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Avian *Mycoplasma lipofaciens* Transmission to Veterinarian

To the Editor: *Mycoplasma* spp. are well-known pathogens in human and veterinary medicine. Mammals, especially primates and including humans, share similar or even identical *Mycoplasma* spp., which might be commensal or pathogenic (*1*). Additionally, sporadic infections of immunocompromised persons with *Mycoplasma* spp. that originated from domestic animals have been reported (*1*); susceptibility in this human population is increased (*2*,*3*). *M. phocicerebrale* is the only *Mycoplasma* pathogen of animals that regularly infects humans, causing a disease called seal fingers (*1*,*4*). However, we report a human infection with an avian *Mycoplasma* organism.

A clinical trial to investigate the capability of *M. lipofaciens* (strain ML64) (*5*) to spread horizontally between infected and noninfected turkey poults in an incubator demonstrated airborne transmission of the pathogen within 24 hours (*6*). During the trial, the veterinarian conducting the study, a 36-year-old man, was monitored for infection. Each day, 2 swabs were taken from both nostrils, starting

from the day before the infected poults hatched (day 0) through day 7 after the poult hatching date. When handling eggs and poults, the veterinarian wore gloves but not a protective mask. Two days after the poults hatched (day 3), the veterinarian reported throat pain and a slight rhinitis, which indicated a respiratory disease. The next day only the rhinitis with minor nasal pain was present.

One nasal swab from each sampling day was used for *Mycoplasma* spp. culture (*5*). Isolated *Mycoplasma* organisms were subjected to an immunobinding assay (*6*) with antiserum against *M. lipofaciens*, *M. buteonis*, *M. falconis*, *M. gypis*, *M. gallisepticum*, *M. meleagridis*, *M. synoviae*, and *M. iowae*, selected because the veterinarian regularly handled these isolates. The second nasal swab from each sampling day was taken to detect *Mycoplasma* DNA by PCR (*5*). The samples for PCR testing were stored at –20°C and tested only when attempts to isolate *Mycoplasma* spp. failed.

Six weeks after the infection, a serum sample from the patient was examined for specific antibodies against *M. lipofaciens* by 3 different methods: modified immunobinding assay (*6*) (which used patient's serum and peroxidase-conjugated goat-antihuman serum [STAR90P, Serotec Ltd, Oxford, UK]), a growth inhibition test, and antibody titers of serum dilutions. The first 2 methods used *M. lipofaciens* (strain ML64) from a turkey trial (*6*) and the reference strains of *M. buteonis* (Bb/T2g) and *M. falconis* (H/T1). For the third method, to determine the titer, 2-fold serial dilutions of the patient's serum samples were prepared and incubated with a bacterial suspension (*M. lipofaciens* strain ML64) containing 3.2×10^2 CFU. The antibody titer was based on the highest serum dilution capable of reducing 50% of the mean CFU count.

Before the infected poults hatched, attempts to isolate *Mycoplasma* organisms and demonstrate *Mycoplasma* DNA from the nasal swabs were unsuccessful. However, from the day of hatching (day 1) until 3 days later (day 4), *Mycoplasma* organisms were isolated from the nasal swabs and identified as *M. lipofaciens*. On day 5, only *Mycoplasma* DNA was demonstrated (Table). Specific antibodies against *M. lipofaciens* (strain ML64) were detected by using the immunobinding assay and growth inhibition test. Antibodies against other *Mycoplasma* spp. were not detected. Antibody titer against *M. lipofaciens* was 128.

M. lipofaciens have been reported from a chicken, turkey, duck (*7*,*8*), and a raptor egg (*5*). Strain ML64 is highly pathogenic for chicken and turkey embryos and can be transmitted by air (*6*,*9*). The veterinarian handling the infected poults was free of nasal *Mycoplasma* organisms a day before contact. His infection occurred concurrent with demonstration of airborne transmission among poults. The isolation of *M. lipofaciens* from his nares for 4 days demonstrates the infectivity and reproductive capability of this *Mycoplasma* strain in humans: as a pure contaminant, isolation for several days would be unlikely. Christensen et al. (*10*) have reisolated different avian *Mycoplasma* strains (*M. gallisepticum*, *M. synoviae*, *M. iowae*) from a human nose, from 12 hours through 1 day after artificial infection, demonstrating differences between avian strain abilities to survive on human mucosa. *M. lipofaciens* invasiveness for humans is underscored by finding specific antibodies against this species 6 weeks after infection. Cross-reactivity to other *Mycoplasma* spp. cannot be excluded but seems unlikely.

This study suggests that *M. lipofaciens* (strain ML64) can be transmitted successfully to humans and may cause clinical symptoms; the study documents nonartificial human infection with an avian *Mycoplasma* sp. These findings should be considered especially for humans highly susceptible to *Mycoplasma* infections, including children and persons with congenital or acquired immunodeficiencies (*2*,*3*).

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Table. Timeline of natural infection of veterinarian with *Mycoplasma lipofaciens* (strain ML64) from infected poults*

	Day							
Finding	0	1†	2	3	4	5	6	7
Isolation	–	*M. l.*	*M. l.*	*M. l.*	*M. l.*	–	–	–
PCR result‡	–	ND	ND	ND	ND	+	–	–
Clinical signs	None	None	None	Throat pain, slight rhinitis	Slight rhinitis, nasal pain	None	None	None

*–, negative; *M.l., Mycoplasma lipofaciens* (identified by immunobinding assay); ND, not done; +, positive.
†Hatching of infected poults and demonstration of aerosol transmission among poults.
‡Detection of mycoplasma DNA per Lierz et al. (*5*).

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Urinary Tract Infection Caused by Capnophilic *Escherichia coli*

To the Editor: Increased atmospheric CO_2 concentrations promote the growth of fastidious microorganisms. However, the possibility that a strain of *Escherichia coli* can be CO_2 dependent is exceptional (*1*).

An isolate of capnophilic *E. coli* was responsible for a urinary tract infection (UTI) in a 77-year-old woman at the University Hospital of Guadalajara (Spain) in November 2002. Urine was cultured on a cystine-lactose-electrolyte-deficient agar plate and incubated at 37°C in an atmosphere containing 6% CO_2 for 1 day. After 24 hours, the culture yielded gram-negative rods (>10^5 CFU/mL) in pure culture. The organism was motile, catalase positive, and oxidase negative. The strain could not be identified by using the MicroScan WalkAway-40 system (DadeBerhing, Inc., West Sacramento, CA, USA). A subculture was performed, and the organism did not grow on sheep blood agar and MacConkey agar plates at 37°C in ambient air. However, a subculture incubated at 37°C for 24 hours in an atmosphere of 6% CO_2 produced smooth colonies 2–3 mm in diameter on sheep blood agar and MacConkey agar plates. The organism fermented lactose, and the indole reaction (BBL DrySlidet, Becton Dickinson Co., Sparks, MD, USA) performed on sheep blood agar was negative. The strain grew well on Schaedler agar plates after anaerobic incubation for 48 hours. The isolate remained capnophilic after 5 subcultures. The strain was identified as *E. coli* by using the Biolog GN2 panel (Biolog, Inc., Hayward, CA, USA) (100%, T = 0,534), after incubation of the panel in an atmosphere containing 6% CO_2 for 1 day. The API 20E system (bioMérieux, Marcy-l'Etoile, France) according to the manufacturer's instructions without $C0_2$ incubation also identified *E. coli* (profile 5004512). The identification was confirmed by means of 16S rDNA sequence analysis (1,472 bp obtained by PCR amplification by a previously reported method [*2*]), which showed 99% similarity with *E. coli* sequence (GenBank accession no. CP000802). The 16S sequence showed similarity with *Shigella* species; however, this identification was not considered because the strain fermented lactose on MacConkey agar and agglutinations with *Shigella* antiserum were negative. The original 16S rDNA sequence was deposited in GenBank (accession no. EU555536).

The antimicrobial drug susceptibility profile was determined by incubating Mueller-Hinton agar plates at 37°C in an atmosphere containing 6% CO_2 by the disk diffusion method, according to National Committee for Clinical Laboratory Standards recommendations (*3*). The isolate was susceptible to ampicillin, amoxicillin/clavulanic acid, piperacillin, cefazolin, cefuroxime, cefotaxime, nitrofurantoin, fosfomycin, trimethoprim-sulfamethoxazole, gentamicin, tobramycin, amikacin, norfloxacin, and ciprofloxacin. MICs were obtained for the following antimicrobial agents with the E-test method (AB Biodisk, Solna, Sweden), performed on Mueller-Hinton agar plates incubated in a 6% CO_2 atmosphere: ampicillin (1.5 μg/mL), amoxicillin (3 μg/mL), cefotaxime (0.064 μg/mL), imipenem (0.094 μg/mL), piperacillin (2 μg/mL), and ciprofloxacin (0.008 μg/mL).

E. coli is the most common pathogen among patients with uncomplicated UTIs (*4*). Two cases of UTIs due to carbon dioxide–dependent strains of *E. coli* have been reported (*1*). The mechanisms for development of CO_2 dependence are unknown (*5*). CO_2 can play a role in the growth of *E. coli* as a substrate for carboxylation reactions (*6*). Other members of the family *En-*