mixed Filipino-White heritage; both had IgG persistently detected beyond 12 months of age and were monitored clinically for retinochoroiditis. Their charts contained no information regarding maternal exposure or risk factors. During the 15-year period, the rate of diagnosed congenital toxoplasmosis was 3.8 (95% CI 1.5–9.2) per million live births. There were no infant deaths for which congenital toxoplasmosis was recorded as a cause. We were unable to study fetal deaths because the corresponding cause-of-death codes were not readily available.

Historically, the lowest prevalence of T. gondii infection has been recorded in the western United States (5). The rate of clinically apparent congenital toxoplasmosis in this study was lower than that found during the late 1980s through early 1990s in the New England Newborn Screening Program initially after birth (2 per 521,555 live births [3.8 per million], versus 5 per 635,000 live births [7.9 per million], respectively) (6). However, the prevalence of T. gondii infection has decreased in the United States since the 1990s (7).

Our study is subject to several limitations. Our approach would only detect clinically apparent cases, and the results should be considered a minimal estimate of congenital infection. Some cases may not have been recorded in the electronic system, but this omission is not likely for severe illness, repeated hospital or clinic visits, or outside consultation. The small number of cases makes the rate of diagnosed congenital toxoplasmosis somewhat imprecise; a few missed cases would increase the rate considerably. In addition, we were not able to evaluate fetal deaths; however, stillbirth is reportedly a rare condition (8).

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DOI: http://dx.doi.org/10.3201/eid2009.131919

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Invasive Infection Caused by Carbapenem-Resistant Acinetobacter soli, Japan

To the Editor: Infections caused by Acinetobacter spp., especially A. baumannii, have been increasingly documented in recent years. Carbapenems tend to be empirically prescribed as first-choice drugs for severe invasive infections caused by Acinetobacter spp. other than A. baumannii because these microbes are usually susceptible to carbapenems. However, infections with carbapenem-resistant Acinetobacter spp. have been increasingly reported during the past 15 years. In A. baumannii, carbapenems are usually inactivated by intrinsic oxacillinase (OXA)-51–like, acquired OXA-23–like, or OXA-58–like carbapenemases. Moreover, production of acquired metallo-β-lactamases (MBLs) of the Verona integron (VIM), imipenenemase (IMP), or New Delhi (NDM) types has been detected among carbapenem-resistant Acinetobacter species, including A. baumannii, A. junii, A. bereziniae, A. nosocomialis, and A. pittii (1). We report a case of infection with carbapenem-resistant A. soli producing another
MBL type, Tripoli MBL 2 (TMB-2), in a man in Japan.

A man in his 60s who had mesenteric injury, pelvic fracture, and intestinal perforation from a traffic accident was admitted to Okazaki City Hospital in Aichi, Japan, on May 3, 2013. After surgery, cefmetazole was prescribed on May 6 (1 g 2×/d for 7 d). On May 12, symptoms of infection developed in the patient, and 2 sets of blood samples were drawn from different vessels for bacterial culture. The following day, cefmetazole was discontinued, and ciprofloxacin (0.3 g 2×/d) and piperacillin/tazobactam (4.5 g 2×/d) were started. Acinetobacter isolates resistant to piperacillin/tazobactam and carbapenems were then recovered from the blood samples, so piperacillin/tazobactam was discontinued on May 14. After that, ceftriaxone (2 g 2×/d) and gentamicin (0.04 g 2×/d) were successively prescribed, in addition to ciprofloxacin; the symptoms of infection improved, and all antimicrobial drugs were discontinued by May 26. Additional blood cultures performed on May 17, 21, and 28 yielded negative results for Acinetobacter spp. However, the patient’s condition worsened on June 5. Meropenem (0.5 g 4×/d) was then given, but the patient died of multiorgan failure on June 7.

The bacterial isolates from the initial blood cultures were identified as A. soli by nucleotide sequencing of the rpoB and gyrB genes and assigned identification no. HK001. MICs of β-lactams and gentamicin, amikacin, levofloxacin, ciprofloxacin, colistin, and tigecycline were below the breakpoints of susceptibility as listed in Clinical and Laboratory Standards Institute document M100-S23. Carbapenem resistance was not transferred from A. soli HK001 to Escherichia coli strain CSH-2 (metB F– NA’ Rif) by conjugation. A double-disk synergy test was initially performed by using sodium mercaptoacetic acid (SMA) (2) and ceftazidime and meropenem disks (Eiken Chemical Co., Ltd, Tokyo, Japan), and results suggested MBL production. The modified Hodge test was then performed, and ertapenem and meropenem disks gave clear positive results (data not shown). PCR was performed to detect bla_OXA-23-like, bla_OXA-24/40-like, bla_OXA-51-like, bla_OXA-58-like, bla_IMP-1 like, bla_VIM-1 like, bla_VIM-2 like, bla_SDM-1 like, bla_SMB-1 like, and bla_TMB-1 like genes. Nucleotide sequence analyses showed that the A. soli isolate harbored bla_TMB-1, and bla_OXA-58. The modified SMA-disk method (3) was reevaluated to determine whether it could successfully detect TMB-2 production in A. soli HK001. Apparent positive results were obtained when disks containing imipenem, meropenem, or ertapenem were used, particularly when the edge-to-edge distance between 2 disks containing SMA and a carbapenem, respectively, was kept at 5 mm (Figure, top row). However, when the distance between the ertapenem and SMA disks was ≥10 mm, MBL production was more difficult to detect (Figure, lower 2 rows). This finding may be the result of co-production of OXA-58 by the isolate.

More than 30 Acinetobacter species had been registered by January 2012 (4). A. soli was initially isolated from the soil of a mountain forest in South Korea in 2007 (5) and has been recovered from blood cultures of 5 neonates in Brazil (6). Carbapenem-resistant A. soli co-harboring bla_IMP-1 and bla_OXA-58–like genes was identified in April 2011 in Japan and is frequently recovered from bacteremia patients (7). TMB-1 was reported in 2012 in an Achromobacter xylosoxidans isolate from a hospital in Tripoli, Libya (8); TMB-2 was later reported in Japan.
(9). The TMB-2-producing \textit{A. soli} strain that we isolated came from a blood culture, indicating that \textit{A. soli} is a potential cause of bloodstream infections or bacteremia. \textit{A. soli} has also been detected in lice and keds of domestic animals (10), indicating that \textit{A. soli} may inhabit natural environments and that injuries and bites by arthropods might present a risk for invasive infections. Isolates of \textit{Acinetobacter} species, particularly those recovered from blood culture, should be identified to species type to enable further evaluation of the clinical significance of carbapenem-resistant \textit{A. soli} strains.

This study was supported by the Ministry of Health, Labour and Welfare, Japan (H25-Shinko-Ippan-003 and H24-Shinko-Ippan-010).

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DOI: http://dx.doi.org/10.3202/eid2009.140117

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\textbf{Spread of Vaccinia Virus to Cattle Herds, Argentina, 2011}

To the Editor: Since 1999, several zoonotic outbreaks of vaccinia virus (VACV) infection have been reported in cattle and humans in rural areas of Brazil. The infections have caused exanthematous lesions on cows and persons who milk them, and thus are detrimental to the milk industry and public health services (1,2). In Brazil during the last decade, VACV outbreaks have been detected from the north to the extreme south of the country (1–4). Because Brazil shares extensive boundaries with other South American countries, humans and cattle on dairy and beef-producing farms in those countries may be at risk of exposure to VACV. To determine if VACV has spread from Brazil to Argentina, we investigated the presence of VACV in serum samples from cattle in Argentina.

During 2011, we obtained serum samples from 100 animals (50 dairy and 50 beef cattle) on farms in Córdoba, Corrientes, Entre Ríos, and Santa Fe Provinces in Argentina (online Technical Appendix, panel A, http://wwwnc.cdc.gov/EID/article/20/9/14-0154-Techapp1.pdf). No VACV cases had been reported in humans or cattle in these provinces. However, Corrientes Province borders the Brazilian state of Rio Grande do Sul, where VACVs (Pelotas 1 and Pelotas 2 viruses) were isolated during an outbreak affecting horses in 2008 (2).

To determine the presence of neutralizing antibodies in the serum samples, we used an orthopoxvirus 70% plaque-reduction neutralization test as described (4). On the basis of previous studies that detected viral DNA in serum samples (4–6), we used real-time PCR to amplify the highly conserved orthopoxvirus vaccinia growth factor (vgf) gene DNA (P.A. Alves, unpub. methods).