P. jirovecii independent of environmental hazards.

Isolation of pathogens from an aborted fetus does not necessarily mean that they have caused the death of the fetus because many agents appear to pass through the fetal-placental unit and cause little damage. However, fungal infection is a major worldwide cause of abortion in cattle (*10*), and the surprising high prevalence of *P. jirovecii* infection found in dead fetuses in our study emphasizes the need to study the possible role of this fungal organism in human abortion.

Our findings could be of potential clinical importance and could open a new field of research, which should be explored. Further research should assess the scope of the problem and design rational preventive strategies, if necessary.

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Avian Influenza Virus (H5N1) in Human, Laos

To the Editor: The first avian influenza (H5N1) outbreak in poultry in Laos occurred in 2003 and subsided in March 2004 after massive killing of poultry to contain the disease. Extensive surveillance from July 2005 through January 2006 did not detect any influenza virus subtypes in chicken, ducks, quails, and pigs in live bird markets in the Vientiane, Champasak, and Savannakhet Provinces (*1*). Avian influenza virus (H5N1) was reintroduced into Laos in February 2006 but showed a lower incidence. Viruses isolated in this country in 2004 belonged to genotype Z, clade 1, and 2006 isolates belonged to clade 2.3.4 (online Appendix Figure, panel A, available from www.cdc.gov/EID/content/15/1/127-appF.htm) (*1*).

Avian influenza (H5N1) had not been reported in humans in Laos until February 27, 2007 (*2*). Our patient was a 15-year-old adolescent girl who lived in a suburb of Vientiane where an outbreak of influenza (H5N1) in poultry had been confirmed on February 7, 2007. Influenza-like symptoms developed in the patient on February 10. She was hospitalized in Vientiane with fever and respiratory symptoms on February 15. On February 17, her parents brought her to a private hospital in Nong Khai Province, Thailand. Oseltamivir was prescribed on February 19. On February 20, she was transferred to the Nong Khai Provincial Hospital because of rapid, progressive, severe pneumonia with acute respiratory distress syndrome. When we suspected avian influenza in this patient, clinical specimens were tested.

A diagnosis of infection with avian influenza (H5N1) was based on positive results obtained by reverse transcription–PCR (RT-PCR), viral isolation in MDCK cells inoculated with an endotracheal suction specimen

collected on February 22, and a 4-fold increase in neutralizing antibody titers from 80 to 320 in paired blood specimens collected on February 25 and March 1, as assayed against autologous virus. This virus isolate was named A/Laos/Nong Khai 1/07(H5N1). Subsequent samples were collected on February 25 and March 7 (day of death). Results of RT-PCR were positive for the sample collected on February 25 only; virus isolation results were negative for both samples.

The virus was screened for a novel reassorted gene by a multiplex RT-PCR and 8 primer pairs specific for each genomic segment of genotype Z, clade 1 virus (*3*). All segments except the polymerase A (PA) segment were amplified, which indicated that the new virus was different from genotype Z viruses. The viral genome was sequenced and submitted to GenBank (accession nos. EU499372–EU499379 for hemagglutinin, nonstructural protein, matrix protein, nucleoprotein, PB1, PB2, neuraminidase, and PA genes, respectively). Phylogenetic analysis showed that this virus belonged to genotype V (online Appendix Figure, panel B) (*4*); phylogenetic analysis of the hemagglutinin gene (www.who.int/csr/disease/avian_influenza/smaltree.pdf) showed that it belonged to clade 2.3.4 (online Appendix Figure, panel A).

Protein sequence at the hemagglutinin cleavage site harbored many basic amino acids (RERR_RKR). One amino acid deletion and 1 amino acid change were found when compared with RERRRKKR, which is present in most avian influenza viruses (H5N1). There was no change in receptor binding site. This virus had glutamic acid at aa 627 in the PB2 protein, aspartic acid at aa position 92 in nonstructural protein 1, and 5 aa deletions at positions 80–84 in the nonstructural protein 1. Analysis of the neuraminidase gene showed a 20-aa deletion in the stalk protein; there was no mutation of histidine to tyrosine at aa position 274, a position shown to be the oseltamivir resistance marker in the neuraminidase 1 viral genome (*5*). Mutations in the matrix 2 gene showed that amantadine resistance was not present in our virus (*6*). Our in vitro assay (*7*) showed that this virus was sensitive to oseltamivir and amantadine.

Since 2003, genotype V influenza viruses (H5N1) have been reported in some East Asian countries. Genetic diversity in the hemagglutinin gene has classified those genotype V viruses into distinct clades. Viruses from avian species in South Korea in 2003 and Japan in 2004 (*8*,*9*) belong to clade 2.5. A/chicken/Shanxi/2/2006 isolate belonged to clade 7. Human cases in People's Republic of China, i.e., A/China/GD01/06, A/Shenzhen/406H/06, A/Jiangsu/1/2007, and A/Jiangsu/2/2007, belong to clade 2.3.4, the same clade as A/chicken/Thailand/NP-172/2006 and the virus from our study.

Highly pathogenic avian influenza viruses (H5N1) that caused outbreaks in Thailand since 2004 belong to genotype Z, clade 1. Introduction of genotype V clade 2.3.4 virus, A/chicken/Thailand/NP-172/2006, to Nakhon Phanom Province occurred in November 2006 (*10*), the same year that clade 2.3.4 virus was introduced into Laos (online Appendix Figure, panel A). On the basis of hemagglutinin gene phylogeny, A/Laos/Nong Khai 1/2007 is closely related to A/chicken/Nongkhai/NIAH 400802/2007 and A/chicken/Thailand/NP-172/2006. Phylogenetic analysis suggested that viruses from these 2 countries shared the same origin. There was extensive movement across the Mekong River even before the bridge linking Nong Khai from Vientiane was opened. However, the route of transmission of genotype V viruses from east Asian to Southeast Asian countries could not be elucidated.

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Fatal HIV Encephalitis in HIV-Seronegative Patients

To the Editor: Acute encephalitis is rarely seen in patients infected with HIV (*1*). In addition, HIV in patients who are seronegative is extremely rare, particularly in the setting of current screening ELISAs (*2*). We report a case of encephalitis and HIV in the same patient, which resulted in death.

A 44-year-old Caucasian woman sought treatment at our hospital with a 1-week history of fever, unsteady gait, and progressive confusion. Her medical history included hypothyroidism, depression, and chronic alcohol abuse. The patient's first tests for HIV were negative at 19 and 12 months prior to admission during routine intake screening for jail inmates (Abbott HIV AB HIV-1/HIV-2 [rDNA] enzyme immunoassay [EIA] kit; Abbott Laboratories, Abbott Park, IL, USA). Six months before admission, the patient had a viral exanthem of blistering rash on her lips, palate, and chest. Two weeks later, she had oral thrush and a leukocyte count of 1,700 cells/μL. An HIV ELISA result was negative. Three months before admission, she was admitted to a different hospital for weakness, abdominal pain, intermittent fever, diarrhea, persistent oral candidiasis, and ethanol withdrawal. She had leukopenia and thrombocytopenia. A fourth HIV ELISA result was negative. The patient had been admitted to our hospital one week before the current admission with symptoms of fever, confusion, and urinary tract infection. Lumbar puncture showed an elevated protein level (106 mg/dL). A fifth HIV test result 6 days before most recent admission was negative. Five days before admission, she had been discharged to a rehabilitation facility.

On this hospitalization, she had fatigue, headache, disequilibrium, dysarthria, and blurred vision. Initial examination showed fever of 101.3°F, poor word recall, and a wide-based gait. Laboratory tests showed mild anemia and a leukocyte count of 2×10^3 cells/μL.

Over the next few days the patient's fever persisted and her mental status fluctuated. Tests on hospital day 2 showed a CD4 count of 101/mL (16.9%). Magnetic resonance imaging (MRI) of the brain showed diffuse symmetric white matter disease (Figure, panel A). Samples sent on hospital day 9 eventually showed wild-type HIV with a viral load >500,000 copies/mL. Repeat cerebrospinal fluid (CSF) test results were negative for cryptoccocus antigen, and PCR results were negative for cytomegalovirus, herpes simplex virus (HSV), and JC polyoma virus. The next day, a sixth HIV ELISA result was negative. The serum level of HIV p24 antigen was 202 pg/mL.

On hospital day 13, the patient began treatment with zidovidine, lamuvidine, didanosine, and nevirapine. Within 24 hours, seizures and catatonia developed in the patient. An electroencephalogram showed diffuse wave form slowing. A repeat MRI showed worsened white matter disease (Figure, panel B). The result of a seventh HIV screening ELISA performed on hospital day 15 was negative. Two days later, the HIV viral load was 241,789 copies/mL. On hospital day 19, her serum levels were within normal limits: immunoglobulin (Ig) M level (164 mg/dL), IgG level (1,440 mg/dL), a 3× normal IgA level (1,060 mg/dL), and no oligoproteins. The CSF had an IgG level >10× normal (72 mg/dL), elevated IgG levels for HSV1 (1:160) and HSV2 (1:40), was negative for virus culture, and showed a negative PCR result for JC polyoma virus. On hospital day 23, the eighth HIV ELISA result was negative. The Abbott HIVAB HIV-1/HIV-2 (rDNA) EIA was used throughout the hospitalization. On hospital day 24, supportive care was withdrawn and the patient