BORSA initially described non-heteroresistant strains of *S. aureus* with oxacillin MIC ≤2 mg/L, which produce ample β-lactamases and are rendered fully susceptible to PRP by β-lactamase-inhibitors (*4*,*6*). Subsequent BORSA strains described have had higher oxacillin MICs (4–8 mg/L) (*4*). The proportion of BORSA among clinical isolates of *S. aureus* varies (1.4%–12.5%) but is usually ?5% (*4*,*10*). A BORSA infection outbreak among dermatology patients with severe skin diseases has also been reported (*10*). Postulated resistance mechanisms include overproduction of conventional penicillinases, production of an inducible, plasmid-mediated, membrane-bound methicillinase, and in some cases, point mutations of penicillin-binding-proteins (*4*). The clinical importance of BORSA is unknown since early clinical/animal data suggest treatment efficacy of PRP (against strains with MIC ≤2 mg/L) (*4*,*6*,*9*). Whether BORSA with higher oxacillin MICs (4–8 mg/L) will respond equally well to PRP is less clear. Further studies into the treatment of BORSA, including pharmacokinetic considerations, are needed (*4*). However, high-dose β-lactam/β-lactamase inhibitor combinations (e.g., ampicillin/sulbactam), as shown in animal models, are at least as effective as PRP (*9*). In conclusion, our report suggests that *mec*A (or PBP2a) detection may help manage serious, community-acquired, non–multidrug-resistant MRSA infections because of the potential confusion between BORSA and CA-MRSA.

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References

1. Vandenesch F, Naimi T, Enright MC, Lina G, Nimmo GR, Heffernan H, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-Valentine leukocidin genes: worldwide emergence. Emerg Infect Dis. 2003;9:978–84.
2. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. Fifteenth informational supplement. NCCLS document M100-S15. Wayne (PA): The Committee; 2005.
3. Ip M, Lyon DJ, Chio F, Enright MC, Cheng AF. Characterization of isolates of methicillin-resistant *Staphylococcus aureus* from Hong Kong by phage typing, pulsed-field gel electrophoresis, and fluorescent amplified-fragment length polymorphism analysis. J Clin Microbiol. 2003;41:4980–5.
4. Chambers HF. Methicillin resistance in staphylococci: molecular and biochemical basis and clinical implications. Clin Microbiol Rev. 1997;10:781–91.
5. Brown DF. Detection of methicillin/oxacillin resistance in staphylococci. J Antimicrob Chemother. 2001;48(Suppl 1):65–70.
6. Varaldo PE. The "borderline methicillin-susceptible" *Staphylococcus aureus*. J Antimicrob Chemother. 1993;31:1–4.
7. Naimi TS, LeDell KH, Como-Sabetti K, Borchardt SM, Boxrud DJ, Etienne J, et al. Comparison of community- and health care-associated methicillin-resistant *Staphylococcus aureus* infection. JAMA. 2003;290:2976–84.
8. Francis JS, Doherty MC, Lopatin U, Johnson CP, Sinha G, Ross T, et al. Severe community-onset pneumonia in healthy adults caused by methicillin-resistant *Staphylococcus aureus* carrying the Panton-Valentine leukocidin gene. Clin Infect Dis. 2005;40:100–7.
9. Hirano L, Bayer AS. Beta-lactam–beta-lactamase-inhibitor combinations are active in experimental endocarditis caused by beta-lactamase–producing oxacillin-resistant staphylococci. Antimicrob Agents Chemother. 1991;35:685–90.
10. Balslev U, Bremmelgaard A, Svejgaard E, Havstreym J, Westh H. An outbreak of borderline oxacillin-resistant *Staphylo-coccus aureus* (BORSA) in a dermatological unit. Microb Drug Resist. 2005;11:78–81.

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Rickettsia massiliae Human Isolation

To the Editor: The number of new rickettsial species that cause diseases in humans is rapidly increasing (*1*). Moreover, many of the species first described in ticks have been recently shown to be pathogenic. Of the 10 species or subspecies found to be pathogens after 1984, a total of 7 were first isolated from ticks (*2*). We report the first isolation of *Rickettsia massiliae* from a patient. The bacterium was isolated in Sicily in 1985 and identified in 2005.

A 45-year-old man was hospitalized in Palermo, Italy, on June 6, 1985, for fever and a rash. He had been febrile since May 25 and did not respond to antimicrobial drug treatment using cefamezin, a first-generation cephalosporin. On examination, he had a necrotic eschar on his right ankle, a maculopapular rash on his palms and soles (online Appendix Figure 1, available at http://www.cdc.gov/ncidod/EID/vol12no01/05-0850-G1.htm), and slight hepatomegaly. Leukocyte count was normal; he received tetracyclines for 13 days and fully recovered. He seroconverted (from 0 to 1:80 between day 11 and day 24) by indirect immunofluorescence to *Rickettsia conorii* (*R. conorii* spot, bioMérieux, Marcy l'Étoile, France).

Four milliliters of heparinized blood sampled before treatment were inoculated in a 25-cm^2 flask containing Vero cells and incubated at 33°C in a CO_2 incubator (*1*). Direct immunofluorescence test on a sample of the patient's serum was positive 7 days later. The strain was stored for 20 years and tested in 2005 at the Unité des Rickettsies for identification, and *R. massiliae* was identified. DNA was extracted from the cell culture supernatant and used as template in 2 previously described polymerase

chain reaction (PCR) assays that targeted a portion of the rickettsial *ompA* gene as well as a portion of the rickettsial *gltA* gene (*3*,*4*). Amplification products of the expected size were obtained from this extract but from no concurrently processed control materials, including 3 negative controls. DNA sequencing of the positive PCR products gave 100% identity with *R. massiliae* for *ompA* (GenBank accession no. RBU43792) and 99.9% homology for *gltA* (GenBank accession no. RSU 59720).

R. massiliae was first isolated from *Rhipicephalus* ticks in Marseilles (*5*). It is transmitted transovarially in *Rhipicephalus turanicus* (*2*). *R. massiliae* is commonly found in *Rhipicephalus sanguineus* or *R. turanicus* in France, Greece, Spain (identified as Bar 29) (*6*), Portugal, Switzerland, Sicily (D. Raoult, unpub. data), Central Africa, and Mali (*2*). *R. massiliae* may be commonly associated with these ticks, which are distributed worldwide.

R. massiliae is grouped phylogenically with *Rickettsia rhipicephali* and *Rickettsia aeschlimannii* (online Appendix Figure 2, available at http://www.cdc.gov/ncidod/EID/vol12no01/05-0850-G2.htm). Bacteria from this group have a natural resistance to rifampin that is associated with an *rpo*B sequence that is different from that of other rickettsiae. This isolate was not tested for antimicrobial drug susceptibly (*7*). Rifampin resistance leads us to believe that this isolate may cause a Mediterranean spotted fever–like disease that was described in children in Spain (*7*,*8*). Serologic findings were recently reported that showed some patients in Barcelona, Spain, with reactions that indicate *R. massiliae* (B29 strain) rather than *R. conorii* (*6*). However, serologic reactions are only presumptive; isolation from a patient is the required to initially describe a new disease (*9*).

This Sicilian index case shows that *R. massiliae* is a human pathogen. It contraindicates using rifampin to treat Mediterranean spotted fever in areas where *R. massiliae* is endemic, as it cannot as yet be differentiated from *R. conorii* infection. *R. massiliae* is a new example of a strain identified in ticks for several years before its first isolation from a human patient (*10*). The longest delay was observed for *Rickettsia parkeri*, which was isolated from ticks in 1939 but not from a patient until 2004. Many authors labeled *R. parkeri* a nonpathogenic rickettsia during this time (*1*). In the present case, the human isolate was obtained before the tick isolate but was not further identified. When this strain was isolated, *R. conorii* was the sole *Rickettsia* sp. found in ticks in southern Europe. Moreover, only 1 tickborne pathogenic *Rickettsia* sp. was believed to circulate in a single area. Since that time, several tickborne rickettsial diseases have been shown to exist in the same area, which prompted us to retrospectively identify this strain. The patient was reexamined in May 2005, after this identification. He is healthy and has no remaining antibodies against *Rickettsia* spp.

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References

1. Raoult D, Roux V. Rickettsioses as paradigms of new or emerging infectious diseases. Clin Microbiol Rev. 1997;10:694–719.
2. Matsumoto K, Ogawa M, Brouqui P, Raoult D, Parola P. Transmission of *Rickettsia massiliae* in the tick, *Rhipicephalus turanicus*. Med Vet Entomol. 2005;19:263–70.
3. Roux V, Fournier PE, Raoult D. Differentiation of spotted fever group rickettsiae by sequencing and analysis of restriction fragment length polymorphism of PCR amplified DNA of the gene encoding the protein r*OmpA*. J Clin Microbiol. 1996;34:2058–65.
4. Roux V, Rydkina E, Eremeeva M, Raoult D. Citrate synthase gene comparison, a new tool for phylogenetic analysis, and its application for the rickettsiae. Int J Syst Bacteriol. 1997;47:252–61.
5. Beati L, Raoult L. *Rickettsia massiliae* sp.nov., a new spotted fever group rickettsia. Int J Syst Bacteriol. 1993;43:839–40.
6. Cardenosa N, Segura F, Raoult D. Serosurvey among Mediterranean spotted fever patients of a new spotted fever group rickettsial strain (Bar29). Eur J Epidemiol. 2003;18:351–6.
7. Drancourt M, Raoult D. Characterization of mutations in the *rpoB* gene in naturally rifampin-resistant *Rickettsia* species. Antimicrob Agents Chemother. 1999;43:2400–3.
8. Bella F, Espejo-Arenas E, Uriz S, Serrano JA, Alegre MD, Tort J. Randomized trial of five-day rifampin versus one-day doxycycline therapy for Mediterranean spotted fever. J Infect Dis. 1991;164:433–4.
9. Parola P, Paddock CD, Raoult D. Tickborne rickettsioses around the world: emerging diseases challenging old concepts. Clin Microbiol Rev. 2005;18:719–56.
10. Paddock CD, Sumner JW, Comer JA, Zaki SR, Goldsmith CS, Goddard J, et al. *Rickettsia parkeri*: a newly recognized cause of spotted fever rickettsiosis in the United States. Clin Infect Dis. 2004;38:805–11.

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