the genera *Eptesicus* and *Rhinolophus* in South Korea. However, nucleotide sequencing showed the presence of prototypical Hantaan virus indicating a spillover infection or laboratory contamination (*7*).

Further screening is necessary to confirm *N. hispida* as a natural reservoir host of the virus. Although the presented bat-associated sequence is obviously distinct from other hantaviruses, which suggests association with a novel natural host, a spillover infection from another, yet unrecognized host cannot be ruled out. However, detection of the virus exclusively in 1 organ (lung but not in liver, kidney, and spleen; data not shown) suggests a persistent infection that is typically observed in natural hosts of hantaviruses (*8*).

To date, only a few reports exist on cases of hemorrhagic fever with renal syndrome in Africa (*9,10*). However, underreporting must be assumed because the symptoms resemble those of many other febrile infections. Moreover, in cases of infections by non–rodent-associated hantaviruses, cross-reactivity with routinely used rodent-borne virus antigens should be limited and may hamper human serodiagnostics (*1*). The results suggest that bats, which are hosts of many emerging pathogens (*5*), may act as natural reservoirs for hantavirus. The effect of this virus on public health remains to be determined.

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Outbreak of Porcine Epidemic Diarrhea in Suckling Piglets, China

To the Editor: Beginning in October 2010, porcine epidemic diarrhea (PED), caused by a coronaviral infection affecting pigs, emerged in China in an outbreak characterized by high mortality rates among suckling piglets. The outbreak overwhelmed >10 provinces in southern China, and >1,000,000 piglets died. This outbreak was distinguished by ≈100% illness among piglets after birth (predominantly within 7 days and

sometimes within only a few hours) and death rates of 80%–100% (online Technical Appendix Table 1, wwwnc.cdc.gov/EID/pdfs/11-1259-Techapp.pdf). Few sows or boars showed any clinical signs during the outbreak, which is not consistent with a recent report from Thailand (*1*). In that outbreak during late 2007, pigs of all ages were affected, exhibiting different degrees of diarrhea and no appetite. We characterized the genetic variation of the PED virus (PEDV) that caused a large-scale outbreak in China during 2010–2011 and compared it with viruses in other outbreaks. We also report a possible novel transmission pathway for PEDV.

A total of 177 samples (intestine, stool, and maternal milk) were collected from pigs from different farms who had diarrhea; 100% of farms had ≥1 porcine sample positive for PEDV. A total of 125/177 porcine samples were confirmed as positive for PEDV by reverse transcription PCR using primers as described (*2*). PEDV was detected in 105 (82.0%) of 128 fecal samples and 20 (40.8%) of 49 sow milk samples. Piglets infected with PEDV showed mild hemorrhage, undigested curdled milk in the stomach, and thin-walled intestines with severe mucosal atrophy and foamy fluid (data not shown).

The spike (S) gene of the family *Coronaviridae* has a high degree of variation and can induce neutralizing antibody (*3*). Reverse transcription PCR products of the 651-bp partial S gene of PEDV and the deduced amino acid sequences were aligned by using ClustalW (www.genome.jp/tools/clustalw), and a neighbor-joining tree with 1,000 bootstraps was constructed. Sequences of the S genes from this outbreak were 99.1%–100.0% homologous and had 88.7%–98.9% nt identity with all reference strains (online Technical Appendix Table 2), 98.5%–98.9% with Thailand strains, and 94.5%–95.1% with vaccine strain CV777. The partial S gene deduced amino acid sequences were compared and also showed a high degree of homology (98.0%–100.0%); they had 85.3%–98.7% identity with all reference strains listed in online Technical Appendix Table 2, 98.0%–98.7% with Thailand strains, and 93.3%–94.7% with vaccine strain CV777 (data not shown).

Phylogenetic analysis indicated that the PEDV in the China outbreak was different from foreign and other domestic strains on the basis of the reported partial S gene sequences. All new strains were clustered in the same branch, close to the cluster of Thailand strains, and far from the cluster of vaccine strain CV777 (Figure).

In the China outbreak, PEDV caused severe diarrheal disease in piglets; heavy economic losses in many provinces resulted, despite use of commercial vaccines (inactivated transmissible gastroenteritis [TGEV H] and porcine epidemic diarrhea [CV777]). To determine why the vaccines showed poor efficacy, we investigated evolution of the virus. Comparison of amino acid sequences from isolates from the outbreak and from the CV777 vaccine strain showed 9 amino acid mutations of fragments containing major hydrophilic regions: 16 (L→H), 18 (S→G), 22 (V→I), 44 (T→S), 89 (G→S), 100 (A→E), 107 (L→F), 130 (I→V) and 160 (I→F) (online Technical Appendix Figure, panel A). Three of these 9 mutations were at positions 16, 18, and 22 in the isolates from China; they influenced the hydrophobicity of the S protein as compared with that for CV777 (online Technical Appendix Figure, panel B).

Phylogenic analysis showed that strain CV777 did not cluster with current common strains and showed

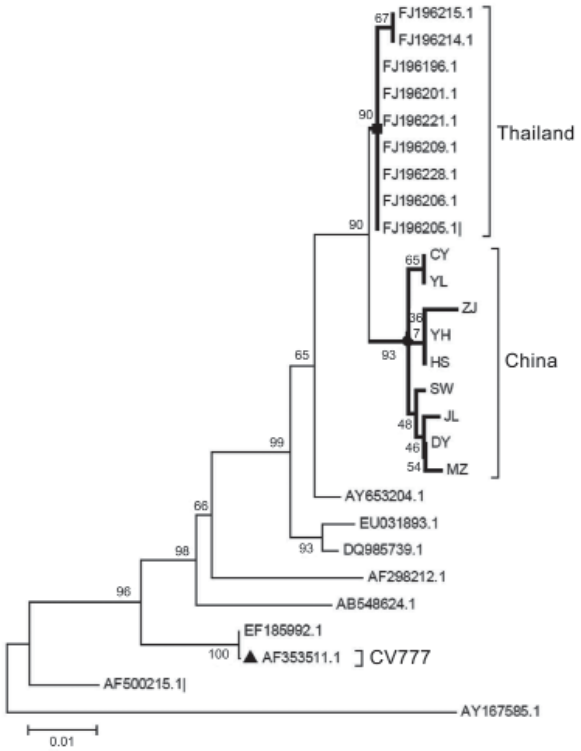

Figure. Phylogenetic tree constructed by using the neighbor-joining method based on the 9 porcine epidemic diarrhea virus (PEDV) sequences identified in a study of porcine epidemic diarrhea in China. Partially amplified spike genes of the PEDV isolates plus 18 PEDV sequences downloaded from GenBank were compared. Sequences included in each cluster are listed in online Technical Appendix Table 3 (wwwnc.cdc.gov/EID/pdfs/11-1259-Techapp.pdf). Strains from Thailand and China and the CV777 vaccine strain are indicated. Scale bar indicates nucleotide substitutions per site.

considerable genetic distance from them. Isolates in the outbreak in China had only a minor nucleotide sequence variation from the Thailand isolates, indicating that the virus has a high genetic relatedness to the Southeast Asia strain. However, previous studies showed that isolates from Europe, South Korea, and China were serologically identical to the prototype CV777 strain (*1*,*4*).

To our knowledge, fecal–oral transmission is probably the main or only route of PEDV transmission (*5*–*7*). In our study, if a fecal sample from a sick piglet was found to be positive for PEDV, we also collected and studied milk from its mother. These results showed that PEDV was present in sow milk (online Technical Appendix Table 3), but the detection rate was lower for these samples (40.8%) than for the fecal samples (82.0%).

On the basis of these results, we hypothesize that sow milk could represent a possible (and potentially major) route for the vertical transmission of PEDV from sow to suckling piglet. This hypothesis could be indirectly verified by our field observation that piglet death rates decreased as a result of fostering (data not shown). Our findings show that PEDV was identified not only in fecal samples from sick piglets, as expected, but also in the milk of the sow, which suggests vertical transmission of the virus.

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Bartonella quintana Transmission from Mite to Family with High Socioeconomic Status

To the Editor: Urban trench fever caused by *Bartonella quintana* has been reported in persons who abuse alcohol and in homeless persons in large cities worldwide. Symptoms vary from asymptomatic intermittent bacteremia to serious complications (*1*). *Pediculus humanus* mites, the known vector of the infection, are not always identified, which raises the possibility that other vectors might also be involved (*2*). We report on an outbreak of *B. quintana* infection among a young family of high socioeconomic status and their visiting relatives.

The family resides in a regional city (population 104,000) in northern Czech Republic in an old, renovated apartment located on the top floor, just under the roof. In the summer of 2007, hundreds of ectoparasitic mites migrated from a whole in the roof and settled on the inner side of a permanently open window before infesting family members. Two weeks later (day 1 of symptom onset), a papular rash and pruritic vesicular lesions were noted by the parents on the body and legs of their 2 children, a 1-year-old girl and a 3-year-old boy. On day 3, the girl's body temperature rose to 38.0°C, and the boy's temperature rose to 39.5°C. The rash resolved in ≈10 days in both children. Vesicular lesions on the girl's buccal mucosal membrane resolved in 5 days. Excoriated areas resulting from spontaneous rupture of lesions or scratching were still visible on day 14.

On day 4, a fever (temperature, 38.5°C) and intense tibialgia, which persisted for 5 days, developed in the 33-year-old father of the infected children. On day 5, a vesicular rash, which resolved in 10 days, developed in the 33-year-old mother. The children's

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Outbreak of Porcine Epidemic Diarrhea in Suckling Piglets, China

Technical Appendix

Table 1. Current farms status in this study, China

Farm	No. sows	Vaccination*	Illness rate ,%/y	Mortality rate. %
YL	1,000	Yes	90	50
HS	1,400	No	100	80
CY	300	Yes	80	90
SW	1,000	Yes	80	100
DY	2,000	Yes	95	100
JL	80	No	75	80
ZJ	90	No	100	90
MZ	100	No	60	100

*Sows were vaccinated with divalent inactivated transmissible gastroenteritis (TGE) and porcine epidemic diarrhea (PED) vaccine before delivery.

Table 2. Accession numbers, district, and collection date of the sequences used*

Accession no.	District	Strain	Collection date
–	Shen-Zhen, Guangdong	JL	2010 Nov
–	Da-Yang, Guangdong	DY	2010 Dec
–	Qing-Yuan, Guangdong	YH	2011 Feb
–	Qing-Yuan, Guangdong	YL	2011 Mar
–	Shi-Wan, Guangdong	SW	2011 Apr
–	Bo-Luo, Guangdong	CY	2011 May
–	He-Shan, Guangdong	HS	2011 May
–	Mei-Zhou, Guangdong	MZ	2011 Jun
	Zhan-Jiang, Guangdong	ZJ	2011 Jul
FJ196196.1	Nakornpathom (Thailand)	07NP01	2007 Dec
FJ196201.1	Chonburi (Thailand)	08CB05	2008 Mar
FJ196205.1	Nakornpathom (Thailand)	08NP03	2008 Jan
FJ196206.1	Nakornpathom (Thailand)	08NP04	2008 Jan
FJ196209.1	Nakornpathom (Thailand)	08NP07	2008 Mar
FJ196214.1	Ratchaburi (Thailand)	08RB02	2008 Jan
FJ196215.1	Ratchaburi (Thailand)	08RB03	2008 Jan
FJ196221.1	Chonburi (Thailand)	KU01CB08	2008 Mar
FJ196228.1	Ratchaburi (Thailand)	KU08RB08	2008 Mar
AY653204.1	Nan-Jing (China)	JS-2004-2	2004 Jul
EU031893.1	Gan-Su (China)	DX	2007 Aug
AF298212.1	South Korea	–	2002 Aug
AF500215.1	South Korea	–	2002 Apr
AY167585.1	South Korea	Chinju99	2002 Oct
EF185992.1	Lan-Zhou (China)	LZC	2006 Dec
AB548624.1	Japan	MK	2010 Mar
DQ985739.1	Hei-Long-Jiang (China)	LJB/03	2006 Sep
AF353511.1	China	CV777	2001 Feb

*–, sequences obtained from present outbreak have no accession no.

Table 3. Detection of PEDV-affected piglets and sows samples by RT-PCR, China*

Farm	Feces and intestine (no. positive/total)	Milk (no. positive/total)
YL	10/10	4/10
HS	24/24	4/6
CY	8/14	2/6
SW	4/6	1/5
DY	26/30	5/12
JL	3/6	–
ZJ	16/22	–
MZ	4/6	–

*PEDV, porcine epidemic diarrhea virus; –, no corresponding samples.

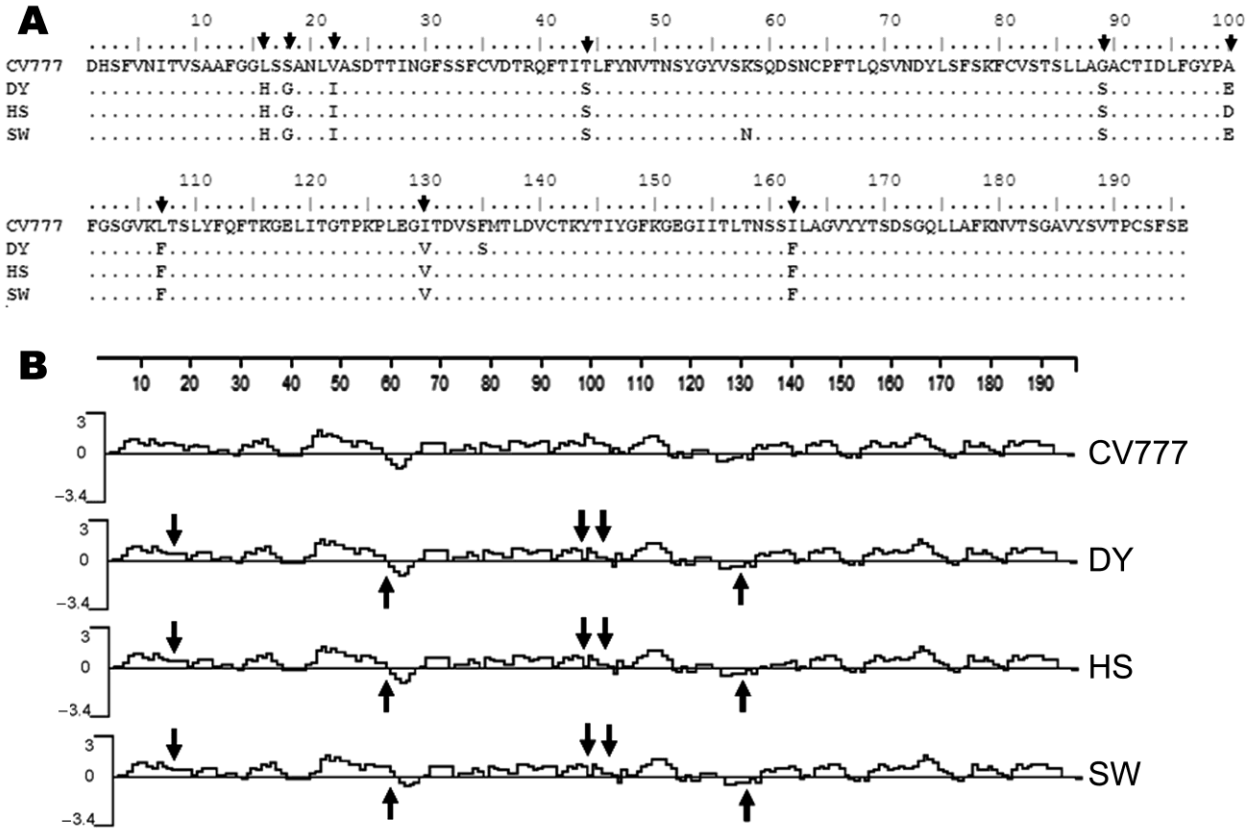

Figure. A) Deduced amino acid sequence comparison of the spike (S) proteins between these isolates from China (DY, HS and SW) and the reference CV777 strain. Only the amino acids different from those in the consensus sequence are indicated. B) Hydrophobicity plots of S generated by the Kyte and Doolittle method by using DNAstar program (www.dnastar.com). Major areas of difference are indicated by arrows.