions. Adsorption with pH1N1, but not dk/NY/96, removed HI antibodies to pH1N1, whereas adsorption with either virus removed HI antibodies against dk/NY/96 (online Technical Appendix Table 4). A comparison of amino acid sequences comprising the known HA antigenic sites on the pH1N1 structure confirmed high sequence identity and structural similarity with dk/NY/96 HA in Sa (12/13 aa residues) and Sb (8/12 aa residues) antigenic sites (data not shown). These results indicate that HI antibodies detected in sea otters are the result of pH1N1 virus infection but cross-react with the avian influenza A(H1N1) virus.

Potential contact between northern elephant seals and sea otters is one possibility; elephant seals’ summer feeding ranges and breeding areas along the Northeast Pacific coast overlap with areas where the Washington sea otter population is distributed (1)

In conclusion, our results show that sea otters are susceptible to infection with influenza A virus and highlight the complex nature of interspecies transmission of influenza viruses in the marine environment. Further surveillance, especially in other sea otter populations, is required to determine virus origin, potential pathogenesis, and consequences for the marine ecosystem.

Acknowledgments

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Sample collection was done in collaboration with United States Geological Survey National Wildlife Health Center, Madison, Wisconsin, USA (H.S. Ip, C.L. White); and Monterey Bay Aquarium, Monterey, California, USA (M.J. Murray)

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Figure. Results of ELISA and hemagglutination inhibition (HI) testing for influenza viruses in serum samples from northern sea otters captured off the coast of Washington, USA, during studies conducted in August 2011 (n = 30) and 2001–2002 (n = 21). A) IgG for influenza A(H1N1) pdm09 strain A/Texas/05/2009 detected by using standard indirect ELISA techniques with HRP-Protein A (Sigma, St. Louis, MO, USA). The ELISA titer was read as the reciprocal of the highest dilution of serum with an OD<sub>450</sub> of ≥0.2 and 2-fold higher than the OD<sub>450</sub> of control wells lacking serum. B) HI for influenza A(H1N1)pdm09 strain A/Mexico/4108/2009. HI titers were determined by using 0.5% turkey red blood cells (RBCs) for influenza A(H1N1)pdm09, seasonal influenza A(H1N1), influenza (H3N2), and influenza B viruses that circulated in North America during 2000–2011 and by using 1% horse RBCs supplemented with 0.5% BSA for avian influenza A(H1N1) virus strain A/duck/New York/96. HI assay was performed as described (www.who.int/influenza/gisrs_laboratory/manual_diagnosis_surveillance_influenza/en). OD, optical density.

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To the Editor: Porcine epidemic diarrhea (PED) was first reported in the United Kingdom in 1971 (1). The disease was characterized by severe enteritis, vomiting, watery diarrhea, dehydration, and a high mortality rate among swine. Subsequently, the causative agent of PED was identified as porcine epidemic diarrhea virus (PEDV), which belongs to the family Coronaviridae (2) and contains an enveloped, single-stranded positive-sense RNA genome. PEDV has been reported in many other countries, including Germany, France, Switzerland, Hungary, Italy, China, South Korea, Thailand, and Vietnam (3) and was first identified in the United States in May 2013. By the end of January of 2014, the outbreak had occurred in 23 US states, where 2,692 confirmed cases (www.aasv.org/news/story.php?id = 6989) caused severe economic losses. Recent studies have shown that all PEDV strains in the United States are clustered together in 1 clade within the subgenogroup 2a and are closely related to a strain from China, AH2012 (4,5).

In the state of Ohio, the first PED case was identified in June of 2013; since then, hundreds of cases have been confirmed by the Animal Disease Diagnostic Laboratory of the Ohio Department of Agriculture. In January of 2014, samples from pigs with unique disease, suspected to be PED, were submitted to this laboratory. Sows were known to be infected, but piglets showed minimal to no clinical signs and no piglets had died.

According to real-time reverse transcription PCR, all samples from the piglets were positive for PEDV. Subsequently, the full-length genome sequence of PEDV (OH851) was determined by using 19 pairs of oligonucleotide primers designed from alignments of the available genomes from PEDVs in the United States (6,7). On the basis of BLAST (http://blast.ncbi.nlm.nih.gov/Blast.cgi) searches, strain OH851 showed 99% and 97% nt identity to PEDVs currently circulating in the United States (Colorado, Iowa, Indiana, Minnesota) for the whole genome and the full-length spike (S) gene, respectively. By distinct contrast, strain OH851 showed only 89% or even lower nucleotide identity to PEDVs currently circulating in the United States in the first 1,170 nt of the S1 region. In that region, nucleotide similarity to that of a PEDV strain from China (CH/HBQX/10, JS120103) was 99%, suggesting that strain OH851 is a new PEDV variant.

Phylogenetic analysis of the complete genome indicated that the novel OH851 PEDV is clustered with other strains of PEDV currently circulating in United States, including another strain from Ohio, OH1414 (Figure, panel A). However, phylogenetic analysis of the full-length S gene showed that strain OH851 is clustered with other strains of PEDV from China and most closely related to a PEDV strain from China, CH/HBQX/10 (8), but distantly related to other PEDV strains currently circulating in the United States and strain AH2012 (Figure, panel B). This finding strongly suggests that strain OH851 is a variant PEDV. In comparison with the S gene of other strains from the United States, the S gene of strain OH851 has 3 deletions (a 1-nt deletion at position 167, a 11-nt deletion at position 176, and a 3-nt deletion at position 416), a 6-nt insertion between positions 474 and 475.