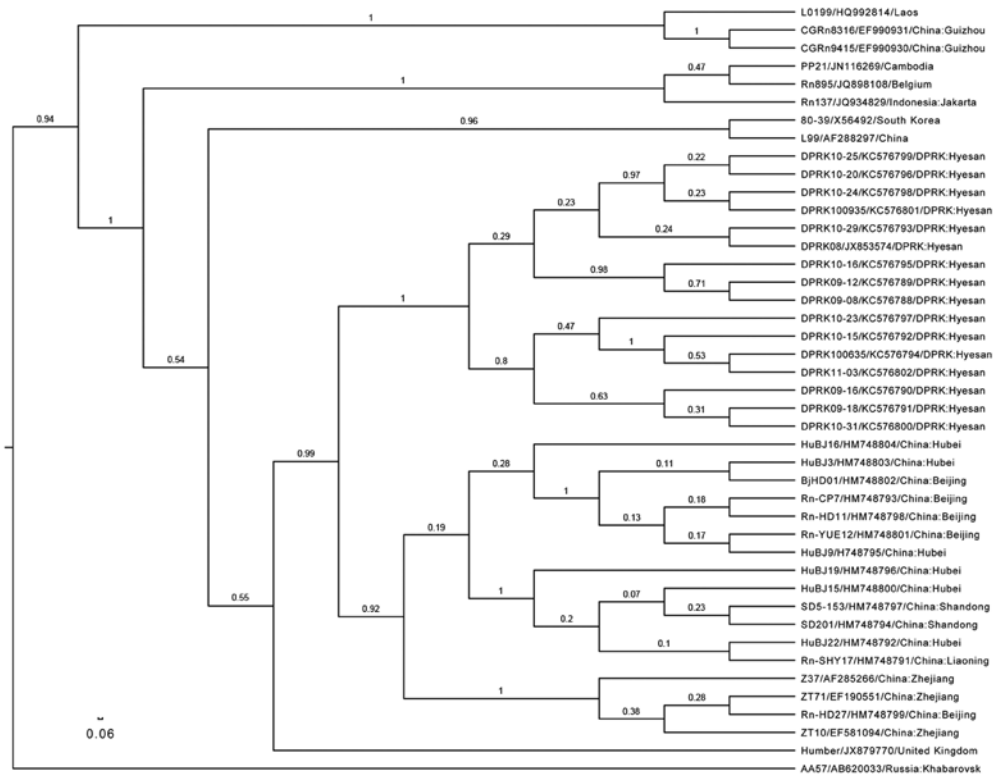

Figure. Phylogenetic tree, based on a 330-bp amplicon of the Seoul virus (SEOV) RNA-dependent RNA polymerase gene, depicted in FigTree 1.4.0 (www.molecularevolution.org/software/phylogenetics/figtree). The tree was generated by using the uncorrelated lognormal distribution relaxed molecular clock model and SRD06 substitution model in BEAST1.74 (*7*). SEOV strain name/GenBank accession no/country: The location is shown in taxa. The posterior number is shown for each branch. Clades A and D were established as described (*2*) Scale bar represents number of nucleotide changes per site.

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Schmallenberg Virus Infection in Dogs, France, 2012

To the Editor: In 2011, Schmallenberg virus (SBV) emerged in Europe (*1*); the virus spread into France in January 2012 (*2*). During January–March 2012, a total of >1,000 cases were reported in France, mainly in stillborn and newborn lambs with congenital malformations.

In March 2012, neurologic disorders were detected in five 15-day-old puppies (Belgian shepherd) from a dog breeding kennel in northwestern France (Orne). We report data suggesting that these puppies were infected with SBV.

In June 2012, the kennel veterinarian contacted a veterinary school (Unité de Médecine de l'Elevage et du Sport Breeding and Sport Medicine Unit, Maisons-Alfort, France) after neurologic signs of ataxia, exotropia, a

head tilt, and stunted growth were observed in a litter of 5 puppies. Four of the puppies had died at 5–6 weeks of age. The veterinarian collected blood samples from the surviving puppy at 3 months of age, and the puppy was euthanized for necropsy. Severe torticollis was observed during the necropsy, but no other macroscopic signs were detected. The brain, including the cerebellum; a part of the spine; and cerebrospinal fluid (CSF) were collected for further investigation. Specific PCR analyses for canine coronavirus, *Neospora caninum*, *Toxoplasma gondii*, and canine minute virus were performed on CSF; all test results were negative. The brain tissue was fixed in formalin and processed for histologic examination. Features of degenerative encephalopathy, including neuronal vacuolation, neuropil vacuolation, and minimal gliosis, were observed.

Because some clinical signs were evocative of SBV infection and the puppy was born in an area where the virus was circulating actively in cattle and sheep, veterinarians decided to investigate SBV as a possible etiology. Serum samples from the 3-month-old puppy and the dam were tested by virus neutralization test (VNT), according to the protocol used for ruminant serum. The results were negative for the puppy but positive (titer 128) for the mother. Specific competitive SBV ELISA (IDVet, Montpellier, France) against the SBV N protein showed similar results.

Real-time reverse transcription PCR (RT-PCR) was performed (*3*) to detect the presence of the SBV genome in the cerebellum. Because the sample was paraffin-embedded, RNA was extracted from 5-μm sections, as described (*4*). All of the extracted cerebellum sections had positive test results (cycle threshold range 33-36); the extraction and PCR controls all showed negative results. To confirm these positive results, conventional RT-PCR was used to amplify a 573-nt sequence of the SBV S segment. The amplification product was sequenced, and a BLAST analysis was performed (www.ncbi.nlm.nih.gov/BLAST). An identity of 100% was obtained with the SBV small gene segment from a ruminant (GenBank accession no. KC108860). An immunohistochemical assay was also performed; the result was negative.

The remaining 7 female dogs in the breeding kennel were tested for SBV in October 2012; 1 showed positive test results by VNT (titer 256), which confirmed that SBV was circulating in the kennel. This positive dam had a litter of puppies in December 2012, but no signs developed, and the puppies were not tested. In March 2013, repeat testing was done on serum samples from the 2 dogs that had shown positive results. Results for both animals were positive by VNT (titers 32 for the dam and 128 for the other dog) and ELISA.

Taken together, specific SBV antibodies in the mother and the SBV genome in her puppy suggest that these dogs experienced SBV infection. The absence of detectable SBV antibodies in the puppy in this investigation suggests that transplacental infection occurred before the onset of fetal immune competence. Maternal infection probably occurred in January or February 2012; entomologic monitoring conducted in France showed the presence of *Culicoides* spp. midges, a vector of SBV, during this period in northwestern France. In addition, because the puppies were born in March 2012 and SBV antibodies were still detectable in the mother in March 2013, the duration of SBV antibodies in dogs appears to be ≥1 year. In cattle and sheep, the SBV genome persists in an infected fetus and is detectable after birth by real-time RT-PCR, despite gestation length (*5*,*6*).

Few reports on orthobunyavirus infections in dogs are available. Two serologic studies from the United States (*7*) and Mexico (*8*) found antibodies against La Crosse virus, South River virus, and Jamestown Canyon virus in dogs. Two other reports described cases in which La Crosse virus was detected in canine littermates who had clinical encephalitis (*9*) or neurologic disorders (*10*).

It is unclear if the apparent SBV infection we detected in these dogs was an isolated event or if other cases occurred elsewhere but were not detected because they were not investigated. Further serologic and clinical surveys are needed to estimate SBV prevalence in dogs and the virus' involvement in the occurrence of neurologic signs in puppies.

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Geographic Co-distribution of Influenza Virus Subtypes H7N9 and H5N1 in Humans, China

To the Editor: Human infection with a novel low pathogenicity influenza A(H7N9) virus in eastern China has recently raised global public health concerns (*1*). The geographic sources of infection have yet to be fully clarified, and confirmed human cases from 1 province have not been linked to those from other provinces. While some studies have identified epidemiologic characteristics of subtype H7N9 cases and clinical differences between these cases and cases of highly pathogenic influenza A(H5N1), another avian influenza affecting parts of China (*2–4*), the spatial epidemiology of human infection with influenza subtypes H7N9 and H5N1 in China has yet to be elucidated. To test the hypothesis of co-distribution of high-risk clusters of both types of infection, we used all available data on human cases in mainland China and investigated the geospatial epidemiologic features.

Data on individual confirmed human cases of influenza (H7N9) from February 19, 2013, through May 17, 2013, and of influenza (H5N1) from October 14, 2005, through May 17, 2013, were collected from the Chinese Center for Disease Control and Prevention. The definitions of these cases have been described (*3*,*5*). A total of 129 confirmed cases of influenza (H7N9) (male:female ratio 2.39:1) and 40 confirmed cases of influenza (H5N1) (male:female ratio 0.90:1) were included in the analysis. The median age of persons with influenza (H7N9) was higher than for persons with influenza (H5N1) (58 years vs. 27 years; $z = -7.73$; $p<0.01$). Most (75.0%) persons with influenza (H5N1) had direct contact (e.g., occupational contact) with poultry (including dead and live birds) or their excrement and urine, whereas most (64.3%) persons with influenza (H7N9) had only indirect exposure to live poultry, mainly during visits to live poultry markets.

Reported cases of influenza (H5N1) were distributed over 40 townships in 16 provinces, whereas cases of influenza (H7N9) were relatively more concentrated, in 108 townships but only 10 provinces (Figure). To identify a spatial overlap between the primary cluster of influenza (H7N9) cases, detected in April 2013 (relative risk [RR] 78.40; $p<0.01$), and the earliest space-time cluster of influenza (H5N1) cases, detected during November 2005–February 2006 (RR 65.27; $p<0.01$), we used spatiotemporal scan statistics with a maximum spatial cluster size of 5% of the population at risk in the spatial window and a maximum temporal cluster size of 25% of the study period in the temporal window (*6*) (Figure). The results suggest that the overlap is not perfect and is concentrated around an area southeast of Taihu Lake (south of Jiangsu Province), bordering the provinces of Anhui and Zhejiang. Smaller clusters of influenza (H7N9) cases were identified in the boundary of Jiangsu and Anhui Province (8 cases; RR 64.86; $p<0.01$) and Jiangxi Province (Nanchang County and Qingshanhu District) (4 cases; RR 105.67; $p<0.01$). A small cluster of influenza (H5N1) cases was detected during 2012–2013 along the boundaries of Guanshanhu, Yunyan, and Nanming Counties in Guizhou Province (3 cases; RR 496.60; $p<0.01$).

In addition, family clustering, defined as $\geq$2 family members with laboratory-confirmed cases, was found for influenza (H7N9) cases during March–April 2013 in Shanghai and Jiangsu Provinces and for influenza (H5N1) cases during December 2007 in Jiangsu Province.