All patients with seropositive samples lived in the urban centers. The mean age of the 6 patients was 36.0 years (range 24–56 years), and 83% were men. Test results for IgM antibodies to SNV conducted on samples in parallel were negative.

The seroprevalence found in this study was caused by patient exposure to hantavirus. However, in the absence of IgM to SNV, we cannot link the respiratory symptoms observed to recent infection with hantavirus. Lack of information about the patients, especially their clinical history and details of travel to bordering countries, did not permit an association of infection with hantavirus contact in French Guiana. The seroprevalence observed is similar to that in Venezuela, where hantaviruses were isolated from rodents in 1999, but is lower than that observed in regions of Brazil (*10*).

The presence of hantaviruses in neighboring countries, as well as frequent travel by people in and out of French Guiana, has encouraged us to continue studying these viruses. We plan to conduct a study to systematically evaluate hantaviruses by serologic analysis and genomic amplification in persons with suggestive pathology. This study will be carried out in parallel with an investigation of rodent reservoirs of hantaviruses.

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Qinghai-like H5N1 from Domestic Cats, Northern Iraq

To the Editor: Natural infection of several cat species with highly pathogenic avian influenza (HPAI) H5N1 viruses in Thailand (*1–4*) and experimental infection of domestic cats with similar viruses have been reported (*5*,*6*). Thus, literature describing HPAI H5N1 infection of cats is limited to descriptions of infections with a subset of clade I viruses. HPAI H5N1 viruses, highly similar to viruses isolated from Qinghai Lake in western People's Republic of China in spring 2005, are now rapidly disseminating throughout Eurasia and Africa. To our knowledge, this is the first report of a Qinghai-like virus detected in domestic cats. This finding is noteworthy because the host range of influenza viruses is determined by the antigenic characteristics of the hemagglutinin and neuraminidase molecules; clade II viruses are antigenically distinct from clade I viruses, and Qinghai-like viruses are genetically distinct from other clade II viruses.

Personal communications in January 2006 from field veterinarians noted deaths of domestic cats that were associated with suspected (eventually confirmed) H5N1 outbreaks in eastern Turkey (2 villages) and Kurdish northern Iraq (Sarcapcarn in Sulymaniyah Governorate and Grd Jotyar in Erbil Governorate). The clinical conditions of the birds did not suggest HPAI to villagers or consulting veterinarians. In both scenarios in Iraq, results of rapid antigen detection tests with the Anigen kit (Suwon, Republic of Korea), while positive for influenza A, were negative for H5, so the outbreaks were not thought to be caused by HPAI, but concern about the unusual deaths in cats remained.

Because the regions are remote and veterinary services limited, such anecdotal reports have rarely been followed up.

After H5N1 influenza was diagnosed in a person in Sarcapcarn, Kurdish northern Iraq, the government of Iraq requested a World Health Organization investigation, which was supported in part by Naval Medical Research Unit No. 3 veterinarians. While investigating the situation in Erbil Governorate, the team was informed of suspicious deaths in cats associated with the death of all 51 chickens in a household in Grd Jotyar (≈10 km north of Erbil City) From February 3 to February 5, 2006, five cats reportedly died; 2 of these were available for examination on February 8. A sick goose from an adjacent household was killed and underwent necropsy with the cats. Gross pathologic changes in cats were similar to those previously reported, except that severe hemorrhagic pancreatitis was observed (*5*,*6*). Tissues from these animals and archived tissues from 1 of the 51 chickens were conveyed to Cairo for virologic examination.

Table. Detection of influenza A H5 by real-time PCR*

Tissue	Chicken	Goose	Cat 1	Cat 2
Abdominal fluid	ND	+	ND	ND
Blood	ND	–	–	ND
Heart	+	ND	ND	ND
Trachea	ND	–	+	+
Lung	+	+	+	+
Kidney	ND	ND	–	ND
Spleen	ND	–	–	–
Pancreas	ND	ND	+	+
Lymph node	ND	ND	–	ND
Liver	+	ND	–	+
Proventriculus	+	ND	N/A	N/A
Small intestine	+	+	–	ND
Large intestine	ND	–	+	+
Cecum	ND	+	ND	ND
Feces	ND	ND	+	ND

*ND, not done. Samples were tested by real time PCR for influenza A (matrix protein) and if positive, for H5 (*7*). All samples denoted as positive were positive for both influenza A and H5. Chicken samples were obtained previously by local veterinarians based on their sampling protocols. Goose and cat samples were obtained by S. Felt; only grossly abnormal tissues were sampled.

An influenza A H5 virus was present in multiple organs in all species from the outbreak site in Grd Jotyar (Table). cDNA for sequencing was amplified directly from RNA extracts from pathologic materials without virus isolation. On the basis of sequence analysis of the full HA1 gene and 219 amino acids of the HA2 gene, the viruses from the goose and 1 cat from Grd Jotyar and from the person who died from Sarcapcarn (sequence derived from PCR amplification from first-passage egg material) are >99% identical at the nucleotide and amino acid levels (GenBank nos. DQ435200–02). Thus, no indication of virus adaptation to cats was found. The viruses from Iraq are most closely related to currently circulating Qinghai-like viruses, but when compared with A/bar-headed goose/Qinghai/65/2005 (H5N1) (GenBank no. DQ095622), they share only 97.4% identity at the nucleic acid level with 3 amino acid substitutions of unknown significance. On the other hand, the virus from the cat is only 93.4% identical to A/tiger/Thailand/CU-T4/2004 (H5N1) (GenBank no. AY972539). These results are not surprising, given that these strains are representative of different clades (*8*,*9*). Sequencing of 1,349 bp of the N gene from cat 1 and the goose (to be submitted to GenBank) show identity at the amino acid level, and that the N genes of viruses infecting the cat and goose are >99% identical to that of A/bar-headed goose/Qinghai/65/2005(H5N1). These findings support the notion that cats may be broadly susceptible to circulating H5N1 viruses and thus may play a role in reassortment, antigenic drift, and transmission.

The route of infection in these cats cannot be determined definitively. How cats behave when eating birds makes both oral and respiratory infection possible. However, the source of infection seems clear in that an identical H5N1 virus was detected in samples from a goose from the same dwelling, and an H5 virus was detected from archived samples from 1 of 51 chickens that died over the course of a few days. The potential for horizontal spread cannot be ruled out since we detected viral RNA in gut, stool, and trachea; clinical signs developed in all cats, and all died from the acute illness 2–4 days after the chicken deaths began; therefore, simultaneous exposure seems likely. Death in cats, spatially and temporally associated with unusual deaths in poultry, especially when the cats show positive results of a rapid antigen detection test for influenza A, should be considered to indicate a presumptive diagnosis of HPAI, and appropriate response should ensue.

Acknowledgments

We thank Elham Botrus Shabo, Saman Najeeb, Faisal Polus, Sura Jabar, Saidawan Omer Yussif, and Burhan Sulaiman for facilitation and technical assistance in sampling and performing necropsies, and Bradford Camp, Odis Kendrick, and Kosar Shaheer for communications and security.

This work was supported by funding through the Naval Medical Research Center work unit GEIS E0018.

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Classifying *Escherichia coli*

To the Editor: Enteropathogenic *Escherichia coli* (EPEC), 1 of the 6 pathotypes of diarrheogenic *E. coli* (DEC), promotes attaching-effacing lesions in eukaryotic cells. These lesions are mediated by intimin, an outer membrane adhesive protein encoded by the *eae* (*E. coli* attaching-effacing) gene (*1*). EPEC is currently subdivided into typical and atypical subgroups. While typical EPEC carry the EPEC adherence factor plasmid (pEAF) that encodes the bundle-forming pilus (BFP) and a complex regulator of various virulence genes (Per) (*1*), atypical EPEC is devoid of pEAF (or does not express a functional BFP) (*1*,*2*). Typical EPEC expresses the localized pattern of adherence (LA), which is characterized by compact bacterial clusters on HeLa and HEp-2 cells (*1*). Conversely, atypical EPEC most often expresses the LA-like pattern (with loose bacterial clusters) or adherence patterns of other DEC pathotypes (*2*).

Enteroaggregative *E. coli* (EAEC), another DEC pathotype, is identified by the characteristic aggregative pattern of adherence (AA) in HeLa/HEp-2 cells; bacteria attach in aggregates to cell surfaces as well as around cells (*1*,*3*). EAEC colonizes the intestinal mucosa, forming a thick biofilm that favors prolonged colonization and induces malnutrition (*1*–*3*). Actually, this pathotype is heterogeneous regarding the presence of putative virulence genes and has recently been subgrouped into typical and atypical EAEC, which carry and lack AggR (a global regulator of EAEC virulence), respectively (*1*,*3*).

We recently conducted a study at the Instituto de Puericultura e Pediatria Martagão Gesteria in Rio de Janeiro, Brazil, on the etiology of diarrhea affecting children of low socioeconomic status (V.B.C. Girão et al., unpub. data). In the study, all *E. coli* isolates were analyzed regarding their adherence patterns in HeLa cells and the presence of specific virulence genes of the DEC pathotypes, according to previously reported methods (*4*,*5*). Among 481 children (<2 years old) with diarrhea who were examined, 16 (3.3%) carried *E. coli* strains that co-expressed LA and AA (LA/AA), a phenotype not found among strains of 99 control children without diarrhea at the same hospital. The LA/AA phenotype was confirmed in individual colonies of each strain as well as in HEp-2 cells. In both cell lineages, prolonged assays (6 hours) showed that a mature biofilm that masked the LA phenotype had developed.

Although LA/AA co-expression in some human *E. coli* has been previously reported by Bouzari et al. (*6*), further information on these isolates is lacking. Moreover, since the expression of LA and AA is used to classify fecal *E. coli* as typical EPEC and EAEC (*1*,*3*), respectively, the classification of such strains within the DEC pathotypes is difficult. To determine their most appropriate classification, we further characterized the 16 LA/AA strains of our collection (Table). Colony hybridization assays used to search for additional *E. coli* virulence genes (*bfpA*, *perA*, E-*hly*, *daaC*, *cdt*, *cnf*, *hly*, *aggR*, *aggC*, *aafC*, *aap*, *shf*, *irp2*, *pet*, *pic*, *astA*, *pap*, *afa*, *sfa*, *efa*, *paa*, *saa*, *enfA*) (*1*,*3*–*5*,*7*) showed that all strains carried *eae*, *bfpA*, and *perA*, and 13 also carried the EAF sequence (a cryptic pEAF marker). Less commonly found genes were *paa*, *shf*, *irp*2, *astA*, and *efa*, and the remaining genes were absent. BFP expression was confirmed in all strains by immunoblot, and positivity in the fluorescent actin staining assay (*8*) demonstrated that they can produce attaching/effacing lesions. PCR analysis of 4 (α, β, γ, and δ) (*9*) of at least 10 recognized intimin subtypes (*1*) showed that subtype δ was the most frequent. Serotyping (*5*) identi-