occurred around the world. Firm evidence indicates that the fifth and sixth cholera pandemics were caused by the classical biotype whereas the most extensive and ongoing seventh pandemic is caused by the El Tor biotype. Since the onset of El Tor dominance in 1961, the classical strains have been gradually replaced by the El Tor strains and are now believed to be extinct. However, reports from Bangladesh (*6*), Mozambique (*7*), and this study have provided sufficient evidence to indicate that the classical cholera toxin gene has reappeared but that for these cases its carrier has been El Tor. Although how the classical cholera toxin in El Tor strains would affect *V. cholerae* pathogenicity is unclear, cholera caused by the classical biotype is more severe, whereas the El Tor biotype is considered to be better able to survive in the environment (*1*,*9*). Given that cholera toxin is directly responsible for the major clinical sign of the disease, such a genetic change could result in substantial alteration in the clinical manifestation of cholera. Additionally, this subtle genetic change may also influence the effectiveness of current cholera vaccines, which could stimulate both antitoxic and antibacterial immunity.

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Mycobacterium avium subsp. *hominissuis* Infection in 2 Pet Dogs, Germany

To the Editor: The genus *Mycobacterium* contains various obligate and opportunistic pathogens of animals, which may also be transmitted to humans and cause disease in, thus exhibiting a considerable zoonotic potential (*1*,*2*). During the past few decades, members of the *Mycobacterium avium-intracellulare* complex (MAIC) emerged as pathogens of human diseases, including lymphadenitis in children, pulmonary tuberculosis-like disease, and disseminated infections (occurring predominantly in immunocompromised persons, particularly AIDS patients) (*1*,*2*). Similarly, important animal diseases are caused by members of this group, e.g., avian tuberculosis and paratuberculosis in ruminants (*1*). MAIC includes *M. intracellulare* and 4 subspecies of *M. avium*, namely, *M. avium* subsp. *avium*, *M. avium* subsp. *hominissuis*, *M. avium* subsp. *silvaticum*, and *M. avium* subsp. *paratuberculosis* (*3*,*4*). Whereas members of the *M. tuberculosis* complex are transmitted by direct host contact, MAIC species are acquired predominantly from environmental sources, including soil, water, dust, and feed. Subclinical infections are common among birds (*1*,*2*).

M. avium strains differ from *M. intracellulare* by containing the insertion sequence (IS) IS*1245* (*3*) and are further discriminated by terms of IS*901* (*4*). Avian isolates (*M. avium* subsp. *avium*) are usually positive for IS*901* and represent the main pathogen of avian tuberculosis (*5*). In contrast, mammalian isolates are IS*901*-negative and have been designated as *M. avium* subsp. *hominissuis* because of their predominant hosts. This subspecies is only weakly virulent for birds but causes disease in animals and humans (*5*).

Even though *M. tuberculosis* and *M. bovis* are the common etiologic agents of canine mycobacteriosis, dogs are reported to be relatively resistant to *M. avium* infection (*6*,*7*). Nonetheless, sporadic cases usually show nonspecific clinical signs, whereas necropsy consistently reveals granulomatous inflammation in numerous organs, including lymph nodes, intestine, spleen, liver, lung, bone marrow, and even spinal cord (*7*,*8*). The predominant involvement of the gastrointestinal tract indicates an oral route of infection (*7*,*8*), and simultaneously increases the risk for human infection by fecal spread of mycobacteria.

Our report concerns 2 young dogs, a 3-year-old miniature schnauzer and a 1-year-old Yorkshire terrier, that lived in different geographic regions in Germany. Both had had therapy-resistant fever, lethargy, progressive weight loss, and generalized lymphadenomegaly for several weeks and were euthanized after a final phase of diarrhea. Necropsy findings, similar in both dogs, included generalized enlargement of lymph nodes with a whitish, granular to greasy cut surface, leading to intraabdominal adhesions by extensive involvement of mesenteric lymph nodes. In the terrier, the greater omentum and a part of the right apical lung lobe showed changes similar to those in the lymph nodes. Furthermore, numerous white 1-mm nodules were found in the spleen (both dogs), liver (schnauzer) and costal pleura (terrier).

Histologic examination showed (pyo-)granulomatous inflammation of lymph nodes, tonsils, liver, spleen, and greater omentum. Additionally, pyogranulomatous pleuropneumonia was present in the terrier, and a granulomatous enteritis and pyelitis in the schnauzer. The granulomatous lesions frequently exhibited central necrosis surrounded by macrophages, epithelioid cells, and few neutrophils (Figure, panel A). However, multinucleated giant cells or mineralization was not observed. In both animals, Ziehl-Neelsen stain demonstrated large numbers of acid-fast bacilli within macrophages (Figure, panel B). Samples of lymph nodes and lung were processed for mycobacterial culture by using standard procedures (Löwenstein-Jensen, Stonebrink medium). Colonies emerging after 2-week incubation at 37°C were investigated by PCR targeting IS*1245* and IS*901* (*3*,*4*). In all samples, *M. avium* subsp. *hominissuis* was identified by growth characteristics as well as presence of an IS*1245*-specific and absence of an IS*901*-specific PCR product. Additionally, sequencing of *hsp65* was conducted (*9*), which indicated *M. avium* subsp. *hominissuis* in both dogs (GenBank accession nos. EU488724 and EU488725).

Despite improved therapeutic approaches, MAIC infection represents a frequent bacterial complication in persons with AIDS. However, several studies showed a very low incidence of *M. avium* subsp. *avium* infections in humans. Thus, most of these HIV-related infections are attributed to *M. avium* subsp. *hominissuis* (*2*,*5*). Unfortunately, the subspecies of *M. avium* was not identified in most canine cases reported in the literature (*7*,*8*). Nonetheless, different serotypes of *M. avium*, corresponding to either *M. avium* subsp. *avium* or *M. avium* subsp. *hominissuis*, have been identified sporadically (*6*,*10*). The source and route of infection were unclear in all reports including ours, albeit repeatedly observed enteritis strongly suggested an oral mode of infection. A common environmental or wildlife reservoir represents the most probable source of *M. avium* infection for both humans and animals. However, there is also evidence of direct transmission (*1*–*3*). Therefore, *M. avium* subsp. *hominissuis* infection in dogs may comprise a considerable zoonotic potential, particularly if pet dogs with close contact to the owner are affected and if prolonged nonspecific clinical signs and intestinal involvement occur, as demonstrated here.

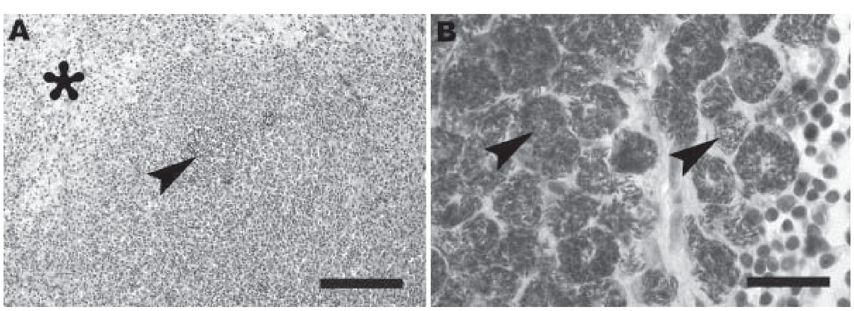

Figure. A) Mesenteric lymph node of Yorkshire Terrier shows diffuse granulomatous lymphadenitis with extensive infiltration of macrophages, foci of pyogranulomatous infl ammation (arrowhead), and focal necrosis (asterisk). Hematoxylin and eosin stain; scale bar represents 100 µm. B) Retropharyngeal lymph node of schnauzer shows innumerable acid-fast bacilli (arrows) within the cytoplasm of macrophages. Ziehl-Neelsen stain; scale bar represents 25 µm.

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Serogroup Y Meningococcal Disease, Colombia

To the Editor: *Neisseria meningitidis* is the etiologic agent of outbreaks, epidemics, and sporadic cases of meningitis or meningococcemia. Such infections have high illness and death rates, especially in children <5 years of age and adolescents. *N. meningitidis* serogroups A, B, C, Y, and W135 cause most meningococcal disease worldwide (*1*).

In Colombia, public health notification is required for all cases of invasive meningococcal disease. This reporting system is supported by a laboratory-based surveillance network for acute bacterial meningitis that has been coordinated by the Microbiology Group at the Instituto Nacional de Salud since 1994 (*2*,*3*). Clinical laboratories in Colombia submit isolates with associated information including geographic origin, specimen source, age, sex, and clinical diagnosis of the patient. Identification is confirmed by traditional phenotypic methods (*4*). Isolates are serogrouped by agglutination using commercial antisera (Difco, Detroit, MI, USA, and Becton Dickinson, Franklin Lakes, NJ, USA) and subtyped by dot blot with monoclonal antibodies (RIVM, Bilthoven, the Netherlands; and Institute Adolfo Lutz [IAL], São Paulo, Brazil) (*5*). Antimicrobial drug susceptibility testing for penicillin and rifampin is performed by the agar dilution, according to Clinical and Laboratory Standards Institute methods (*6*); for the breakpoints, we used those recommended by the Mesa Española de Normalización de la Sensibilidad y Resistencia a los Antimicrobianos (MENSURA) group (*7*). The reference laboratory participates in an external quality assurance program coordinated by the Pan American Health Organization (Sistema Regional de Vacunas [SIREVA] II, PAHO, Washington, DC, USA) with the Carlos III Institute, Madrid, Spain, and the IAL.

From 1994 through 2006, 434 *N. meningitidis* isolates were received by the Microbiology Group, from 22 of 35 departments (political divisions) and the Capital District: 119 (27.4%) from Antioquia, 117 (27.0%) from Bogotá, DC, 72 (16.6%) from Valle, 25 (5.8%) from Risaralda, 21 (4.8%) from Caldas, and 80 (18.4%) from 18 other departments. Distribution by department is published at the Institute's website (www.ins.gov.co) (*8*). According to public health reports, the reference laboratory is receiving ≈27% of the clinical case isolates. A slight majority (53.8%) were cultured from male patients. The age of patients was available for 396 isolates: 254 (64.1%) were <1–9 years of age, 71 (17.9%) 10–19 years, 41 (10.4%) 20–39 years, 21 (5.3%) 40–59 years, and 9 (2.3%) >59 years. Three hundred ninety-two isolates (90.3%) were recovered from cerebrospinal fluid and 42 (9.7%) from blood cultures. The diagnosis for 420 (96.8%) patients was meningitis; 11 (2.5%) patients had sepsis or bacteremia, and 3 (0.7%) had other invasive diseases (pneumonia, encephalopathy, or cellulitis).

Serogroup distribution was 338 (77.9%) group B, 42 (9.7%) group C, 40 (9.2%) group Y, and 2 (0.5%) group W135; 12 isolates were nongroupable. There was little annual variation for groups B and C, but there was an unexpected increase in serogroup Y (Figure), from 0% in 1994 to 50% in 2006. When the period 1994–2002 was compared with 2003–2006, this change was significant, increasing from 2.2% to 29.5% ($p<0.001$).

Antimicrobial drug–susceptibility testing showed that 17% of the isolates had intermediate resistance to penicillin (MIC 0.125–1.0 μg/mL) and 0.5% high resistance (≥2.0 μg/mL); only 1 isolate was resistant to rifampin (≥4.0 μg/mL). Penicillin resistance was not associated with any specific serogroup.