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 (*1*).

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 complication of congenital toxoplasmosis (*7*). Although we found a low rate of diagnosed congenital toxoplasmosis in northern California, population-based studies to evaluate rates of the disease in other geographic areas would be beneficial.

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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), imipenemase (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, measured by the agar dilution method in accordance with the guideline M07-A9 of the Clinical and Laboratory Standards Institute (http://clsi.org), were as follows: sulbactam/ampicillin, >128 mg/L; piperacillin, >128 mg/L; tazobactam/piperacillin, >128 mg/L; cefotaxime, >64 mg/L; ceftazidime, >64 mg/L; aztreonam, 64 mg/L; cefmetazole, >128 mg/L; imipenem, 8 mg/L; meropenem, 32 mg/L; and doripenem, 32 mg/L. However, MICs of 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^r Rif^s) 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}, bla_{IMP-2}, bla_{VIM-1}, bla_{VIM-2}, bla_{NDM-1}, bla_{SMB-1}, and bla_{TMB-1} genes. Nucleotide sequence analyses showed that the *A. soli* isolate harbored bla_{TMB-2} 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

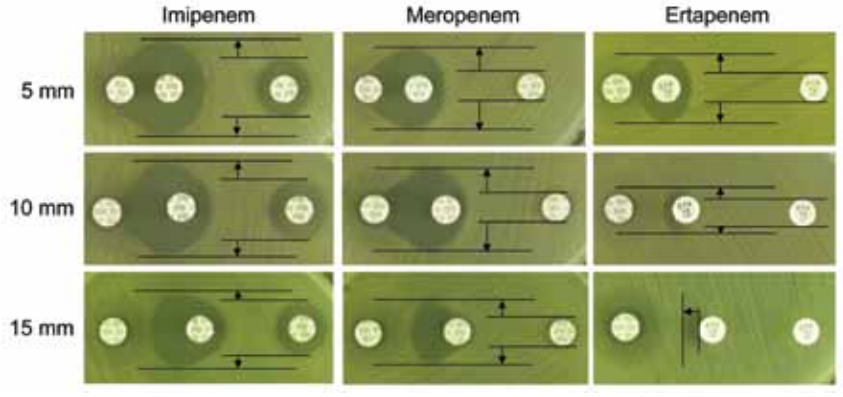

Figure. Results of double-disk synergy testing of the *Acinetobacter soli* isolate HK001 identified in a man in Japan. Testing was performed by using disks containing sodium mercaptoacetic acid (SMA) and the carbapenems imipenem, meropenem, and ertapenem. Apparent expansion of growth inhibition zone around a carbapenem disk placed near a SMA disk compared with that around a disk of carbapenem alone is seen on Mueller-Hinton agar if the isolate produces metallo-β-lactamases (*2*,*3*). When the edge-to-edge distance between 2 disks containing a carbapenem and SMA, respectively, was kept at 5 mm, expansion of the growth inhibition zone became clearer than for those kept at a distance of 10 mm and 15 mm, regardless of carbapenems used. Vertical expansion of growth inhibition zones by the effect of SMA is indicated by arrows; ertapenem gave the clearest result when the disk distance was kept at 5 mm (top right panel), even though *A. soli* HK001 co-produces oxacillinase 58–like carbapenemase, which is hardly inhibited by SMA.

(*9*). The TMB-2–producing *A. soli* strain that we isolated came from a blood culture, indicating that *A. soli* is a potential cause of bloodstream infections or bacteremia. *A. soli* has also been detected in lice and keds of domestic animals (*10*), indicating that *A. soli* may inhabit natural environments and that injuries and bites by arthropods might present a risk for invasive infections. Isolates of *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 *A. soli* strains.

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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).