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Serologic Screening for *Neospora caninum*, France

To the Editor: In the June 2008 issue of Emerging Infectious Diseases, McCann et al. (*1*) reported on serologic screening for *Neospora caninum* antibodies and reported a lack of serologic evidence for *Neospora* infection in humans in England, where prevalence of infection with the closely related parasite *Toxoplasma gondii* also is low. Only limited data are available on human exposure to *Neospora*. We investigated the seroprevalence of *N. caninum* in humans in France, where *Toxoplasma* spp. seroprevalence is high.

Our study comprised 500 serum samples from healthy women, followed at the Cochin–Port Royal University Hospital in 1997 within the framework of toxoplasmosis surveillance during pregnancy, and 400 serum samples from HIV-infected patients. All serum samples were submitted to anti-*Toxoplasma* antibody testing by using indirect immunofluorescence (IIF; Toxo-spot IFI; bioMérieux, Marcy l'Etoile, France) and ELISA (Platelia Toxo IgG and IgM; BioRad, Hercules, CA, USA). An in-house microplate IIF test previously validated in cattle was used for simultaneous detection of anti-*Neospora* and anti-*Toxoplasma* immunoglobulin (Ig) G on the same microplate. All samples were screened at dilutions of 1:20 and 1:80, as is usually done in anti-*Toxoplasma* IIF assays in humans. Correlation between the anti-*Toxoplasma* IIF commercial test and in-house IIF was excellent (kappa coefficient = 0.98) and allowed us to compare the antibody titers against both parasites. Forty (8%) samples from immunocompetent persons and 21 (4%) from immunocompromised persons yielded a weak fluorescence when diluted 1:20. All but 4 had significant titers of anti-*Toxoplasma* IgG (>200 IU/mL in 77% of cases), which suggests low-level cross-reactions. Whereas titers of >200 and >320 are considered sufficient to diagnose neosporosis in dogs and cattle, respectively (*2*), positivity threshold was difficult to resolve in the absence of a positive human control. We decided on a positivity threshold of 1:80, which is similar to the threshold defined by others in further studies using an indirect fluorescence antibody test in humans (*3*,*4*). None of the 500 samples from immunocompetent persons were positive for *Neospora* antibodies when assessed at a dilution of 1:80. Within the group of immunocompromised persons, 3 were positive for *Neospora* antibodies at a titer of 80, and 1 was positive at a titer of 160. Three of these 4 HIV-infected patients had high titers of anti-*Toxoplasma* IgG (>2,000 IU/mL), suggesting *Toxoplasma* serologic reactivation. We found no evidence of *Neospora* infection or exposure in immunocompetent persons but could not exclude possible *Neospora* infection associated with *Toxoplasma* infection or reactivation in immunocompromised persons.

Taken together, our data agree with data from other studies conducted in European countries (*1*,*5*), which suggest that neosporosis in healthy humans is unlikely. However, the *Neospora* spp. seropositivity of some HIV-infected patients, although weak compared with the level of seropositivity in cattle or dogs, could suggest circulation of the parasite within immunocompromised hosts, a hypothesis supported by Lobato et al. (*3*). However, our observation of a strong serologic reactivation against *T. gondii* in 3 of 4 patients with anti-*Neospora* titers >80 mostly favors cross-reactivity involving homologous antigens of both parasites and nonspecific antibody binding from polyclonal stimulation of the immune system. Finally, one should keep in mind that the positive predictive value of a serologic test used in screening in low-prevalence populations is low. Large-scale studies are needed to more precisely determine the potential role of this parasite in immunodeficient humans and to isolate the parasite or detect *Neospora* DNA in such patients.

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Escherichia coli and *Klebsiella pneumoniae* Carbapenemase in Long-term Care Facility, Illinois, USA

To the Editor: *Escherichia coli* harboring *Klebsiella pneumoniae* carbapenemases (KPCs) are now rarely being reported. Worldwide, KPC-2 has been detected in Israel and the People's Republic of China (*1*,*2*). Within the United States, carbapenem-resistant *E. coli* carrying bla_{KPC} has been isolated in New Jersey (*3*) and Cleveland, Ohio (*4*), and 7 carbapenem-resistant *E. coli* isolates were obtained from 3 different hospitals in Brooklyn, New York (*5*). Urban et al. (*6*) recently reported 9 KPC-2 and KPC-3 carbapenemases in urinary *E. coli* isolates from 7 long-term care facilities. We report such an isolate from a resident of a long-term care facility.

This case involved a 68-year-old female resident of a long-term care facility in Centralia, Illinois, who had multiple chronic medical problems, including cerebral palsy, a seizure disorder, and recurrent urinary tract infections. A urine culture grew $>10^5$ CFU/mL of *E. coli* susceptible to amikacin, gentamicin, tobramycin, piperacillin/tazobactam, trimethoprim/sulfamethoxazole, imipenem, and nitrofurantoin. Tigecycline susceptibility was not determined. Trimethoprim/sulfamethoxazole therapy was initiated. Follow-up urine culture almost 3 weeks later again grew $>10^5$ CFU/mL of *E. coli*, now susceptible to amikacin, gentamicin, tobramycin, nitrofurantoin, and tigecycline. The isolate was resistant to imipenem and meropenem. A modified Hodge test demonstrated production of a carbapenemase (*7*), and the bla_{KPC} gene was detected by PCR at the Centers for Disease Control and Prevention (CDC). The patient was treated with a 10-day course of nitrofurantoin, 100 mg by gastrostomy tube 2× per day. Chart review indicated that contact precautions were instituted only after discovery of the second *E. coli* isolate.

Seventeen days later, a repeat urine culture grew $>10^5$ CFU/mL of *K. pneumoniae* susceptible only to amikacin, gentamicin, tobramycin, and tigecycline. No treatment was given. Follow-up urine culture grew $>10^5$ CFU/mL of *K. pneumoniae* again with a similar resistance pattern. The modified Hodge test result was positive (*7*) and was confirmed as bla_{KPC} positive by PCR at CDC. The resident was transferred to an acute care facility for further evaluation and was treated with amikacin. At completion of therapy, a repeat urine culture was negative for organisms.

Our case, like that of Urban et al. (*6*), involved a urinary isolate from a resident of a long-term care facility. As increasing numbers of resistant gram-negative rods colonize such patients, the patients may acquire a bacterium carrying a KPC plasmid conferring broad-spectrum resistance as described in our patient. These plasmids may then be laterally transferred to other gram-negatives, which may have occurred in this case.

Our case underscores the gravity of the evolutionary process of emergent, multidrug–resistant enterobacteriaceae. Even though *E. coli* strains that harbor carbapenemase genes are not ubiquitous, additional therapeutic interventions are needed to prevent the spread of these bacteria, which are likely to infect increasing numbers of patients.

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