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Address for correspondence: Kerstin Wernike, Institute of Diagnostic Virology, Friedrich-Loeffler-Institut, Südufer 10, 17493 Greifswald-Insel Riems, Germany; email: kerstin.wernike@fli.de

Establishment of *Amblyomma maculatum* Ticks and *Rickettsia parkeri* in the Northeastern United States

Goudarz Molaei, Noelle Khalil, Carmen J. Ramos, Christopher D. Paddock

Author affiliations: Yale School of Public Health, New Haven, Connecticut, USA (G. Molaei); Connecticut Agricultural Experiment Station, New Haven (G. Molaei, N. Khalil); Centers for Disease Control and Prevention, Atlanta, Georgia, USA (C.J. Ramos, C.D. Paddock)

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We document a case of *Rickettsia parkeri* rickettsiosis in a patient in Connecticut, USA, who became ill after a bite from a Gulf Coast tick (*Amblyomma maculatum*). We used PCR to amplify *R. parkeri* DNA from the detached tick. The patient showed a 4-fold rise in IgG reactive with *R. parkeri* antigens.

Native and invasive tick species pose serious public health concerns in the United States, particularly in northeastern states. Recent and rapid expansion of the lone star tick (*Amblyomma americanum*) into ranges with pervasive blacklegged tick (*Ixodes scapularis*) populations has increased the number of recognized tickborne pathogens that circulate in that densely populated region. In addition to *Borrelia burgdorferi*, the causative agent of Lyme disease, ≥ 7 additional tickborne pathogens are now endemic to the northeastern United States: *Ehrlichia chaffeensis*, *Ehrlichia ewingii*, Heartland virus, *Anaplasma phagocytophilum*, *Borrelia miyamotoi*, *Babesia microti*, and Powassan virus (1). Multiple factors, including climate change and anthropogenic modifications to the environment, have affected rapid expansion of the ranges of medically relevant tick species and associated pathogens. That expansion has been reflected by dramatic increases in the numbers of reported cases of tickborne diseases in the northeastern United States since the beginning of the 21st Century (1).

The Gulf Coast tick (*Amblyomma maculatum*) was first identified in the United States in 1844. As recently as the middle of the 20th Century, the tick's range was restricted predominantly to coastal regions of states bordering the Gulf of Mexico as far west as Texas and the southern Atlantic coast only as far north as southern North Carolina (Figure 1) (2,3). Established *A. maculatum* tick populations now exist in states hundreds of miles inland (Arkansas, Missouri, Kentucky, Illinois, Indiana) and along the Atlantic coast as far north as Connecticut (4–9). Migratory grassland birds serve a crucial role in the spread of Gulf Coast ticks to locations in central and northern states that possess favorable environmental conditions for the tick's survival (2,8).

The Gulf Coast tick was relatively unknown and infrequently studied until recognition of *Rickettsia parkeri* spotted fever rickettsiosis in 2004 (2). In contrast to its more widely recognized cousins, blacklegged and lone star ticks, which prefer predominantly woodland habitats, Gulf Coast ticks favor grassland habitats. During the past 250 years, huge swathes of native grasslands and savannahs in the eastern United States have been transformed into agricultural areas and rangeland, creating habitats no longer favorable for Gulf Coast ticks. Paradoxically, contemporary reclamation of native grasslands through conservation

efforts in the northeastern United States might have inadvertently led to establishment of Gulf Coast ticks in that region. The recent discovery of established populations of Gulf Coast ticks in reclaimed grasslands at the former Freshkills landfill on Staten Island, New York, is a salient example of this phenomenon (7,8). Of note, restored grassland habitats often occur near or within shorelines, parks, and wildlife areas proximate to and frequented by persons residing in densely populated metropolitan areas.

The Gulf Coast tick is the principal vector of *R. parkeri*, a pathogen that causes a rickettsiosis similar to but milder than Rocky Mountain spotted fever (2). Rates of *R. parkeri* infection are as high as 56% among questing adult Gulf Coast ticks in some regions, and 23%–53% of adult specimens obtained in Connecticut, New York, and New Jersey are infected (6–9). Gulf Coast ticks readily bite humans, posing another risk for tickborne disease in northeastern United States, particularly among persons who reside and recreate near or within New York, New York; New Haven, Connecticut; Newark, New Jersey; and Philadelphia, Pennsylvania, where recently established tick populations have been identified in areas where human infections with this pathogen had not been previously documented (Figure 1).

In August 2023, a 29-year-old woman discovered a tick attached to the nape of her neck after visiting a beach in Fairfield County, Connecticut. The tick (Figure 2, panels A, B) was attached for ≤ 4 hours before it

was removed. Within 3 days, a small, erythematous, crusted lesion with a smaller satellite papule developed at the bite site (Figure 2, panel C), after which the patient experienced chills, fatigue, cervical lymphadenopathy, myalgia, severe headache, and mild confusion. Approximately 10 days later, several small erythematous macules developed on her arm and legs (Figure 2, panel D). The patient recovered rapidly after treatment with doxycycline. We performed PCR to amplify *R. parkeri* DNA from the detached tick; indirect immunofluorescence antibody assay results of patient serum samples revealed IgG reactive with antigens of *R. parkeri* at titers of <32 at 15 days and 256 at 24 days after illness onset (Appendix, <https://wwwnc.cdc.gov/EID/article/30/10/24-0821-App1.pdf>).

Because of morphologic similarities between Gulf Coast ticks and American dog ticks (*Dermacentor variabilis*, the principal vector of Rocky Mountain spotted fever in the northeastern United States), the 2 species can be misidentified. Because most tick species are associated with a unique suite of pathogens, it is critical to improve regional capacity for accurately detecting and identifying specific ticks and the pathogens they transmit in the northeastern United States, an area already endemic for Lyme disease, hard tick relapsing fever, Rocky Mountain spotted fever, ehrlichiosis, anaplasmosis, and Powassan virus infections (10). The rapidly changing dynamics and evolving risks of tickborne diseases across this region reinforce the need for awareness of and education on tick bite prevention strategies, including using repellents registered with the Environmental

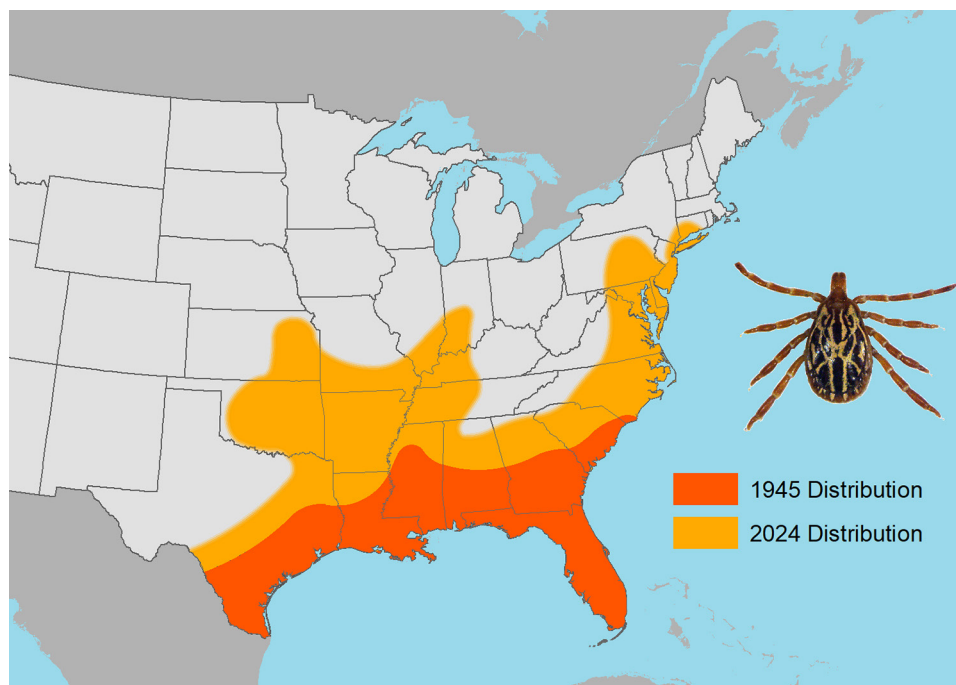


Figure 1. Generalized distributions of the Gulf Coast tick, *Amblyomma maculatum* (pictured), a human-biting tick species newly established in the northeastern United States, in 1945 compared with 2024. Establishment was defined as >6 ticks of the same life stage identified within a 12-month period or ticks of >1 life stage identified within a 12-month period. Data from references 2–9, <https://www.dep.pa.gov/Business/ProgramIntegration/Vector-Management/Ticks/Pages/default.aspx>, and https://www.in.gov/health/idepd/zoonotic-and-vectorborne-epidemiology-entomology/vector-borne-diseases/tick-borne-diseases/amblyomma-maculatum-gulf-coast-tick/#Geographic_Distribution.

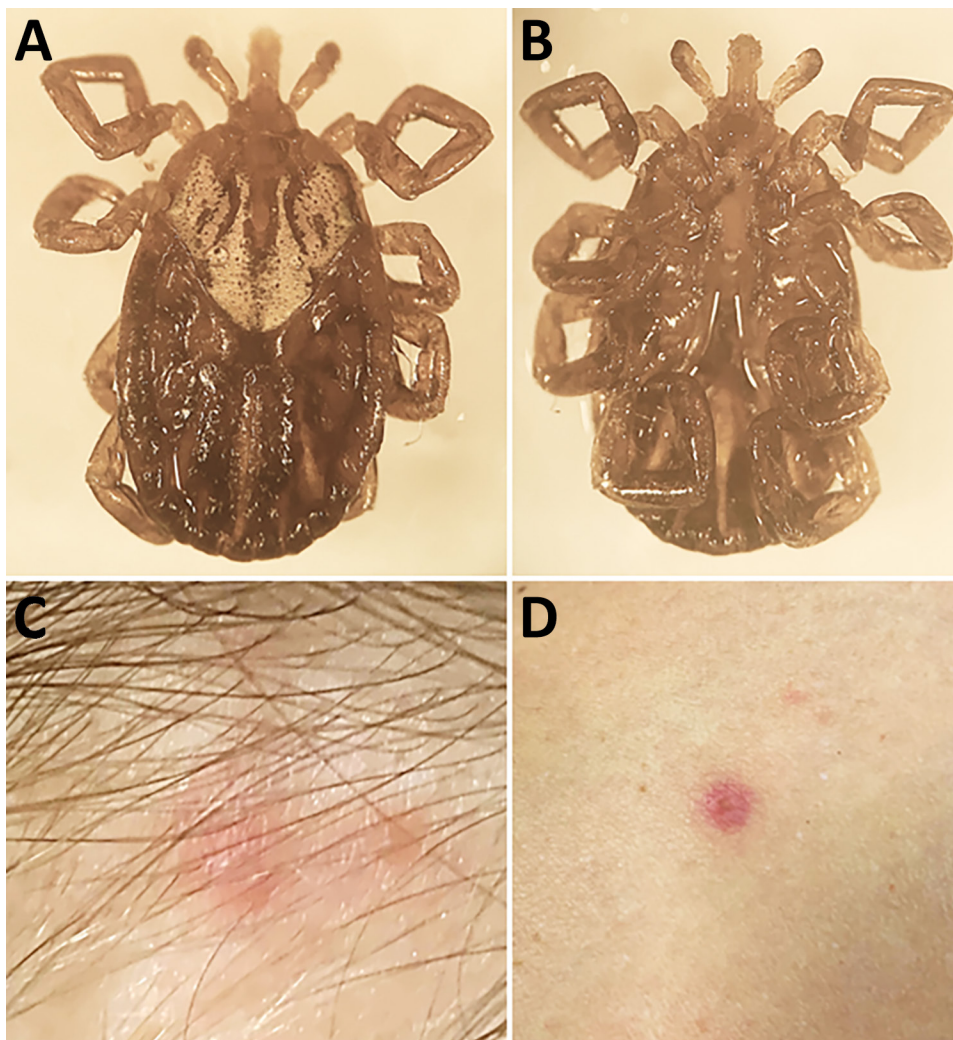


Figure 2. Biting *Amblyomma maculatum* tick removed from a woman in Connecticut, USA, and signs of *Rickettsia parkeri* rickettsiosis. A, B) Dorsal (A) and ventral (B) images of the tick. C, D) A small, erythematous, crusted lesion with a smaller satellite papule that developed at the bite site (C) and 1 of several small erythematous macules that developed on her arm and legs (D).

Protection Agency and performing regular, thorough tick checks after exposure to tick-infested areas.

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About the Author

Dr. Molaei is a research scientist and director of the Tick and Tick-borne Pathogen Surveillance Program at the Center for Vector Biology and Zoonotic Diseases and the Northeast Regional Center for Excellence in Vector-Borne Diseases of the Connecticut Agricultural Experiment Station, and also an associate clinical professor at the Yale School of Public Health. His current research interests include the ecology and epidemiology of tickborne and mosquitoborne pathogens of human health concern.

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Address for correspondence: Goudarz Molaei, Department of Entomology, Connecticut Agricultural Experiment Station, 123 Huntington St, New Haven, CT 06511, USA; email: goudarz.molaei@ct.gov

Fort Sherman Virus Infection in Human, Peru, 2020

Edmilson F. de Oliveira-Filho, César Augusto Cabezas Sánchez, Dora Esther Valencia Manosalva, Maribel Dana Figueroa Romero, Nancy Susy Merino Sarmiento, Adolfo Ismael Marcelo Ñique, Edward Málaga-Trillo, Andres Moreira-Soto, Maria Paquita García Mendoza, Jan Felix Drexler

Author affiliations: Charité-Universitätsmedizin Berlin, Freie Universität Berlin and Humboldt-Universität zu Berlin, Institute of Virology, Berlin, Germany (E.F. de Oliveira-Filho, A. Moreira-Soto, J.F. Drexler); Instituto Nacional de Salud, Lima, Peru (C.A. Cabezas Sánchez, M.D. Figueroa Romero, N.S. Merino Sarmiento, A.I.M. Ñique, M.P. García Mendoza); Laboratorio de Referencia Regional de Salud Pública de Lambayeque, Lambayeque, Perú (D.E. Valencia Manosalva); Universidad Peruana Cayetano Heredia, Lima (E. Málaga-Trillo); German Centre for Infection Research, Berlin (J.F. Drexler)

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Fort Sherman virus (FSV) was isolated in Panama in 1985 from a US soldier. We report a case of human FSV infection in a febrile patient from northern coastal Peru in 2020. FSV infections spanning ≈35 years and a distance of 2,000 km warrant diagnostics, genomic surveillance, and investigation of transmission cycles.

In 1985, the orthobunyavirus Fort Sherman virus (FSV) was discovered in a US soldier with acute febrile disease who was based in a jungle warfare training center in Panama (1). Two FSV strains were isolated from mosquitoes in Argentina in 1965 and 1982 (2). FSV was found in healthy horses in Brazil in 2018, (2) and in horses in Argentina showing neurologic and abortive disease in 2013 (3). Serologic analyses of horse-associated FSV strains have suggested a broad vertebrate host range in peridomestic animals; seroprevalence has ranged from 2.9% in goats to 22.0% in horses in Brazil (2) and 5.7% in humans in Argentina (4).

We describe a case of human FSV infection in a patient with febrile illness sampled in March 2020 in the city of Chiclayo in Lambayeque department on the northern coast of Peru (Figure 1). The patient was a 61-year-old man with no recent travel history and fever of 38°C. Results of diagnostic tests were negative, including dengue virus (DENV)-specific real-time reverse transcription PCR (RT-PCR) and broadly reactive nested RT-PCRs targeting flaviviruses and alphaviruses. Expanded diagnostic investigation yielded a positive result for orthobunyaviruses using a broadly reactive RT-PCR (Appendix, <https://wwwnc.cdc.gov/EID/article/30/10/24-0124-App1.pdf>). We identified the virus as FSV by sequencing of the screening PCR amplicon (Appendix). We obtained complete coding sequences of all 3 genome segments by amplifying overlapping genome fragments using nested RT-PCR, followed by Sanger sequencing (Appendix). Virus isolation failed despite repeated attempts, potentially because of sample degradation and a relatively low viral load of 3.7×10^2 viral RNA copies/mL of blood quantified using published FSV-specific real-time RT-PCR (2).

To investigate the extent of FSV infection in Lambayeque, we examined all 582 available serum samples from febrile persons sent for diagnostics to the local reference laboratory from Peru's Ministry of Health during 2020 using RT-PCR for orthobunyaviruses. Of the samples, 70.4% (410/582 [95% CI 66.6–74.0]) tested positive for DENV, but no samples tested positive for FSV, other orthobunyaviruses, alphaviruses, or other flaviviruses (Appendix Table 3, Appendix Figure).