ing factor in the analysis. However, recently published work has indicated no statistically significant differences in the verotoxin proteins encoded by SF-O157 or non–SF-O157 strains or in their level of toxicity (*9*). Other virulence factors may contribute to increased likelihood of HUS (*4*).

Our data suggest that infection with SF-O157 results in less severe colitis than does the more common non–SF-O157 infection. Less severe colitis could result in a lower risk for renal disease because less verotoxin would be translocated into the bloodstream and bound to the kidneys. However, patients infected with SF-O157 had anuria for longer periods and consequently had longer sessions of peritoneal and hemodialysis. Although unknown bacterial or host inflammatory cytokines may contribute to enhanced disease progression, this observation is surprising and requires further investigation. Additional research is needed to learn more about the virulence of SF-O157 strains and establish other host factors that contribute to disease progression.

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Co-infection with Dengue Virus and Pandemic (H1N1) 2009 Virus

To the Editor: Dengue is a mosquito-borne viral infection caused by 4 related dengue viruses. Each of these viruses is capable of causing classic dengue fever or dengue hemorrhagic fever (DHF), but may also cause nonspecific febrile illnesses. As a result, dengue is often difficult to diagnose clinically, especially because peak dengue season often coincides with that of other common febrile illnesses in tropical regions (*1*). Concurrent outbreaks of influenza and dengue have been reported (*2*,*3*); this circumstance often leads to delayed recognition of the presence of one or other disease in the community.

In April 2009, a new strain of influenza A virus known as pandemic (H1N1) 2009 virus was first detected in the United States (*4*). Pandemic (H1N1) 2009 infections were first detected in Puerto Rico in June 2009, and 59 deaths caused by the virus have been confirmed to date. This influenza outbreak coincided with the typical dengue season in Puerto Rico, which led to diagnostic difficulties; both infections disproportionately affected young persons, who often had similar, nonspecific symptoms.

We describe a case of laboratory-confirmed co-infection of dengue virus and pandemic (H1N1) 2009, and discuss the difficulties in distinguishing the 2 illnesses clinically. A 33-year-old woman (healthcare worker) in Ponce, Puerto Rico, sought treatment at an emergency department of a hospital in the southern part of the island with a 3-day history of febrile illness. Her symptoms began with throat irritation and earache; subsequently, cough, fever, and headache developed. She reported palpitations and generalized malaise but no other symptoms. The patient had no notable medical his-

tory and denied taking any medicines apart from over-the-counter antipyretics. She reported recent exposure to influenza at work and multiple recent mosquito bites. On physical examination, she had a temperature of 37°C, a heart rate of 91 bpm, and blood pressure of 125/82 mm Hg. A tourniquet test result was positive. Her pharynx was erythematous without exudate, and she had rhinorrhea. She had no lymphadenopathy, rash, petechiae, or purpura. Several small, red papules, which the patient described as recent mosquito bites, were on her legs. The remainder of her examination showed no unusual findings.

Laboratory studies showed a leukocyte count of 5,300 cells/mm^3 with a normal differential count, hematocrit 35.2%, and thrombocyte count of 239,000 cells/dL. Results of a chest radiograph was unremarkable. A nasopharyngeal swab was positive for influenza A virus by rapid test. Nasopharyngeal and serum samples were sent to the Centers for Disease Control and Prevention (Dengue Branch) for influenza and dengue testing. The patient was diagnosed with suspected pandemic (H1N1) 2009 infection and prescribed oseltamivir for 5 days. She returned for a follow-up visit 12 days after the onset of symptoms. She reported having 2 more days of fever after her initial visit, but had no rash, petechiae, bleeding, or progression of respiratory symptoms. A second serum specimen was obtained during this visit.

The initial serum specimen was positive for dengue virus by serotype-specific, singleplex, real-time reverse transcription–PCR (*5*). Her nasopharyngeal specimen was positive by PCR for pandemic (H1N1) 2009 influenza. The second, convalescent-phase, serum specimen was negative for dengue immunoglobulin (Ig) M by IgM antibody–capture ELISA. The acute-phase and convalescent-phase samples were positive for IgG against dengue by ELISA (*6*), which indicated a secondary dengue infection. IgG titers exceeded the limits of the test for acute-phase and convalescent-phase samples, showing unusually elevated levels of IgG against dengue.

Distinguishing dengue and influenza by clinical features alone can be difficult. In an investigation of simultaneous dengue and influenza A outbreaks in Puerto Rico in 1977, similar percentages of persons with confirmed dengue and confirmed influenza had classic dengue symptoms (*2*). Hemorrhagic manifestations, like those typically seen in DHF, have been reported with influenza A in prior outbreaks (*7*,*8*) and with pandemic (H1N1) 2009 (Centers for Disease Control and Prevention, unpub. data). Previous influenza A outbreaks were initially believed to be outbreaks of DHF until careful laboratory investigation proved otherwise (*8*). Our patient did not have the typical signs and symptoms of dengue (rash, eye pain, thrombocytopenia, arthralgia, petechiae, or bleeding) that would differentiate her condition from that of patients with other febrile illnesses. She did have a positive tourniquet test result and fever, which have been advocated as screening criteria for dengue infection in children, at the time of initial examination (*9*). Data for the specificity and sensitivity of these criteria in adults are sparse, however, and some studies have shown a high incidence of positive tourniquet test results in patients with laboratory-confirmed influenza (*7*,*8*).

Our report demonstrates that co-infection with dengue virus and pandemic (H1N1) 2009 can occur. Previous studies also have shown cases of probable co-infection with seasonal influenza and dengue (*1*,*10*), including 1 fatal case (*1*). Because many dengue-endemic countries are experiencing pandemic (H1N1) 2009 outbreaks, providers should consider the possibility of viral co-infection, especially in severe cases, and should consider testing for both viruses.

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Bovine Tuberculosis in Buffaloes, Southern Africa

To the Editor: Emergence of bovine tuberculosis (TB) in wildlife in southern Africa has implications not only for the conservation of the wildlife species affected (*1*) but also for the health of humans and livestock living at the wildlife–livestock–human interface (*2*). Bovine TB in South Africa's Kruger National Park was first found in African buffaloes (*Syncerus caffer*) in 1990 (*3*) and likely entered the park by cattle-to-buffalo transmission (*4*). Bovine TB infection has been spreading northward; in 2003, infection was confirmed in a buffalo ≈60 km south of the Limpopo River. In 2005, a case was confirmed only 6 km south of the river (D. Keet, unpub. data). In 2008, we isolated *Mycobacterium bovis* from African buffaloes in Zimbabwe.

During October 9–13, 2008, a total of 38 buffaloes from 4 herds were captured in Gonarezhou National Park (south of the Mabalauta area; 22.0553°S, 31.4265°E). Blood samples were collected, sampled buffaloes were marked and released, and 3 adult females in each herd were equipped with radio collars. Buffalo tissue samples were collected, packaged, shipped, and handled at the Agricultural Research Council–Onderstepoort Veterinary Institute according to procedures recommended for controlling the spread of foot-and-mouth disease virus. Interferon-γ assay (*5*) results were positive for bovine TB for 4 (10.5%) buffaloes: 2 adult females and 1 young adult male from the same herd and 1 adult female from another herd.

Four months later, a radio-collared adult female and the young adult male, each of which had had positive interferon-γ assay results, were sedated and euthanized, and necropsies were performed in the field. Samples for histopathologic examination and culture were collected from lymph nodes of the head and thorax. No acid-fast organisms were detected, but the histologic findings were strongly suggestive of paucibacillary TB. *M. bovis* was isolated from the retropharyngeal lymph nodes of both buffaloes and from the bronchial and head lymph nodes of 1 of them. Both isolates were typed by analysis of variable number of tandem repeat (VNTR) sequences at 6 loci (exact tandem repeat A–F) (*6*) and compared with the VNTR profiles of ≈75 isolates from Kruger. All isolates showed an identical VNTR profile (7544*52.3), which suggests an epidemiologic link between the *M. bovis* infections in the 2 parks. However, the exact tandem repeat loci had lower discriminatory power among Kruger isolates than did IS*6110* restriction fragment length polymorphism typing (T. Hlokwe, unpub. data) (*4*). A typing regimen comprising different typing methods and markers will be useful for more accurately determining the genetic relationship between the isolates from the 2 parks, Gonarezhou and Kruger.

The confirmation of results for bovine TB–infected buffaloes in Zimbabwe (Gonarezhou National Park) raises several questions regarding the transboundary spread of animal disease and has considerable management implications for the Great Limpopo Transfrontier Conservation Area. The most likely scenario is buffalo-to-buffalo contact across the boundary because the bovine TB cases reported here were located <45 km from the unfenced northern boundary of Kruger National Park. Buffaloes, especially bulls and young heifers, frequently move from herd to herd and may contribute to the spread of *M. bovis* by mixing with unexposed herds (*7*). Although transboundary movements of buffaloes between Kruger and Gonarezhou have not been specifically documented, uncontrolled movements across the Limpopo River do occur (de Garine-Wichatitsky, unpub. data). However, >12 wild species in Kruger have now been found to be infected by bovine TB (*2*). Most of these species are probably not effective sources of *M. bovis* infection for buffaloes, but the disease epidemiology could rely on multihost reservoirs (*8*). Thus, a second scenario could be a buffalo-to–unidentified wild species–to-buffalo pathway, because species like greater kudu (*Tragelaphus strepsiceros*) appear to be able to maintain, spread, and even drive a bovine TB epidemic (*4*,*9*). A third scenario involves movement of infected livestock across the boundaries of the 3 countries of the Great Limpopo Transfrontier Conservation Area, resulting in cattle-to-buffalo transmission of bovine TB. As a last scenario, we cannot rule out the possibility that bovine TB infection of buffaloes has remained silent and undetected for decades in Zimbabwe.

The management implications of bovine TB in buffaloes in Gonarezhou National Park are considerable. Once bovine TB is established in a native free-ranging maintenance host, eradication is unlikely (*2*,*10*). Evaluation