terobacteriaceae (such as some strains of *Klebsiella* spp.) and other organisms (such as *Staphylococcus aureus*), can have similar requirements (*7*,*8*).

There is not 1 best way of performing urine cultures. Guidelines for the diagnosis of UTI includes the use of sheep blood agar and either MacConkey agar or a similar selective medium for routine urine culture. The plates should be incubated overnight (at least 16 hours) at 37°C in ambient air; alternatively, the blood agar plate can be incubated in elevated (3%–8%) CO_2 (*9*). For fastidious microorganisms, chocolate agar can be added to the MacConkey agar and the plates incubated in 5% CO_2 for 2 days (*9*).

The real incidence of these infections is unknown, but the rarity of these strains suggests that the incidence is low. However, the real incidence of UTI caused by capnophilic *E. coli* may be underestimated because urine cultures are not usually incubated in CO_2. In addition, urine cultures are not performed for many women with uncomplicated cystitis. Other fastidious uropathogens such as *Haemophilus influenzae* and *H. parainfluenzae*, also require special media and incubation in an atmosphere of CO_2 (*9*). The low frequency of these strains suggests that incubation of routine urine cultures in an atmosphere containing CO_2 is not necessary. Incubation in CO_2 should be ordered only if the patient has pyuria and a previous negative urine culture after incubation in ambient air or if the patient is unresponsive to empiric therapy and routine urine culture is negative. Good clinician–laboratory communication is vital. Further studies should be performed to ascertain the real incidence of UTIs caused by capnophilic strains of *E. coli.*

Because no breakpoints are available for antimicrobial agents against capnophilic strains of *E. coli*, we used published interpretative criteria or *Enterobacteriaceae* (*3*). The strain was susceptible to all antimicrobial agents that we tested. The impact of CO_2 on the susceptibility of capnophilic strains of *E. coli* is unknown. Susceptibility of some antimicrobial agents such as quinolones can be influenced by the pH change and enhanced growth that occur during CO_2 incubation when testing capnophilic organisms (*10*).

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Duck Migration and Past Influenza A (H5N1) Outbreak Areas

To the Editor: In 2005 and 2006, the highly pathogenic avian influenza (HPAI) virus subtype H5N1 rapidly spread from Asia through Europe, the Middle East, and Africa. Waterbirds are considered the natural reservoir of low pathogenic avian influenza viruses (*1*), but their potential role in the spread of HPAI (H5N1), along with legal and illegal poultry and wildlife trade (*2*), is yet to be clarified.

The garganey (*Anas querquedula*) is the most numerous duck migrating between Eurasia and Africa: ≈2 million gather in the wetlands of Western Africa every northern winter (*3*). We report on a spatial correlation between the 2007 migration path of a garganey monitored through satellite telemetry and areas that had major HPAI (H5N1) outbreaks from 2005 through 2007.

Seven garganeys were captured, sampled, and fitted with a 12-g satellite transmitter in northern Nigeria (Hadejia-Nguru Wetlands; 12°48′N; 10°44′E) in the period February 7–15, 2007. All cloacal and tracheal swabs tested negative for avian influenza virus by real-time reverse transcription–PCR analysis of the matrix gene. One second-year (>9-month-

old) female garganey migrated from northern Nigeria to Russia in April–May 2007 (online Appendix Figure, available from www.cdc.gov/EID/content/14/7/1164–appG.htm), where she remained until the end of July. During this 6-week spring migration over the Sahara Desert, Mediterranean Sea, and Eastern Europe, this duck stopped at 3 main stopover sites in Crete, Turkey (Bosphorus region), and Romania (Danube River delta). The duck migrated back to the Danube delta in August, where it remained until November, when the signal was lost. Other garganeys we monitored stopped transmitting before initiating spring migration (n = 3) or remained in West Africa during spring and summer (n = 3), which suggests a stress linked to capture or constraint from the transmitter attachment.

This transcontinental migration path connects several areas of past major HPAI (H5N1) outbreaks (online Appendix Figure). The wintering area in Nigeria where this duck was caught and remained for 8 weeks before spring migration is located where a large number of outbreaks have occurred repeatedly since February 2006 (the closest being 30 km away). This bird reached its breeding ground in Russia near Moscow and stayed for 2 months in an area that had several outbreaks in backyard poultry in February 2007 (the closest being 30 km away). Finally, the Danube delta, used as a resting ground for 3 months in late summer and autumn, is also an area with recurring outbreaks since October 2005 in wild and domestic birds, with the most recent case reported in November 2007 (the closest being 10 km away). The initial spread of HPAI virus (H5N1) from Eurasia to Africa occurred in autumn and winter 2005–06. The migratory movements we observed during spring and summer in this study were not temporally correlated with any reported HPAI (H5N1) outbreak, either in sequence or period; hence, they should not be interpreted as evidence of the role of wild bird in expansions of the virus.

During spring migration from Nigeria to Russia, the garganey stopped several days in wetlands situated close to areas of past outbreaks in the Danube delta (4 days at a distance of 1–4 km from October 2005 outbreaks) and Lake Kus, Turkey (8 days at a distance of 10–30 km from October 2005 outbreaks). The occurrence of past outbreaks indicates that the duck used wetlands favorable to HPAI virus (H5N1) transmission as stopover sites. The relatively long stopover periods enabled prolonged contact of migratory ducks with local domestic and wild bird populations or through shared water, thus prolonging the potential for virus transmission. Considering the persistence of infectivity of HPAI virus (H5N1) in aquatic habitats (*4*), the number of migratory ducks congregating at stopover sites from various geographic origins and destinations, and the asynchronous timing of the arrival and departure of migratory ducks (*5*), we believe that these sites may provide locations for disease transmission and possible spread upon movement of wild birds.

The satellite-fitted female garganey covered distances between stopover sites of >2,000 km in <2 days, traveling at an estimated speed of 60 km/h. This large-scale movement in a short period, coupled with experimental exposure trials demonstrating viral shedding of up to 4 days in ducks with no clinical signs of infection (*6*), is consistent with potential viral transmission over great distances.

These facts illustrate how a pathogen such as HPAI virus (H5N1) can potentially be transported rapidly by migratory birds across continents. However, the physiologic impact of an HPAI (H5N1) infection on the ability of birds to migrate long distances is still unknown (*7*) and to date, most empirical evidence suggests that wild birds have only moved short distances (a few hundred kilometers) likely carrying HPAI virus (H5N1) (*8*). Despite extensive global wildlife surveillance efforts and with the exception of a few reported cases of HPAI (H5N1) infection in apparently healthy wild ducks (*9*,*10*), evidence of wild bird involvement in the spread of HPAI virus (H5N1) over long distances is still lacking.

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Dihydrofolate Reductase I164L Mutation in *Plasmodium falciparum*, Madagascar

To the Editor: Malaria remains a major public health problem and a primary cause of illness in Madagascar (*1*). Since 2005, the National Malaria Control Program has revised its treatment policy and replaced chloroquine (CQ) with artesunate plus amodiaquine as first-line therapy for uncomplicated malaria and CQ with sulfadoxine-pyrimethamine (SP) for prevention of malaria during pregnancy. The latter choice was partially supported by high effectiveness of SP and absence of pyrimethamine resistance in Madagascar, in contrast to proximal African countries such as the Comoros Islands (*2*,*3*).

Analysis of the molecular basis of antimalarial drug resistance has demonstrated that mutations in the dihydrofolate reductase (*dhfr*) and dihydropteroate synthase genes are associated with development of SP resistance. It has been assumed that pyrimethamine resistance conferred by multiple mutations arose through stepwise selection of the S108N single mutant (except for the A16V/S108T allele). This single-point mutation decreases the sensitivity of *dhfr* to pyrimethamine in vitro by ≈10× (*4*). Subsequent mutations, such as N51I and C59R, cause additional decreases in the sensitivity of *dhfr* to pyrimethamine. Parasites with a triple-mutant allele (51I/59R/108N) are less sensitive to pyrimethamine in vitro, and patients infected with these parasites have a high probability of not responding to SP treatment (*5*).

Addition of I164L to 51I/59R/108N creates a quadruple-mutant allele and decreases the sensitivity of *dhfr* by ≈1,000× (*4*), eliminating the clinical effectiveness of SP, as observed in Southeast Asia and South America. However, the situation in Africa seems to be different because most studies conducted since the mid 1990s have shown the quadruple mutant to be rare, even in areas of intensive pyrimethamine use (*6*). Increasing SP resistance is principally a result of rapid selection for parasites that carry a triple-mutant allele that arose in Southeast Asia and has spread widely in Africa (*7*,*8*).

In 2006, blood samples were obtained from 114 children 6 months to 15 years of age enrolled in a clinical trial monitoring the efficacy of SP in treatment of uncomplicated *Plasmodium falciparum* malaria. The *dhfr* gene from pretreatment samples was sequenced at the Genomics Platform of the Pasteur Institute in Paris, France. Four (3%) samples contained the 108N single-mutant allele, 37 (32%) contained the 51I/59R/108N triple-mutant allele, and 1 (<1%) contained the I164L single-mutant allele. This latter allele was obtained from the blood of a 15-year-old girl from Ejeda in southern Madagascar. At enrollment in the trial, she had an axillary temperature of 37.8°C and a *P. falciparum* asexual parasite count of 74,880/μL. She was treated with the standard SP regimen (25 mg/kg sulfadoxine and 1.25 mg/kg pyrimethamine as a single dose on day 0). On the basis of the World Health Organization 2003 protocol (*9*), early treatment failure was noted on day 2, when the patient had signs of malaria with a temperature of 40°C and a parasite count of 770/μL. She was successfully retreated with a rescue regimen (quinine, 8 mg base/kg, 3 times a day for 7 days).

To confirm detection of the I164L allele, parasite DNA was extracted from blood spots obtained on days 0, 1, and 2 and sequenced. DNA templates were sent to a second independent laboratory (Department of Genome Sciences, University of Washington, Seattle, WA, USA) to rule out misiden-