Equine Piroplasmosis

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KEYWORDS

- *Theileria equi* • *Babesia caballi* • Erythrocytic parasite • Tick-borne disease
- Anemia

KEY POINTS

- The disease equine piroplasmosis is caused by tick-transmitted, intraerythrocytic parasites *Theileria equi* and *Babesia caballi*.
- Clinical signs of acute infection generally include those related to intravascular hemolysis and thrombocytopenia. All infected animals that survive become subclinical carriers and are reservoirs for transmission.
- The United States and Canada are considered nonendemic for equine piroplasmosis, yet recent outbreaks in the United States have elucidated challenges with control and prevention.
- Control of infection and disease in nonendemic areas is provided by import surveillance and can only be completed by a certified laboratory.
- Alleviation of clinical symptoms and clearance of the parasite can be accomplished with imidocarb diproprionate.

INTRODUCTION

Equine piroplasmosis is an infectious, tick-borne disease caused by the hemoprotozoan parasites *Theileria* (previously *Babesia*) *equi* and *Babesia caballi*. Piroplasmosis affects all wild and domestic equids and clinical presentation is related to intravascular hemolysis and associated systemic illness. Infection with either parasite can lead to a

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similar clinical presentation yet *T equi* and *B caballi* are distinct in terms of disease severity, life cycle, infection dynamics, persistence in the horse, and drug susceptibility. Although most horses recover from the initial phase of the disease, infection can be fatal. Horses that survive acute disease inevitably become inapparent carriers and exhibit no clinical signs of infection yet can serve as reservoirs for transmission to naive horses.[1] Carriers represent challenges in diagnosis, eradication, and control measures. The parasites and their natural tick vectors are endemic to most countries with tropical and subtropical climates yet the United States and Canada, and a small number of other nations, are considered "free" or nonendemic.[2,3] Goals of infection and disease control vary tremendously between endemic and nonendemic nations, and as demonstrated by the "silent" re-emergence of *T equi* in 2009 on a ranch in Texas, endeavors to control transmission especially in areas that wish to remain nonendemic must continue.[4]

ETIOLOGY

Theileria equi and *B caballi* are obligate intraerythrocytic apicomplexan parasites that infect only equids. Taxonomy of the causative agents of piroplasmosis has been in question since their discovery and remains controversial for *T equi*.[5,6] It was recognized in 1998 that *T equi* did not fit into "babesia" taxonomy given its size and an extraerythrocytic stage within equine peripheral blood mononuclear cells (PBMCs). Molecular phylogenetic investigations and recent genomic analyses support both babesia and theileria lineages, possibly placing it between the two, perhaps in a new genus.

The recent analysis of the genome for the laboratory or "Florida" strain of *T equi*, which was isolated in the 1970s, has also allowed more detailed insight into the parasite's molecular pathways and genetic structure.[5] These genomic data and additional research further confirmed the assumption that geographic isolates of *T equi* can genetically differ greatly from one another.[7] Globally, it is recognized that *T equi* causes more disease in some areas than others. For example, *T equi* is recognized as a common reason for a horse to be admitted to an equine intensive care unit in South Africa, whereas only mild clinical disease was reported in one horse during the US outbreak that affected more than 400 horses.[4,8] Genomic differences have not yet been correlated with virulence. Recently, a *T equi* variant was isolated from a Mexican-origin stray horse intercepted in south Texas and data collected thus far indicate that the parasite is genetically distinct from the *T equi* found in the Texas outbreak, being much more closely related to parasites identified in South African horses and zebras.[7] The impact this information will have on diagnostic testing, therapeutics, and vaccine development is currently under investigation.

Transmission of infection can occur via an infected tick or through iatrogenic blood transfer. The precise tick-vector-parasite-host requirements for infection or clinical disease are not fully understood. Clinically inapparent infection can occur and data indicate that the risk of life-threatening clinical disease increases with the presence of factors, such as immunologic naivety and increased density of infected ticks and horses.[9] Ticks competent for transmission are biologic vectors and therefore incredibly efficient. Competent tick vectors are found in almost all climates globally. Only hard ticks, or ixodid ticks, are capable of transmitting *T equi* and *B caballi* naturally, and in North America, there are five species of ticks that are known to act as competent vectors (**Box 1**).[1,10–14] Other species have been implicated as potential vectors, but confirmation requires additional research.