The Evolving Medical and Veterinary Importance of the Gulf Coast tick (Acari: Ixodidae)

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ABSTRACT *Amblyomma maculatum* Koch (the Gulf Coast tick) is a three-host, ixodid tick that is distributed throughout much of the southeastern and south-central United States, as well as several countries throughout Central and South America. A considerable amount of scientific literature followed the original description of *A. maculatum* in 1844; nonetheless, the Gulf Coast tick was not recognized as a vector of any known pathogen of animals or humans for >150 years. It is now identified as the principal vector of *Hepatozoon americanum*, the agent responsible for American canine hepatozoonosis, and *Rickettsia parkeri*, the cause of an emerging, eschar-associated spotted fever group rickettsiosis identified throughout much of the Western Hemisphere. Coincident with these discoveries has been recognition that the geographical distribution of *A. maculatum* in the United States is far more extensive than described 70 yr ago, supporting the idea that range and abundance of certain tick species, particularly those with diverse host preferences, are not fixed in time or space, and may change over relatively short intervals. Renewed interest in the Gulf Coast tick reinforces the notion that the perceived importance of a particular tick species to human or animal health can be relatively fluid, and may shift dramatically with changes in the distribution and abundance of the arthropod, its vertebrate hosts, or the microbial agents that transit among these organisms.

KEY WORDS Gulf Coast tick, *Amblyomma maculatum*, *Rickettsia parkeri*, *Hepatozoon americanum*

Introduction

The Gulf Coast tick (*Amblyomma maculatum* Koch) is a three-host, Nearctic and Neotropical ixodid species distributed throughout much of the Western Hemisphere. Morphological similarities between *A. maculatum* and the closely related species *Amblyomma triste* Koch and *Amblyomma tigrinum* Koch have confounded the recognized distribution of *A. maculatum* in the Americas (Kohls 1956, Guglielmone et al. 1982, Mendoza-Uribe and Chávez-Chorocco 2004, Estrada-Pena et al. 2005, Mertins et al. 2010, Abarca et al. 2012); nonetheless, authenticated collection records of *A. maculatum* are described from Belize, Costa Rica, Colombia, Ecuador, Guatemala, Honduras, Mexico, Nicaragua, Peru, Venezuela, and the United States (Estrada-Pena et al. 2005).

Scientific interest in *A. maculatum* has cycled considerably in the past 100 yr. During the first half of the 20th century, the Gulf Coast tick was recognized predominantly by its detrimental economic impact on livestock in the southern United States. Despite recurring discoveries of novel tick-borne pathogens from the late 1970s through the mid-1990s (Piesman and Spielman 1980; Burgdorfer et al. 1982; Spielman et al. 1993; Anderson et al. 1993; Telford et al. 1996, 1997; Paddock and Yabsley 2007), the medical and veterinary importance of *A. maculatum* remained largely unrecognized. Interest in the Gulf Coast tick was rekindled in 1998 when *A. maculatum* was identified a potential vector of *Hepatozoon americanum*, the agent of American canine hepatozoonosis (Mathew et al. 1998), and again in 2004, following the discovery of *Rickettsia parkeri* rickettsiosis, a rickettsial disease of humans transmitted by the Gulf Coast tick (Paddock et al. 2004). A comprehensive description of the zoogeography, phenology, life history, biology, and ecology of *A. maculatum* was presented recently (Teel et al. 2010); this review summarizes current knowledge regarding the dynamic nature of the Gulf Coast tick's impact on the health of humans and animals in the United States.

History

In 1844, German arachnologist C.L. Koch described the type specimen of *A. maculatum* as a large ornate tick, collected from the somewhat ambiguous locale of "Carolina" (Koch 1844; Fig. 1). The physical characteristics, biology, geographical distribution, host associations, and detrimental effects to livestock of the Gulf Coast tick were subsequently documented during the

The findings and conclusions are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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Fig. 1. Left, a 19th century drawing of an adult male specimen of the Gulf Coast tick, by C. L. Koch, who noted, "The nature of the large designs is elongate, oval, domed, long rows of fine, dotted, pale yellow with dark iron buff; the posterior margin shields over iron buff, alternating with a white spot. Legs iron buff, anterior margin of the limbs narrow white, 1 2/3 inch long in males." (Koch 1844). Right, photograph of an adult male *A. maculatum.* Image courtesy of Ellen Stromdahl and Graham Snodgrass (United States Army Public Health Command, Aberdeen Proving Ground, MD).

first half of the 20th century (Hooker et al. 1912), particularly after it was determined that parasitism by *A. maculatum* caused considerable economic loss to the livestock industry in the southern United States by facilitating severe and sometimes lethal myiasis caused by the larvae of the primary screwworm, *Cochliomyia hominivorax* (Coquerel) (Bishopp and Hixson 1936, Spicer and Dove 1938, Hixson 1940, Blakeslee et al. 1947). As many as 40–80% of screwworm infestations of livestock reported during the 1930s were attributable to Gulf Coast ticks. Surveys for screwworm infestations in Texas during 1937 revealed a frequency of infection among cattle in nine littoral counties that was approximately six times greater than the rate observed among 13 southern counties more distant from the coast, attributed largely to the distribution of *A. maculatum* (Spicer and Dove 1938). Gulf Coast ticks characteristically concentrate on the inner surface of ear of livestock where their feeding activity create cutaneous lesions and exudates that provide an ideal substrate for screwworm flies to deposit their eggs. Bishopp and Hixson (1936) described the gruesome and often devastating consequences of this process:

> The mass of ticks in the ear causes it to swell and become stiff. This prevents the host from flicking the ear and thus permits blowflies to oviposit undisturbed. The resulting larvae burrow into the ear tissues, and the blood and serum discharged attract other flies. The entire ear may be eaten off in a few days, and the jaw, throat and eye may become infested as a result of the discharges running from the ear. If treatment is neglected, the skull may be laid bare and the animal may die.

Ranchers occasionally cut the ears off of their cattle in a drastic effort to stem this condition (Gladney et al. 1977). Following successful eradication of screwworm populations in the 1970s, many ranchers relaxed tick control measures, resulting in an expansion of Gulf Coast tick populations, as well as increases in economic losses attributable to injuries caused directly by tick infestations (Koch and Hair 1975, Gladney 1976, Gladney et al. 1977).

E.V. Cowdry was the first investigator to document a microbial agent in *A. maculatum* when he described minute intracellular bacteria in the tissues and eggs of specimens collected near the Mississippi Gulf Coast town of Vancleave (Cowdry 1923). The Gulf Coast tick was not identified conclusively as a vector of any known pathogen until the end of the 20th century (Mathew et al. 1998); nonetheless, a team of medical entomologists led by R. R. Parker from the Rocky Mountain Laboratory in Hamilton, MT, postulated a role for disease transmission by this tick many decades earlier. During 1937, Parker and coworkers evaluated >4,000 specimens of *Dermacentor variabilis* (Say), *Amblyomma americanum* (L.), and *Rhipicephalus sanguineus* (Latreille) ticks collected in Liberty County, TX, following reports of several illnesses diagnosed as Rocky Mountain spotted fever (RMSF). Surprisingly, none of these tick species revealed evidence of infection with a pathogenic *Rickettsia* species; nonetheless, two strains of a previously unknown spotted fever group *Rickettsia*, subsequently designated as the "maculatum agent," were isolated from pools of *A. maculatum* ticks collected from the same locality (Parker et al. 1939). Male guinea pigs infected experimentally with the the maculatum agent developed fevers of short duration and moderate scrotal swelling and erythema, but always recovered, unlike guinea pigs inoculated with *Rickettsia rickettsii* (the agent of RMSF), which generally resulted in higher fevers, more severe scrotal reactions, and were often lethal to the animal. Parker maintained that, "Final decision as to whether

maculatum infection is or is not a new disease entity must await further research" (Parker 1940).

Several years later and under similar circumstances, investigators were unable to detect *R. rickettsii* from any of >3,200 *Amblyomma*, *Dermacentor*, and *Ixodes* ticks collected during 1947–1949 in Jackson County in southeastern Mississippi, despite multiple reports from that county of serologically confirmed RMSF during the previous decade. However, seven isolates of the maculatum agent were cultured from 19 pools comprising 488 Gulf Coast ticks from that area (Philip and White 1955). The maculatum agent was eventually recognized as a unique spotted fever group *Rickettsia* species, and named *R. parkeri* (Lackman et al. 1965). *R. parkeri* remained relatively unnoticed for the next several decades until it was confirmed as a pathogen of humans in 2004 (Paddock et al. 2004).

Ecological and Anthropogenic Influences on the Distribution of *A. maculatum*-Associated Diseases

Until relatively recently, the U.S. distribution of *A. maculatum* was described as a continuous and somewhat narrow band extending approximately 100–150 miles inland from the Gulf Coast of Texas, across the southern states, to the Atlantic Coast of South Carolina (Hooker et al. 1912, Bishopp and Hixson 1936, Cooley and Kohls 1944, Bishopp and Trembley 1945; Fig. 2). Data accumulated during the past 50 yr suggest qualitative and quantitative changes in the historically accepted range of *A. maculatum*, including established populations >250 miles inland in several states bordering the Gulf of Mexico and Atlantic Ocean and in several land-locked states, including Arkansas, Kansas, Kentucky, and Oklahoma (Semtner and Hair 1973, Goddard and Paddock 2005, Goddard 2007, Trout et al. 2010, Teel et al. 2010, Pagac et al. 2014; Fig. 3). The dynamics of these changes have important implications concerning the diseases transmitted by the Gulf Coast tick. Indeed, autochthonously acquired infections with *R. parkeri* and *H. americanum* have been identified repeatedly in states outside the mid-20th century range estimates of *A. maculatum*, reflecting the critical link between the changing ecology of the Gulf Coast tick and the epidemiology of the diseases it transmits.

Permanent populations of *A. maculatum* now exist in several states where few or no records of this species existed during the first half of the 20th century. The largest and best characterized inland incursion has occurred in the southern Great Plains. Incidental collection records for *A. maculatum* in southeastern Oklahoma were first documented during the early 1940s (Cooley and Kohls 1944, Bishopp and Trembley 1945). By 1972, the distribution of *A. maculatum* had expanded considerably to include 18 counties of northeastern and south-central Oklahoma, as well as parts of southeastern Kansas (Semtner and Hair 1973). During the following 30 yr, Gulf Coast ticks were identified throughout 65% of Oklahoma counties and 18% of Kansas counties, subsequently coalescing to form a contiguous inland distribution that now involves thousands of square miles of upland prairie (Barker et al. 2004, Teel et al. 2010). The epidemiological consequences of this inland range incursion are reflected in part by occurrence of >100 confirmed canine cases of American canine hepatozoonosis in Oklahoma since the recognition of this disease in the late 1990s (Cummings et al. 2005, Johnson et al. 2009).

Examples of regional and incremental changes in the distribution and relative abundance of Gulf Coast ticks also exist in other states that were considered outside of the historically recognized boundaries of *A. maculatum*. The earliest collection records of *A. maculatum* in North Carolina are from the early 1980s (Harrison et al. 1997); nonetheless, increasing numbers of Gulf Coast tick specimens submitted to the North Carolina Department of Environment and Natural Resources during a tick-attachment project from 2009–2010 prompted surveys that identified *A. maculatum* throughout several counties in the Piedmont and Coastal Plain regions of the state (Varela-Stokes et al. 2011). In Arkansas, Gulf Coast ticks were identified only rarely and sporadically during the 1950s through the 1980s (Lancaster 1973, Koch 1982); however, surveys conducted during 2006–2009 consistently identified immature and adult specimens of *A. maculatum* from domesticated animals and wildlife in least 25 counties of Arkansas (Trout et al. 2010a,b). Gulf Coast ticks have been collected in western Kentucky since the mid-1980s (Goddard and Norment 1983, Snoddy et al. 1984, Bloemer et al. 1988, Scoles 2004, Jiang et al. 2012, Pagac et al. 2014), and a confirmed case of *R. parkeri* rickettsiosis was identified in 2006 from Mercer County in central Kentucky (Paddock et al. 2008). Collectively, these observations indicate that established populations of *A. maculatum* occur in Arkansas, Kentucky, and North Carolina, far beyond what was considered historically the northern boundaries for this tick.

In 1936, Bishopp and Hixson suggested that southern coastal habitats with "rather high rainfall, humidity, and temperature" defined the necessary parameters for distribution of the Gulf Coast tick. Nonetheless, *A. maculatum* is extremely well-adapted to relatively drier grassland and savanna landscapes, particularly the coastal upland and tall-grass prairies of the United States (Hixson 1940, Semtner and Hair 1973, Teel et al. 2010), where it effectively regulates water balance in these open habitats. Indeed, *A. maculatum* has a low net transpiration rate for all its feeding stages that allows it to be surprisingly xerophilic when compared with other ixodid species distributed sympatrically across the southeastern United States (Hair et al. 1975, Goddard 1997, Needham and Teel 1991, Yoder et al. 2008). Host-seeking adult *A. maculatum* ticks remain active during mid-to-late summer, are characteristically found in open, nonshaded habitats, and are most responsive to host stimuli during the hottest period of the day (Scrifres et al. 1988, Clark et al. 1998, Goddard and Varela-Stokes 2009, Goddard et al. 2011, Pagac et al. 2014). Furthermore, adult *A. maculatum* ticks often quest on vegetation for several days without descending to the duff layer (Fleetwood and Teel 1983, Needham and Teel 1986), and it is believed that the

Fig. 2. A distribution map of *A. maculatum* in the United States for 1945 determined using tick collection data from the United States Bureau of Entomology and Plant Quarantine (Bishopp and Trembley 1945). Large dots indicate specific sites from which Gulf Coast ticks were collected, and small dots represent the probable distribution of the Gulf Coast tick, determined by the authors. Reprinted with permission from the *Journal of Parasitology.*

low net transpiration rate of the Gulf Coast tick contributes to its survival and success in native grassland habitats of Kansas and Oklahoma (Yoder et al. 2008). These environments are comparatively more xeric and hundreds of miles distant from the moisture-rich coastal areas considered by early investigators as the required habitat for this species.

In the United States, the Gulf Coast tick has been collected from at least 71 species of birds and mammals, comprising seven taxonomic classes of vertebrates (Teel et al. 2010). The immature stages of *A. maculatum* parasitize a great diversity of passerine birds, including many members of the Corvidae, Emberizidae, Icteridae, Laniidae, Mimidae, and Trogodytidae (Hixson 1940; Semtner and Hair 1973; Teel et al. 1988, 1998, 2010; Robbins et al. 2010; Florin et al. 2014), as well as bobwhite quail (*Colinus virginianus*), cotton rats (*Sigmodon hispidus hispidus*), and various species of mice and voles (Teel et al. 2010). Adult-stage Gulf Coast ticks parasitize a varied group of medium-to-large mammalian hosts that include white-tailed deer (*Odocoileus virginianus*), coyotes (*Canis latrans*), dogs, cattle, horses, sheep, and swine (Teel et al. 2010, Duell et al. 2013). The wide host range of *A. maculatum* and its capacity to withstand relatively drier, hotter, and more exposed habitats contribute in part to recently recognized range extensions; nonetheless, physiological plasticity is insufficient to explain entirely the contemporaneous spread of this ixodid species. In this context, it is possible that various anthropogenic interventions, including those that create preferred habitats or increase the abundance and distribution of certain host species, have also contributed to this process.

It is likely that various domesticated and wildlife hosts of *A. maculatum*, particularly several species whose distributions have been affected profoundly by recent human activities, have been involved in phoretic transfers of Gulf Coast ticks to favorable habitats during the last 50 yr. As one example, it is hypothesized that Gulf Coast ticks were introduced to Kansas and Oklahoma attached to cattle from the Gulf Coast, when ranchers relocated these animals to forage-rich pasturage of the upland prairie (Semtner and Hair 1973). European swine (*Sus scrofa*) were introduced initially into Florida during the 16th century by Spanish explorers and the release of free-ranging swine throughout much of the southeastern United States continued until the middle of the 20th century. Feral pigs can serve as hosts for large numbers of adult Gulf Coast ticks (Bishopp and Hixson 1936, Hanson and Karstad 1959, Smith 1982, Greiner et al. 1984, Allan et al. 2001, Shender et al. 2002, Sanders et al. 2013). Because these animals birth large litters, are highly adaptive to various habitats and food sources, and have essentially no predators other than humans, feral swine populations in the United States have increased in scope and magnitude, with estimates of >4 million animals by 2007 (Clay 2007). During the past 20 yr, substantial northward range extensions of feral swine have been recognized in Arkansas, Georgia, Mississippi, and North Carolina

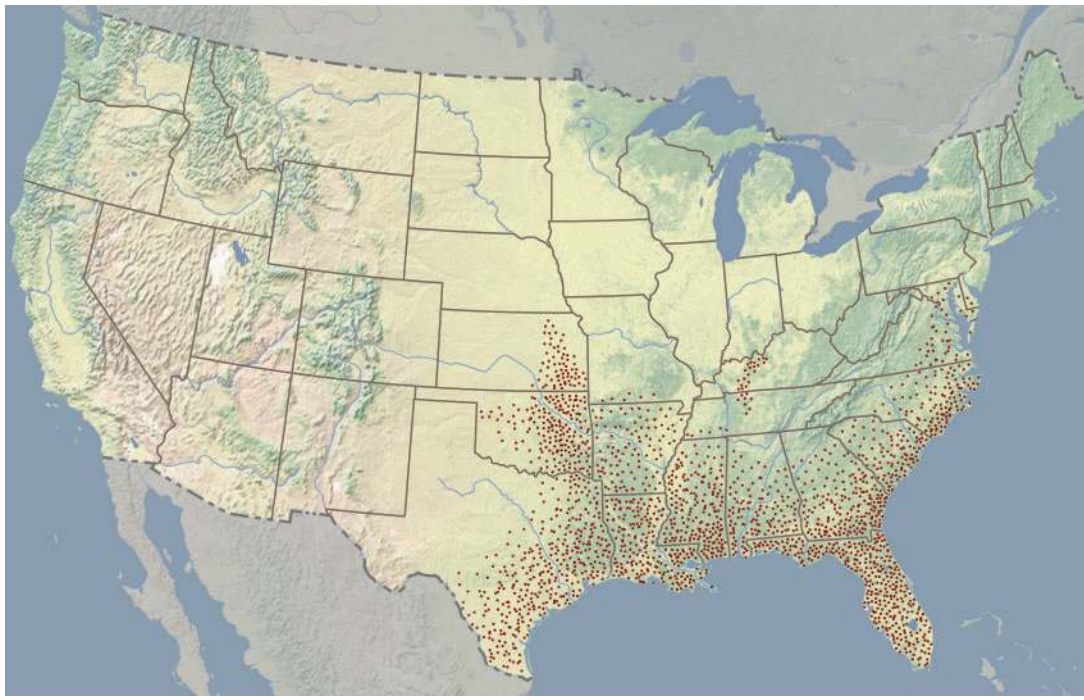

Fig. 3. An estimated distribution of *A. maculatum* in the United States for 2014 that interpolates contemporary data (Wilson and Baker 1972, Goddard and Norment 1983, Harrison et al. 1997, Clark et al. 1998, Williams et al. 1999, Reeves et al. 2002, Barker et al. 2004, Goddard and Paddock 2005, Cohen et al. 2010, Teel et al. 2010, Trout et al. 2010, Brown et al. 2011, Fornadel et al. 2011, Varela-Stokes et al. 2011, Wright et al. 2011, Pagac et al. 2014, and Florin et al. 2014). The range is shown as dark red stippling against a physical geographical base map. Loose stippling along borders represent areas where distribution and abundance may be expected to vary annually. The colors of the relief topographic map depict modern landcover conditions (naturalearthdata.com). Map courtesy of R. Ryan Lash, Centers for Disease Control and Prevention, Atlanta, GA.

(http://swine.vet.uga.edu/nfsms), including some that roughly approximate recently identified range extensions of *A. maculatum*.

White-tailed deer populations have undergone near-exponential growth in multiple regions of the eastern United States. At the end of the 19th century, following several decades of overhunting and habitat loss, fewer than 500,000 deer existed in the United States. Intensive conservation efforts, coupled with expansive environmental changes that inadvertently provided ideal habitats for deer to proliferate and increase in number to approximately 18 million by 1992 (Paddock and Yabsley, 2007). Gulf Coast ticks have been collected from white-tailed deer in at least 12 states in the central and southeastern United States (Samuel and Trainer 1970, Kellogg et al. 1971, Strickland et al. 1981, Bloemer et al. 1988, Lavender and Oliver 1996, Trout and Steelman 2010), and surveys of hunter-killed deer from Arkansas, Florida, Oklahoma, and Texas document infestations with *A. maculatum* that range from 18 to 100% (Samuel and Trainer 1970, Allan et al. 2001, Barker et al. 2004, Trout and Steelman 2010). Some data suggest that infestation rates of white-tailed deer have increased in several regions of the United States where Gulf Coast ticks have recently established. As recently as 1973, there were no records of *A. maculatum* collected from white-tailed deer in Arkansas (Lancaster 1973); nonetheless, by 2008, nymphal and adult-stage Gulf Coast ticks were collected from 20% of 250 hunter-killed white-tailed deer sampled from 19 counties throughout the state (Trout et al. 2010a,b).

Migrating birds are capable of transporting ectoparasites and their associated pathogens great distances and are likely involved in the spread of several vector-borne diseases to new regions (Rand et al. 1998, Hubalek 2004, Scott et al. 2012, Mukherjee et al. 2013, Hasle 2013). Because grassland birds are considered particularly important hosts for the maintenance and dispersal of immature stages of *A. maculatum*, it is likely that these species carry Gulf Coast ticks and their bacterial associates to new areas (Florin et al. 2014). The Eastern meadowlark (*Sturnella magna*), a short-distance migrant (<2,500 km) of the central flyway, is believed responsible in part, for the colonization and stability of Gulf Coast tick populations in Kansas and Oklahoma (Ketchum et al. 2009). Immature *A. maculatum* ticks were collected from 13 species of migratory songbirds during netting surveillance activities in Maryland during 2008–2010, and it is conceivable that this process may establish new populations of the Gulf Coast tick in some mid-Atlantic states (Florin et al. 2014).

Tremendous shifts in wildfire management practices have occurred throughout the United States during the past century that possibly represent important and

intriguing links to avian-dependant distributional changes of *A. maculatum*. The Coastal Plains are identified consistently as the perennial range of the Gulf Coast tick. Historically, this region was represented by remarkably fire-resistant and fire-adjusted pine flatwood forests and grasslands that were maintained by frequent, low-intensity surface fires during the growing season, ignited primarily by lightning strikes at 1- to 3-yr intervals (Komarek 1968, Frost 1998). High-frequency, low-intensity fires create and preserve specific habitat requirements of many disturbance-dependant birds, including grassland- and savanna-affiliated species that serve as important hosts of immature *A. maculatum* ticks (Shriver et al. 1996, Madden et al. 1999, Hunter et al. 2001, Tucker et al. 2004, Thatcher et al. 2006). From the 1920s until the late 1980s, aggressive fire exclusion and suppression policies existed in the United States that disrupted natural fire regimes and inadvertently diminished suitable habitats for many species of birds, as grasslands, savannas, and woodlands reverted to closed-canopy forests (Hunter et al. 2001, Nowacki and Abrams 2008).

The restoration of habitats suitable for grassland birds through prescribed burning has been increasingly applied throughout eastern North America (Madden et al. 1999, Wilcox and Giuliano 2011). Prescribed fire regimes may also favor populations of Gulf Coast ticks, which reestablish relatively rapidly in burned areas (Scrifres et al. 1988, Gleim et al. 2013). Observations from Ft. Campbell, a 105,068 acre U.S. Army installation located on the border between southwestern Kentucky and north-central Tennessee provides a salient example where anthropogenic influences have potentially affected the distribution of *A. maculatum* and *R. parkeri*. At Ft. Campbell, thousands of prescribed burns, including many that involve hundreds of acres, are conducted annually to facilitate visibility and maneuverability of troops involved in field training excercises and to reduce underbrush that might otherwise accumulate to create uncontrolled wildfires. Another consequence of these activities has been the restoration of breeding habitats for certain grassland birds, including Henslow's sparrows (Hunter et al. 2001). Beginning in 2000, tick attachment surveys identified specimens of *A. maculatum* from personnel at Fort Campbell, hundreds of miles north the traditionally recognized range limit of the Gulf Coast tick (Jiang et al. 2012, E. Y. Stromdahl, unpublished data). Subsequent studies have verified that established populations of Gulf Coast ticks exist at Ft. Campbell, of which an estimated 15% are infected with *R. parkeri* (Pagac et al. 2014).

Medical Importance

Nuisance. During the early 1900s, *A. maculatum* was recognized as one of the most aggressive human-biting ticks in the United States (Hooker et al. 1912). Contemporary tick bite surveys consistently identify the Gulf Coast tick among the top four ixodid species parasitizing humans in the southern United States, typically following distantly in biting frequency to *A. americanum* and *D. variabilis* (Merten and Durden 2000; Table 1). Nonetheless, some data indicate that Gulf Coast ticks bite humans with a greater frequency than previously believed. For example, *A. maculatum* represented approximately 12% of the total number of ticks removed from humans and sent for identification to the Mississippi Department of Health during 1990–1999, second in frequency only to *A. americanum* (Goddard 2002). Similar results were obtained from a tick attachment project in North Carolina during 2008 through 2012, where Gulf Coast ticks represented approximately 15% of ticks found crawling on or embedded in humans. (M. Toliver, unpublished data).

Although Gulf Coast ticks generally comprise only 1–3% of reported tick attachments, it is also possible that some specimens are misidentified occasionally as *D. variabilis*, a sympatrically distributed tick that closely resembles *A. maculatum* on casual inspection (Fig. 4). If the mouthparts are missing, which may occur when the tick is removed from the tick bite victim, the morphological differences between adult females of these species are even more subtle to an untrained observer (Stromdahl and Hickling 2012). The distinctions between the immature stages of these two species are also difficult to discern, resulting in occasional misidentification of larvae or nymphs (Lancaster 1973, Bishopp and Trembley 1945).

Tick Paralysis. Paralysis of humans following attachment by adult *A. maculatum* has been reported at least twice. As with most other tick species responsible for paralysis, not all *A. macualtum* ticks will necessarily produce this condition while feeding. The initial description involved a 7-yr-old girl from northern Louisiana who presented with generalized, symmetrical flaccid paresis, weakness, and lethargy. The child was afebrile, but physical examination revealed ataxia, suppression of deep and superficial reflexes, and adiadokokinesia. A partially engorged female *A. maculatum* tick was found attached to her scalp. When the tick was removed her neurologic symptoms improved rapidly and all abnormal findings resolved within 48 hr (Paffenbarger 1951). The other published report described a 22-yr-old man from Jalisco, Mexico, who presented with flaccid paraplegia and arreflexia. Within 48 hr after two female *A. maculatum* ticks detached from the patient, he showed spontaneous and progressive recovery of all neurologic deficits (Espinoza-Gomez et al. 2011).

***Rickettsia parkeri* Rickettsiosis.** Also known as American boutonneuse fever (Goddard 2004, Goddard and Varela-Stokes 2009) and Tidewater spotted fever (Wright et al. 2011), confirmed disease caused by *R. parkeri* in a human patient was first reported in 2004 (Paddock et al. 2004). Unrecognized infections undoubtedly occurred in humans for many decades before the index case, suggested by descriptions of nonfatal cases of RMSF associated with inoculation ulcers, regional adenopathy, and a sparse rash from coastal areas of Viriginia and Maryland during the 1920s and 1930s (Spencer 1926, Shipley 1932). More recently, cases of a relatively mild, eschar-associated, spotted fever group rickettsoisis were described in patients from South and North Carolina, several years prior to

Table 1. Percentage of *A. maculatum* ticks submitted for identification in surveys of tick attachments to humans in the United States, 1974 through 2012

State(s)	Year(s) of survey	Total no. of submitted ticks	No. (%) represented by *A. maculatum*	Reference(s)
South Carolina	1974–1975	15, 660	110 (0.7)	Loving et al. 1978
Georgia and South Carolina	1990–1998	1,270	15 (1.2)	Felz et al. 1996, Felz and Durden 1999
Mississippi	1990–1999	119	14 (11.8)	Goddard 2002
Texas	2004–2008	903	10 (1.1)	Williamson et al. 2010
Georgia	2005–2006	603	19 (3.2)	E. R. Gleim, unpublished data
North Carolina	2008–2012	746	110 (14.7)	M. Toliver, unpublished data

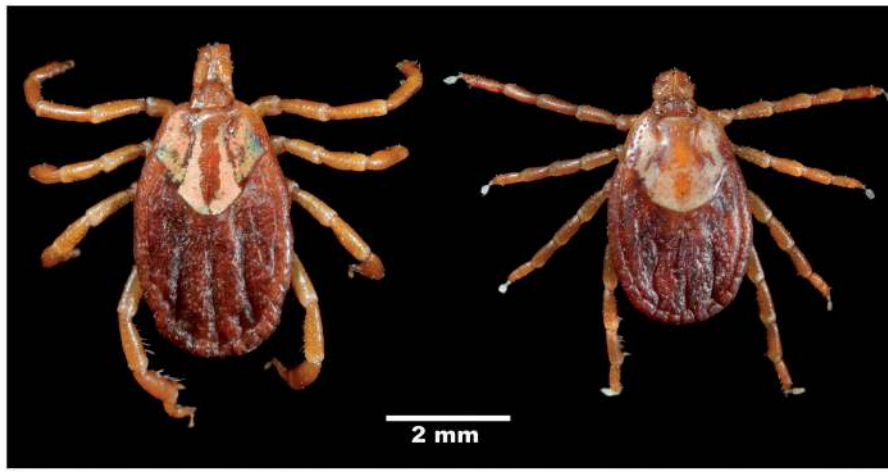

Fig. 4. Morphological similarities between adult female specimens of *A. maculatum* (left) and *D. variabilis* (right).

the first documented case of *R. parkeri* rickettsiosis in 2004 (Cox and Sexton 1995, Krusell et al. 2002).

Through 2014, at least 37 case patients from 9 U. S. states have been identified (Paddock et al. 2008, Cragun et al. 2010, Meyers et al. 2013, Ekenna et al. 2014, Kaskas et al. 2014, CDC unpublished data; Fig. 5). Cases of *R. parkeri* rickettsiosis have also been confirmed from Argentina and Uruguay where *A. triste* and *A. tigrinum* serve as vectors (Romer et al. 2011, 2014; Portillo et al. 2013). In the United States, cases of disease have been identified during April through October with approximately 80% of the recognized illnesses occurring during July through September. Confirmed infections have resulted from bites of nymphal and adult-stage Gulf Coast ticks (Whitman et al. 2007, Paddock et al. 2008), but the preponderance of mid-to-late summer occurrences of this disease, when coupled with the phenology of *A. maculatum* in the southeastern United States (Hixson 1940, Cilek and Olson 2000, Teel et al. 2010, Gleim et al. 2013), suggest that most cases *R. parkeri* rickettsiosis result from bites of adult rather than immature ticks. The period of attachment required to transmit the infection has not been determined conclusively, although one patient developed disease after an attachment period of <8 h (Whitman et al. 2007). Approximately 75% of the known case patients are men. In contrast to RMSF, there are no published descriptions of *R. parkeri* rickettsiosis in young children; indeed, most patients with this infection are considerably older and the median age from case reports is 53 years (range, 23 to 83 yr).

A necrotic inoculation eschar develops in almost all patients, typically 2 to 10 d following the bite of an infected tick. The lesions are generally nontender, or mildy tender ulcers, approximately 0.5 to 2 cm in width, that are often surrounded by an erythematous halo and a few scattered petechiae (Fig. 6A and B). Fever (generally 38°C to 40°C) develops within a few days after the formation of the eschar. The exanthem follows within a few days of the onset of fever, and consists of 15 to >100 maculopapular or vesiculopapular lesions that range from 0.2 to 0.5 cm and primarily involve the trunk and extremities (Fig. 6C–E). Most patients have headaches and generalized body aches. Regional lymphadenopathy has been documented in fewer than 30% of cases reported from Argentina and the United States, but for reasons currently unknown, this manifestation is reported with much greater frequency among *R. parkeri*-infected patients in Uruguay (Paddock et al. 2008, Conti-Díaz et al. 2009, Romer et al. 2014). Infected patients respond within 24 to 72 h following treatment with doxycycline and typically recover with no residual sequelae (Whitman et al. 2007, Paddock et al. 2008, Cragun et al. 2010, Meyers et al. 2013, Ekenna et al. 2014; Kaskas et al. 2014).

Fig. 5. Case-patient locations of *R. parkeri* rickettsiosis in the United States, 2002–2014. States where *R. parkeri* has been identified in adult Gulf Coast ticks are shaded gray. Data from Paddock et al. 2004, Whitman et al. 2007, Paddock et al. 2008, Cragun et al. 2010, Meyers et al. 2013, Ekenna et al. 2014, Kaskas et al. 2014, and CDC unpublished data.

Clinical differences between *R. parkeri* rickettsiosis and RMSF include the inoculation eschar, a vesicular rash, and severity of disease (Table 2). *R. parkeri* rickettsiosis is a considerably milder rickettsiosis than RMSF. Less than a third of patients have been hospitalized, and there have been no reported severe manifestations or deaths. Despite these differences, it is likely that some U.S. cases previously categorized as RMSF represented *R. parkeri* rickettsiosis (Raoult and Paddock 2005, Paddock 2005, Vaughn et al. 2014). Accordingly, the reporting category for RMSF in the United States was modified in 2010 to include diseases caused by *R. parkeri* and other spotted fever group *Rickettsia* species (Council of State and Territorial Epidemiologists 2009, Openshaw et al. 2010).

R. parkeri has been detected in Gulf Coast ticks from Alabama, Delaware, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, Tennessee, Texas, and Virginia, approximating the distribution of human cases (Fig. 5). Current estimates of infection rates of questing adult ticks range from 8 to 56%, remarkably higher than the infection prevalence of any other recognized tick-borne rickettsial pathogen in the United States (Table 3). *R. parkeri* has been detected in nymphal and adult stages of *A. maculatum*, and is distributed throughout multiple tissues, including salivary glands, midgut, Malpighian tubules, and ovaries, and in the saliva, of infected ticks (Edwards et al. 2011, Budachetri et al. 2014). *R. parkeri* has been identified infrequently in specimens of other important human-biting ticks in the United States, including 0.4–1.0% of adult *A. americanum* ticks collected in Georgia, Kentucky, Mississippi, Tennessee, and Virginia (Goddard and Norment 1986, Cohen et al. 2009, Gaines et al. 2014) and in 0.3–2.3% of *D. variabilis* collected in Kentucky, Texas, and Virginia (Williamson et al. 2010, Fritzen et al. 2011, Fornadel et al. 2011, Henning et al. 2014), as well as some rare reports from *Ixodes scapularis* Say, *Rh. sanguineus*, and *Haemaphysalis leporispalustris* (Packard) (Leydet and Liang 2013, Henning et al. 2014). *R. parkeri* has been detected in *A. maculatum* ticks in Peru (Flores-Mendoza et al. 2013), as well as in *A. triste* ticks from Argentina, Brazil, and Uruguay, and in *A. tigrinum* ticks from Argentina, Bolivia, and Uruguay, reflecting a close association between this *Rickettsia* species and ticks of the *A. maculatum* species complex (Venzal et al. 2004, 2012; Pacheco et al. 2006; Silveira et al. 2007; Nava et al. 2008a,b; Tomassone et al. 2010; Cicuttin and Nava 2012; Romer et al. 2014).

The natural history of *R. parkeri* is incompletely known. Transovarial and transstadial transmission of the agent have been reported in *A. americanum* and *A. triste* ticks (Goddard 2003, Nieri-Bastros et al. 2013), although neither process has been demonstrated conclusively in Gulf Coast ticks. The effect of *R. parkeri* on the fitness of its arthropod host has been examined in *A. triste*, where the molting success of infected nymphal-stage ticks was 66%, compared with 92% for noninfected nymphs, suggesting that *R. parkeri* has a

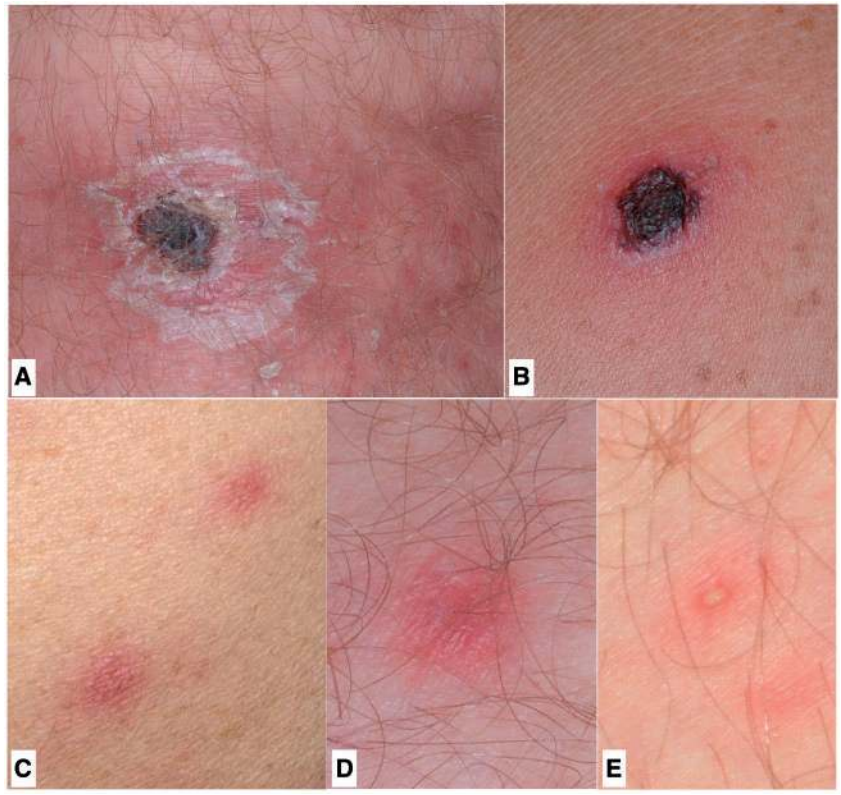

Fig. 6. Clinical appearance of cutaneous lesions associated with *R. parkeri* rickettsiosis in humans, including inoculation eschars (A and B) and maculopapular, vesicular, or pustular exanthems (C through E).

Table 2. Comparison of the clinical characteristics of *R. parkeri* rickettsiosis and Rocky Mountain spotted fever (RMSF) in the United States

Characteristic	*R. parkeri* rickettsiosis[a] ($n = 21$)	RMSF[b] ($n = 398$)
	Percentage of patients with characteristic[c]	
Fever	100	99
Inoculation eschar	95	NR[d]
Any rash	90	92
Any macules or papules	86	53
Any vesicles or pustules	33	NR
Any petechiae	14	52
Headache	86	80
Myalgia	76	60
Nausea or vomiting	10	66
Diarrhea	0	25
Coma, seizures, delirium, or confusion	0	27
Death	0	8

[a] Data from Paddock et al. 2004, Whitman et al. 2007, Paddock et al. 2008, Cragun et al. 2010, Meyers et al. 2013, and Ekenna et al. 2014.

[b] Data from Hazard et al. 1969, Kaplowitz et al. 1981, Kirk et al. 1990, Conlon et al. 1996, and Buckingham et al. 2007.

[c] Percentages determined from the number of patients for whom the clinical characteristic was specifically evaluated.

[d] NR, not reported in the clinical description of any case series.

moderately deleterious effect on the survival of *A. triste* and that amplifying hosts may be needed for the perpetuation of this *Rickettsia* species in nature (Nieri-Bastos et al. 2013). In areas of the United States endemic for *R. parkeri*, northern bobwhite quail, cotton rats, and cattle demonstrate high levels of antibody reactive with spotted fever group rickettsiae (Edwards et al. 2010; Moraru et al. 2013a,b); nonetheless, *R. parkeri* has never been isolated in culture, or detected by molecular methods from any of these species in nature, or in any other animal other than domestic dogs (Tomassone et al. 2010, Grasperge et al. 2012).

In laboratory experiments, *R. parkeri* has been detected in the skin, blood, or spleen of experimentally infected cotton rats as long as 7 d following subcutaneous inoculation with infected Vero cells. The infections were typically cleared rapidly, and immature *A. maculatum* ticks were unable to acquire *R. parkeri* while feeding from infected animals (Moraru et al. 2013a,b). Preliminary evidence indicates that replication of *R. parkeri* within the skin of experimentally infected C3H/HeJ mice is greatly enhanced by the feeding process of *A. maculatum* ticks, to suggest that salivary components released by the tick into the feeding site alter the cutaneous microenvironment and facilitate infection of the vertebrate host by *R. parkeri* (Grasperge et al. 2014).

Table 3. Prevalence rates of *R. parkeri* infection in questing adult Gulf Coast ticks, by state and county, in the United States, 1999–2014

Location	Year	No. of ticks examined	Percent infected (95% CI)	Reference
Florida				
Franklin Co.	2004	25	12 (0–25)	Sumner et al. 2007
	2005	37	8 (0–17)	Paddock et al. 2010
	2007	92	26 (17–35)	Paddock et al. 2010
Georgia				
Bulloch Co.	1999	20	10 (0–23)	Sumner et al. 2007
	2003	16	31 (8–54)	
	2005	24	12 (0–25)	
Mississippi				
Jackson Co.	2007	62	40 (28–52)	Paddock et al. 2010
	2011–2012	83	33 (22–43)	Budachetri et al. 2014
Multiple counties[a]	2008–2009	698	15 (12–18)	Ferrari et al. 2012
North Carolina				
Wake Co.	2010	34	56 (39–73)	Varela-Stokes et al. 2011
Mecklenburg Co.	2010	67	18 (9–27)	
Virginia				
Chesapeake Co.	2010	49	47 (33–61)	Wright et al. 2011
Fairfax Co.	2010	211	37 (30–44)	Fornadel et al. 2011
Multiple counties[b]	2011	97	56 (46–66)	Nadolny et al. 2014
	2012	131	55 (47–63)	
Kentucky				
Muhlenberg Co.	2012	36	14 (3–25)	Pagac et al. 2014
Delaware				
Kent Co.	2012–2013	26	8 (0–18)	Florin et al. 2013, 2014

[a] Clay, Harrison, Hinds, Jackson, Oktibbeha, and Webster.
[b] Including some or all of the following cities and counties: Middlesex, Northhampton, James City, York, Isle of Wight, Sufolk, Portsmouth, Norfolk, Chesapeake, and Viginia Beach.

Natural Infections With Other Recognized Pathogens. Other pathogens have been identified sporadically in adult Gulf Coast ticks, including *Ehrlichia chaffeensis* (the etiologic agent of human monocytic ehrlichiosis) from Florida, Kansas, and Texas (Williamson et al. 2010, R. Ganta unpublished data, J. Sumner and C.D.P. unpublished data), and *Rickettsia felis* (the agent of cat flea rickettsiosis) from Virginia and Mississippi (Jiang et al. 2012). The role of *A. maculatum* in the epidemiology of these diseases is presently unknown.

Veterinary Importance

Annoyance and Economic Impact. Extensive parasitism of cattle by Gulf Coast ticks causes measurable weight and blood loss (Gladney et al. 1977, Stacey et al. 1978, Williams et al. 1978). One study showed that tick-free Hereford cattle gained almost 27 kg more weight than cattle infested with *A. maculatum* during an 8-week period (Stacey et al. 1978). Alterations in blood chemistry and hematology, including leukocytosis and elevated serum creatine kinase, occur among cattle exposed to Gulf Coast ticks, particularly during the first week of infestation (Riley et al. 1995). Dogs are often parasitized by Gulf Coast ticks in areas where *A. maculatum* occurs in United States. Gulf Coast ticks were second in frequency to *A. americanum* among ticks collected from dogs that were walked along transects of a wooded area of north-central Oklahoma (Barrett et al. 2014) and represented 22% of the total number of ticks collected from dogs receiving care at veterinary clinics or housed at animal shelters in Orangeburg County, SC, during 1994, second in frequency only to *D. variabilis* (Clark et al. 1996). Adult and nymphal *A. maculatum* represented 8.1% of ticks infesting family-owned dogs in Bulloch County, GA, during 1996–2003, and 13% of the ticks were recovered from animal shelter dogs in Emanuel County, GA, during 2002–2003 (Wells et al. 2004).

Gotch Ear. Adult Gulf Coast ticks are among the largest hard tick species indigenous to United States, and possess relatively long and stout mouthparts. When multiple adult *A. maculatum* ticks attach to the inner and outer aspects of the external ear of livestock, the skin swells and cracks, and soon becomes covered by a thick crust of serum, blood, and tick feces. The annoyance and irritation causes animals to rub against trees, posts, and other objects, often causing further injury to their ears (Gladney et al. 1977). In some cases, extensive inflammation and edema leads to destruction of the supporting cartilage, and the ear becomes drooped, thickened, curled, or cupped, resulting in the condition known as "gotch ear" (Fig. 7A and B). The etymology of the term is not clear; nonetheless the earliest known reference to gotch ear describes how extensive feeding by ticks on the ears of horses in south Texas resulted in the ears to droop (Adams 1905). In some localities, the term "gotch tick" was applied to *A. maculatum* because of its frequent association with this condition (Bishopp and Hixson 1936).

Gotch ear is described most frequently for cattle, although horses, mules, sheep, and goats (Fig. 7C and D) may also develop this condition, and affected livestock bring lower prices at auction (Bishopp and

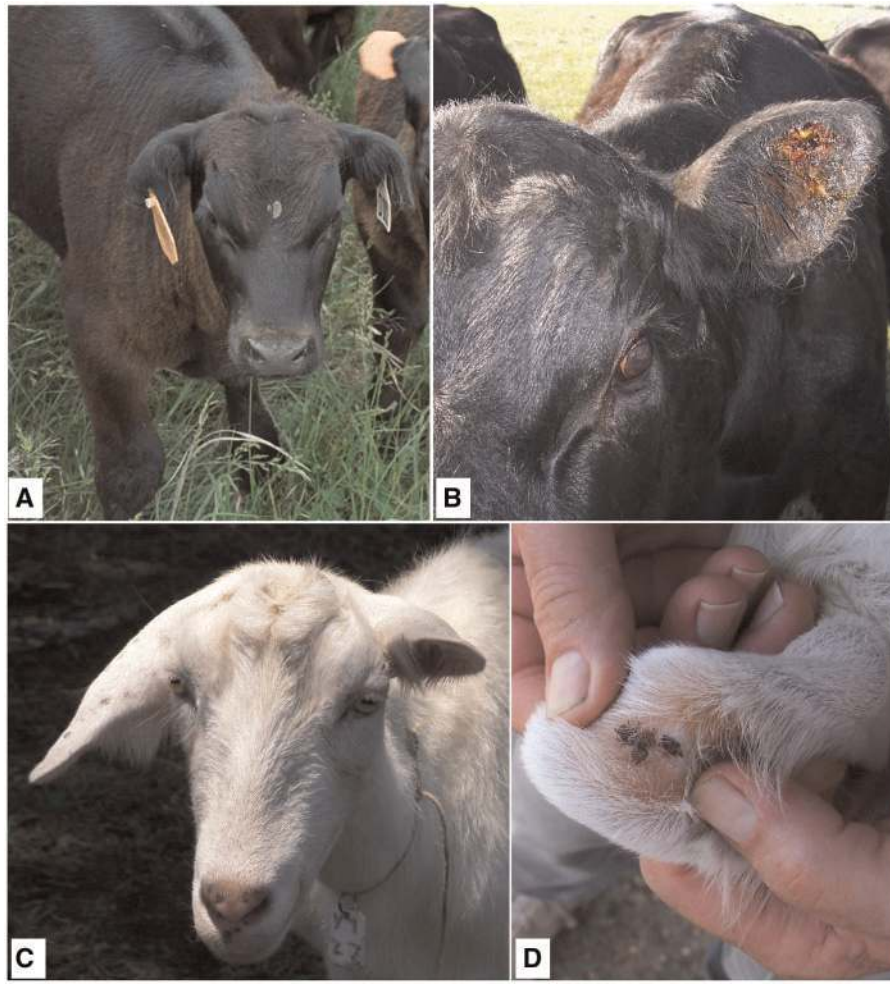

Fig. 7. Gotch ear in cattle (A, B) and a goat (C, D), showing the drooped and stiffened appearance that results from attachment and feeding by multiple adult *A. maculatum* to the inner surface of the ear (D). The irriation caused by attached *A. maculatum* ticks may result in generalized edema, inflammation, and proteinaceous exudates (C) and eventually lead to destruction of the supporting cartilage. Images courtesy of Michael Dryden, D.V.M., Ph.D. (Kansas State University, Manhattan, KS), and Kristine T. Edwards, D.V.M., Ph.D. (Mississippi State University, Starkville, MS).

Trembley 1945, Drummond and Whetstone 1970, Gladney 1976, Ivy et al. 1978, Williams et al. 1978, Byford et al. 1992, Mullens 2003, Edwards 2011). The pathogenesis of gotch ear is unknown, although it occurs infrequently when fewer than 10 adult ticks are attached to an ear, to suggest that the tissue reaction results from cumulative responses to salivary proteins secreted by Gulf Coast ticks during the feeding process (Edwards et al. 2011).

Hepatozoon americanum. American canine hepatozoonosis (ACH) is a severe and potentially fatal infection of dogs caused by the apicomplexan parasite *H. americanum* (Ewing and Panciera 2003, Potter and Macintire 2010, Allen et al. 2011). In 1978, investigators in Texas described naturally occurring hepatozonoosis in canids for the first time in the Western Hemisphere, first from a coyote (Davis et al. 1978), and subsequently from dogs (Craig et al. 1978, Barton et al. 1985). Additional reports of this protozoan infection from the central and southeastern United States prompted a search for a tick vector. In 1998, investigators described an infection cycle of a novel disease agent, *H. americanum*, involving acquisition and subsequent transmission of the parasite by *A. maculatum* (Mathew et al. 1998). Although dogs are believed to be accidental hosts for *H. americanum*, autochthonous cases of ACH have now been documented from Alabama, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Oklahoma, Tennessee, Texas, and Virginia (Allen et al. 2008, Li et al. 2008). Most infections are recognized during June through October (Macintire et al. 1997).

The life cycle of *H. americanum* involves multiple stages within invertebrate and vertebrate hosts. *A. maculatum* is the only known definitive host (Ewing et al. 2002a). Gamonts of *H. americanum* are acquired by

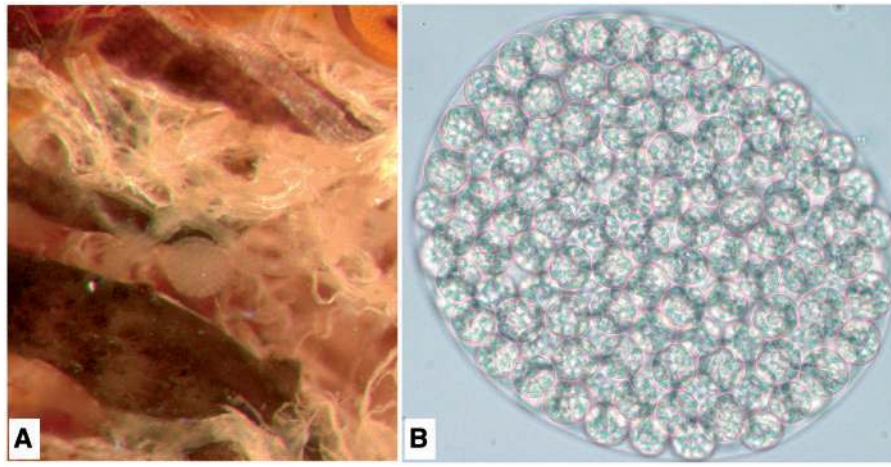

Fig. 8. Mature oocysts of *Hepatozoon americanum* in the hemocoel of an infected *A. maculatum* tick (A). Wet mount of an individual oocyst containing numerous sporocyts (B). Images courtesy of Eileen Johnson, D.V.M., Ph.D. (Oklahoma State University, Stillwater, OK).

larval and nymphal stage *A. maculatum* ticks with the bloodmeal (Mathew et al. 1998; Ewing et al. 2002a,b). Once inside the tick, ingested gamonts invade gut epithelial cells, become enveloped in a parasitophorous vacuole, and subsequently undergo gametogenesis and fertilization. The parasites are passaged transtadially within the ixodid host, and sporogonic development requires approximately 40 d. Mature oocysts are eventually released into the hemocoel of the tick (Fig. 8A). Experimentally infected ticks contain 10 to >500 oocysts; each oocyst contains >200 sporocysts (Fig. 8B), and each sporocyst contains 10–26 sporozoites (Mathew et al. 1999). Few data exist regarding the frequency of *H. americanum* in populations of *A. maculatum*, and naturally infected ticks have been described infrequently (Vincent-Johnson et al.1997).

Infection of vertebrate intermediate hosts occurs when newly molted nymphs or adults containing oocysts are ingested during grooming or by consuming an infected tick attached to prey (Mathew et al. 1998, Kocan et al. 2000, Garrett et al. 2005). Nonetheless, neither dogs nor wild carnivores are considered preferred hosts for immature stages of *A. maculatum*, so that it is more likely that predation of *H. americanum*-infected animals is the primary route of acquisition (Johnson et al. 2009). Experimental studies also reveal that cotton rats, house mice (*Mus musculus*), and rabbits (*Oryctolagus cuniculus*) are effective paratenic hosts for *H. americanum* (Johnson et al. 2008b). A distinct tissue stage of *H. americanum*, known as the cystozoite, develops in cardiac muscle, skeletal muscle, kidneys, lungs, and spleen of paratenic hosts approximately 1 mo after the animal ingests sporulated oocyts of *H. americanum*. Cystozoites reside singly within cytoplasmic vacuoles of macrophages and appear as crescentic or comma-shaped parasites with a densely basophilic nucleus that measure approximately 2 by 12 μm (Johnson et al. 2008a). Dogs that consume animals containing cystozoites of *H. americanum* subsequently develop ACH (Johnson et al. 2008b).

Dogs that ingest oocysts or cystozoites of *H. americanum* develop clinical signs within 4 to 5 wk. Sporozoites penetrate the gut mucosa and are eventually disseminated to various organs and tissues, although the primary target is striated muscle. Salient features of ACH include fever (39° to 41°C), lethargy, mucopurulent ocular discharge, pain, altered gait, and muscle atrophy, especially of the head. Laboratory findings demonstrate a persistent and predominantly neutrophilic leukocytosis that may range from $20–200 \times 10^9$ cells per liter. Other findings include a mild normochromic, normocytic anemia, hypoglycemia, hypoalbumineamia, and elevated levels of serum alkaline phosphatase (Barton et al. 1985, MacIntire et al. 1997, Potter and Macintire 2010). Radiographs often reveal disseminated proliferation of the periosteum, particularly around the diaphysis of the long bones of the limbs. These lesions range from slightly elevated plaques of porous bone to concentrically thickened regions that are generally most severe on the femur and humerus (Fig. 9A and B). The pathogenesis of these unusual lesions is not fully understood (Panciera et al. 2000). Infection may persist in some dogs >5 yr; however, most dogs with ACH die within 1 to 2 yr without supportive therapy (Allen et al. 2011, Ewing et al. 2003). There are no antimicrobials that cure the infection in dogs, although combination treatment with trimethoprim-sulfadiazine, clindamycin, and pyrimethamine followed by decoquinate diminishes clinical disease (Potter and Macintire 2010).

Three distinct stages occur in infected dogs (Cummings et al. 2005). The most frequently identified forms are lamellated mucopolysaccharide cysts, approximately 80 μm to >300 μm in width, that surround a centrally located macrophage containing the parasite as it undergoes merogony (Fig. 9C). These "onion skin"

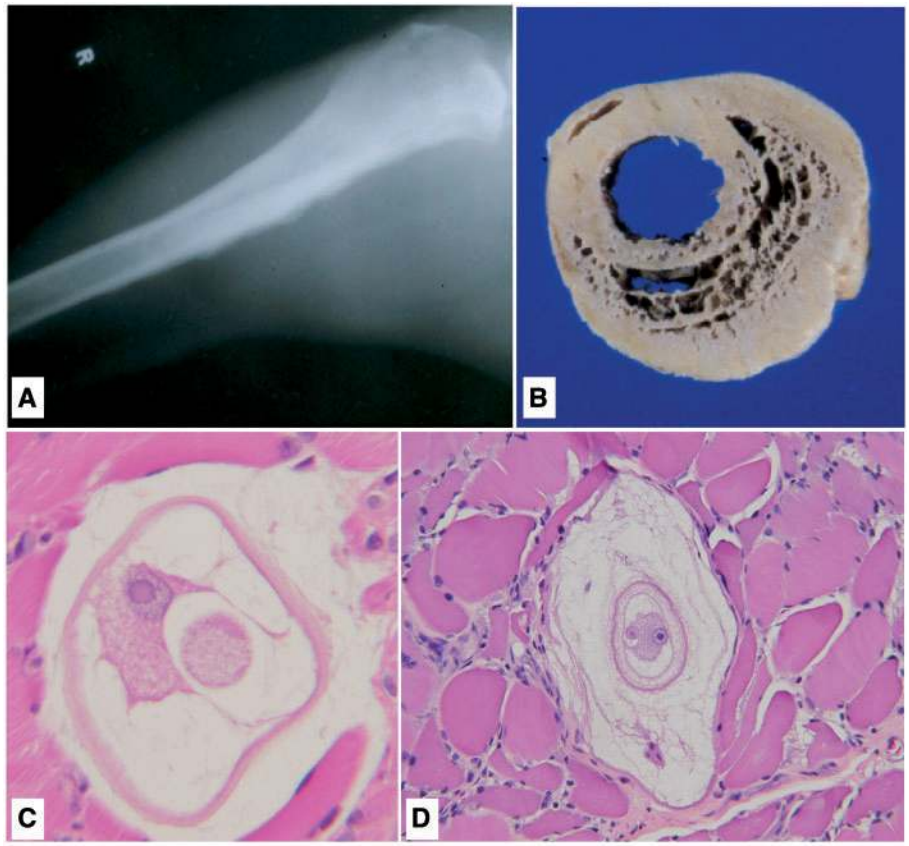

Fig. 9. Clinical and pathological manifestations of canine infection with *Hepatozoon americanum*. Radiograph showing periosteal proliferation involving the diaphysis of a femur in an infected dog (A). Cross section of a long bone from an infected dog showing concentric thickening of periosteum (B). *H. americanum* cysts in the skeletal muscle of an infected dog (C and D). The parasite is contained within a macrophage (C) and becomes surrounded by a lamellated mucoplysaccharide cyst (D). Inflammatory infiltrates are absent until merogony is completed and the cyst ruptures to release merozoites. Images courtesy of Roger Panciera, D.V.M., Ph.D. (Oklahoma State University, Stillwater, OK).

cysts are most often situated between skeletal muscle fibers (Fig. 9D), less frequently in the heart, and only rarely in other organs and tissues, including pancreas, spleen, liver, lung, mesentery, salivary glands, stomach, intestines, and lymph nodes (Panciera et al. 1998). There is minimal to no host inflammatory response associated with the cyst stage; however, when the parasite completes merogony, the cyst wall disintegrates and releases large numbers of merozoites that initiate a pyogranulomatous inflammatory cell response. Eventually the infiltrates are predominated by macrophages that contain ovoid, $3.5 \times 5.5\,\mu m^2$ parasites that undergo gametogeny. Gamonts, the third and final stage of *H. americanum* identified in dogs, are elongate parasites that measure 8 to 10 μm and are found within the cytoplasm of circulating monocytes. Canine parasitemias are characteristically quite low, and <0.1% of peripheral blood leukocytes contain gamonts (Vincent-Johnson et al. 1997). Accordingly, an examination of several thousand leukocytes is often required to identify a gamont in blood smears of naturally infected dogs (Barton et al. 1985).

Naturally occurring infections with *H. americanum* have been described in coyotes in Oklahoma and Texas (Mercer et al. 1988, Starkey et al. 2013). Skeletal and cardiac muscle lesions caused by *H. americanum* were documented in approximately half of free-ranging coyotes surveyed for this infection in central Oklahoma during 1998–1999 (Kocan et al. 1999, 2000); nonetheless, the prevalence of disease in nature in unknown and it is likely that many other wild carnivores, including ocelots (*Felis pardalis*) and bobcats (*Lynx rufus*) also serve as intermediate hosts for *H. americanum* (Mercer et al. 1988).

Rickettsia parkeri. Gulf Coast ticks infected with *R. parkeri* have been collected from cattle, sheep, and goats (Philip and White 1955, Edwards et al. 2010), and from various species of wildlife, including black bears (*Ursus americanus luteolus*), feral pigs, white-tailed deer, cotton rats, and coyotes (Sumner et al. 2007, Yabsley et al. 2009, Trout and Steelman 2010, Fornadel et al. 2011, Leydet and Liang 2013); nonetheless, no data describe clinical disease in animals. Calves exposed to *R. parkeri*-infected Gulf Coast ticks, or

inoculated subcutaneously, intradermally, or intravenously with *R. parkeri*-infected cell culture, remained afebrile and alert despite occasional and transient rickettsemias (Edwards et al. 2010). Canine infection with *R. parkeri* has been documented from the blood of a dog infested with *R. parkeri*-infected *A. tigrinum* ticks in Cercado province in Bolivia (Tomasonne et al. 2010) and in blood specimens sampled from 13% of 93 asymptomatic dogs housed at animal shelters in five parishes in southern Louisiana (Grasperge et al. 2012).

Experimental Infections with Other Pathogens. *Ehrlichia ruminantium* causes a systemic and often fatal disease known as heartwater in wild and domesticated ruminants across sub-Saharan Africa and some islands in the Caribbean. The pathogen is transmitted by multiple African *Amblyomma* species. Laboratory investigations indicate that immature stages of Gulf Coast ticks can acquire *E. ruminantium* while feeding from infected animals and transmit the pathogen transstadially to subsequent life stages at a level of efficiency similar to *Amblyomma variegatum* (F.), a recognized vector of *E. ruminatium* in nature (Uilenberg et al. 1982). Infected *A. maculatum* ticks are also capable of transmitting multiple different strains of *E. ruminantium* to noninfected animals, and the prepatent periods and severity of subsequent disease are similar to those that occur when *A. variegatum* is used as a vector (Mahan et al. 2000; Table 4). Collectively, these data emphasize the importance of *A. maculatum* as a potential vector of *E. ruminantium* in the Western Hemisphere, particularly if the pathogen is introduced to the American continent by the importation of exotic animals from Africa or by tick-infested migratory birds from islands in the Caribbean (Uilenburg et al. 1982, Mahan et al. 2000).

A recently described *Ehrlichia* species, designated the Panola Mountain *Ehrlichia*, has been detected in specimens of adult Gulf Coast ticks collected in Florida, Georgia, and Texas (Loftis et al. 2010). Panola Mountain *Ehrlichia* also occurs naturally in *A. americanum* ticks and is transmissible to vertebrates (Loftis et al. 2008a,b;Yabsley et al. 2008). Similar to *E. chaffeensis*, Panola Mountain *Ehrlichia* circulates as asymptomatic infections in white-tailed deer, which are the likely vertebrate reservoir (Yabsley et al. 2008). However, it causes mild illness in goats, dogs, and possibly other species, and *A. maculatum* has been shown to be an efficient experimental vector of this agent (Loftis et al. 2008a, Qurollo et al. 2013).

Table 4. Acquistion of *E. ruminantium* (Elevage strain) from infected sheep and transmission to noninfected sheep by various *Amblyomma* spp. ticks (adapted from Mahan et al. 2000)

Amblyomma sp.	Percent infected[a] (n)	Transmission to sheep[b]	Incubation period (d)	Days to death
A. americanum	0.7 (139)	–	NA[c]	NA
A. cajennense	1.1 (272)	–	NA	NA
A. maculatum	50.7 (274)	+	17	22
A. variegatum	43.4 (290)	+	15	18

[a] Acquistion of *E. ruminatium* as evaluated by PCR of adult ticks infected as nymphs following attachment to infected sheep.
[b] No transmission (–) of *E. ruminantium* confirmed by susceptibility of sheep to lethal challenge; transmission confirmed (+) by positive brain smears and cultures.
[c] Not applicable.

Other Microbial Associates

"*Candidatus* Rickettsia andeanae" was first described from ixodid ticks collected in Peru, and subsequently in multiple tick species in Argentina, Brazil, and the United States (Blair et al. 2004, Sumner et al. 2007, Paddock et al. 2010, Abaraca et al. 2012, Flores-Mendoza et al. 2013, Nieri-Bastos et al. 2014). In the United States, "*Ca*. R. andeanae" has been detected in Gulf Coast ticks from Florida, Georgia, Louisiana, Kansas, Mississippi, North Carolina, Oklahoma, Tennessee, and Virginia (Wright et al. 2011, Varela-Stokes et al. 2011, Jiang et al. 2012, Fornadel et al. 2011, Ferrari et al. 2012, Leydet and Liang 2013, Budachetri et al. 2014). Infection rates vary considerably and appear to be related inversely to the prevalence of infection with *R. parkeri* in a given region. For example, approximately 1–5% of *A. maculatum* adults from Florida, Mississippi, North Carolina, and Virginia are infected with "*Ca*. R. andeanae," but in these same areas, it is not uncommon to find that >20% of ticks are infected with *R. parkeri*. In contrast, >50% of Gulf Coast ticks in Oklahoma and Kansas are infected with "*Ca*. R. andeanae," and infections with *R. parkeri* in adult Gulf Coast ticks have not been identified (Barrett et al. 2014, Paddock et al., unpublished data). Coinfections with both species have been identified occasionally in *A. maculaltum* ticks in the United States (Varela-Stokes et al. 2011, Ferrari et al. 2012, Leydet and Liang 2013).

It is unknown if "*Ca*. R. andeanae" causes disease in human or animal hosts. Molecular techniques failed to detect "*Ca.* R. andeanae" in salivary glands dissected from infected ticks (Budachetri et al. 2014), to suggest that this *Rickettsia* species is not transmissible to vertebrates; therefore, a report describing its detection in the blood of 13 species of tick-infested passerine songbirds captured in southern Louisiana requires further investigation (Mukherjee et al. 2013). Recent isolation of "*Ca.* R. andeanae" in cell culture should provide a more detailed analysis for species characterization and its capacity to elicit disease in vertebrate hosts (Luce-Fedrow et al. 2012, Ferrari et al. 2013a,b), as well as its interactions with *R. parker*i within Gulf Coast ticks.

Many other bacteria, including potential endosymbionts, have been identified recently in Gulf Coast ticks. "*Candidatus* Rickettsia amblyommii," a *Rickettsia* species of uncertain pathogenicity to humans or animals, has been detected occasionally in specimens of *A. maculatum* in Arkansas, Florida, Mississippi, and Virginia (Yabsley et al. 2009; Trout et al. 2010a,b; Fornadel et al. 2011; Budachetri et al. 2014). Other bacterial associates include a *Francisella* species, detected frequently in specimens of *A. maculatum* collected from Florida, Kentucky, Mississippi, and Oklahoma (Scoles 2004, Rounds et al. 2012, Williams-Newkirk et al. 2014, Budachetri et al. 2014), *Midichloria*

mitochondrii (Williams-Newkirk et al. 2014), and multiple species of Enterobacteriaceae from the genera *Raoultella*, *Ewingella*, *Escherichia*, and *Klebsiella* (Budachetri et al. 2014). An unknown *Borrelia* species has been identified by immunofluorescence antibody staining in a Gulf Coast tick collected from a dog in Alabama (Wright et al. 1997), and *Borrelia lonestari* has been detected in Gulf Coast ticks collected from animals in Arkansas (Fryxell et al. 2012). A novel *Borrelia* species detected recently in questing adult *A. maculatum* ticks from Mississippi shows closest identity to the reptile-associated spirochete, *Borrelia turcica* (Lee et al. 2014).

Conclusions

There has been a remarkable ebb and flow of interest in the Gulf Coast tick during the past century. These changes reinforce the notion that the perceived importance of a particular tick species to human or animal health is relatively fluid, and may shift dramatically with changes in the distribution and abundance of the arthropod, its vertebrate hosts, or the microbial agents that transit among these organisms (Stromdahl and Hickling 2012). In the same context, the geographical distributions of many tick species are not fixed in time or space, and recognized boundaries may expand or diminish over relatively short intervals.

Estimates of Gulf Coast tick distributions are derived from data collected predominantly during the past 100 years, and these have been used to constitute the "normal" or "baseline" range of *A. maculatum*. An alternate view of these data is that current incursions of Gulf Coast tick populations into areas far beyond historical boundaries might also reflect a *return* of this species to a more ancestral distribution, rather than unprecedented encroachments within novel habitats (Teel et al. 2010). By example, a late 19th-century monograph of ticks of Virginia (Niles 1898) likely misidentified a Gulf Coast tick specimen as "*Dermacentor occidentalis* Marx" (Hooker et al. 1912), to suggest that *A. maculatum* existed in Virginia during the early 20th century, yet approximately 60 yr later, repeated acarological surveys in Virginia determined that *A. maculatum* was not established in this state (Sonenshine et al. 1965, Sonenshine 1979). As recently as 1971, no specimens of *A. maculatum* were identified among any of 110 white-tailed deer examined from Virginia during a multistate survey of ectoparasites of deer (Kellogg et al. 1971). Nonetheless, contemporary surveys indicate that Gulf Coast ticks are established throughout multiple southeastern and northern counties areas of Virginia (Wright et al. 2011, Fornadel et al. 2011, Orr et al. 2013, Nadolny et al. 2014).

There are no longitudinal density studies of *A. maculatum* that distinguish recent invasions from previously overlooked populations, and the current evidence for changes in spatial distributions of Gulf Coast ticks in the United States is largely anecdotal; however, compelling data suggest that dynamic changes in range have indeed occurred in many states. These are supported further by a recent analysis of tick mitochondrial 16S rRNA haplotypes of *A. maculatum* populations from 10 sites throughout northern, central, and southern Mississippi, which revealed no significant differences between the distributions of polymorphisms identified in coastal populations and those identified in northern populations, approximately 100 miles beyond historical range estimates, suggesting relatively recent migration events from the south to the north (Ferrari et al. 2013a,b).

The confirmation of disease caused by *R. parkeri* has encouraged rickettsiologists to reconsider this formally obscure bacterium, and investigators now use *R. parkeri* as a model for exploring the microbiology and pathogenesis of other spotted fever group *Rickettsia* species. Because naturally acquired isolates of *R. parkeri* lack plasmids (Baldridge et al. 2010), this spotted fever group *Rickettsia* can be used for transformation studies and analysis of rickettsial gene function (Burkhardt et al. 2011, Welch et al. 2012). Recent investigations with *R. parkeri* have identified host cytoskeletal proteins involved in the assembly of actin bundles that spotted fever group *Rickettsia* species use to invade and spread through mammalian cells (Serio et al. 2010, Reed et al. 2012, 2014). A growing catalogue of resources is available to investigators with interest in exploring the biology and dynamics between *A. maculatum* and *R. parkeri* that include >25 low-passage isolates of *R. parkeri* from ticks and humans (Paddock et al. 2010, Fornadel et al. 2011, Varela-Stokes et al. 2011), the characterization of the full suite of genes encoding the salivary gland proteins of *A. maculatum* (Karim et al. 2011), and molecular techniques to measure even small differences in transcriptional expression of *A. maculatum* (Browning et al. 2012). As an example, investigators have determined recently that genetic manipulation of a selenocysteine-specific elongation factor in *A. maculatum* alters total antioxidant capacity in the tick salivary gland cells, and that the transcriptional levels of catalase and selenoprotein M, are significantly diminished in *R. parkeri*-infected ticks (Adamson et al. 2013).

The complexity of microbial communities residing within Gulf Coast ticks continues to expand as investigators apply increasingly specific molecular techniques to probe for other organisms (Budachetri et al. 2014), and it is likely that other, as-yet undiscovered bacteria, viruses, or protozoans are harbored within or transmitted by *A. maculatum*. As Telford and Goethe (2008) recently noted:

> Pathogens, vectors and reservoir hosts exist in predictable ecological assemblages. An extension of this idea is that microbes exist in guilds, which are the basic units of community structure and represent groups of unrelated taxa that share a common resource. . . .because of microbial guilds, rarely does just one agent emerge.

Salient examples of the guild concept include *I. scapularis* and *A. americanum*. Neither of these species were considered as ticks of medical or veterinary

importance until the latter part of the 20th century; nonetheless, *I. scapularis* is now recognized as vector of at least six important pathogens, including *Babesia microti*, *B. burgdorferi*, *Anaplasma phagocytophilum*, deer tick virus, *Borrelia miyamotoi*, and an *Ehrlichia muris*-like agent (Piesman and Spielman 1980; Burgdorfer et al. 1982; Telford et al. 1996, 1997; Scoles et al. 2001; Pritt et al. 2011), and *A. americanum* is associated with a growing list of infectious agents that include *E. chaffeensis*, *E. ewingii*, Panola Mountain *Ehrlichia*, *Cytauxzoon felis*, and Heartland virus (Anderson et al. 1993, Wolf et al. 2000, Childs and Paddock 2003, Loftis et al. 2008b, Reichard et al. 2010, Savage et al. 2013).

More than 170 yr after the original description of *A. maculatum*, many unanswered questions remain concerning its biology, ecology, and associated pathogens. To what extent, if any, does *R. parkeri* use animal hosts for replication and multiplication? Do infections with *R. parkeri* and *H. americanum* influence the fecundity or survival of *A. maculatum*? What is the prevalence of infection with these pathogens in immature Gulf Coast ticks? Is "*Ca*. R. andeanae" a potential pathogen? Does it interfere with *R. parkeri* within the tick host? Are there other, as-yet undiscovered bacteria, viruses, or protozoans transmissible from *A. maculatum* to vertebrate hosts? The next decade will undoubtedly yield new information about the Gulf Coast tick, its microbial associates, and their impact on human and animal health in the Western Hemisphere.

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