ies suggest that \( \alpha_1 \)-adrenergic receptors are likely involved in mediating this response. Use of prazosin by Chabot et al, in their study, would have helped solve this issue. In addition, there is a wide interspecies variation in the pulmonary vascular response to hypoxia, as well as in the affinities of various drugs acting on \( \alpha_1 \)-adrenergic receptors.

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Ehrlichiosis in the United States

To the Editor:

We read with interest the recently published clinical review by Faul et al (July 1999) on tick-borne pulmonary disease. However, the authors stated that the sole causative agent of ehrlichiosis in the United States is the rickettsia Ehrlichia chaffeensis. It has recently been recognized that are at least two similar but distinct human diseases caused by Ehrlichia species in the United States. Human monocytic ehrlichiosis is caused by E chaffeensis. Human granulocytic ehrlichiosis (HGE), first described in 1994, is caused by a species closely related to the Ehrlichia equi/plagiorchis group. While the clinical presentation of these two forms of ehrlichiosis may be similar, HGE appears to be a more virulent illness. Our review on the respiratory manifestations of tick-borne disease failed to determine which form of ehrlichiosis is associated with pneumonia and respiratory failure.

More recently, an additional ehrlichial species, Ehrlichia ewingii, has been identified to cause clinical disease that is indistinguishable from infection caused by E chaffeensis or the agent of HGE. Unfortunately, the clinical manifestations of these four patients from Missouri who were identified with this infection were not given in detail, so we could not determine from the article whether these patients had any respiratory manifestations from their rickettsial infection. Thus, there now appears to be documentation of at least three separate ehrlichial infectious agents in the United States. Others may be recognized as technology allows better identification of these important emerging infections.

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REFERENCES


To the Editor:

We wish to thank Byrd et al for their interest in our article. Our comment that Ehrlichia chaffeensis is the sole causative agent of human ehrlichiosis is an error in writing, as documented by our references to human granulocytic ehrlichiosis (HGE) and Ehrlichia ewingii causing human disease in the same paragraph.

Indeed, there appear to be at least four species of Ehrlichia that infect humans: E chaffeensis, E sennetsu, E canis, and the agent of HGE. Other species have been identified in ticks and dogs, including E equi and E platys, but their role as potential human pathogens is still unclear.

The precise identification of tick-borne pathogens is fundamental to our understanding of their epidemiology and prevention. Since the submission of our paper, there have been additional developments in the diagnosis of tick-borne diseases. The use of polymerase chain reaction technology promises to allow further identification of Ehrlichia species in a variety of settings (in particular in seronegative hosts). In addition, a recent report describes the use of Borrelia burgdorferi-specific serum immune complexes in the diagnosis of Lyme disease, including in subjects with early (seronegative) erythema migrans.

When a patient presents with pulmonary symptoms and exposure to ticks, the chest physician is faced with the task of identifying (and treating) the pathogen most likely to fit the clinical picture. Clinical features of skin rashes, nerve palsies, and fever may help to distinguish one tick-borne disease from another. However, the accurate identification of different species of Ehrlichia on the basis of clinical features alone is more challenging, in part because the existing data on human ehrlichiosis are limited to case reports and small case series. It is reasonable to assume that each of the Ehrlichia species may lead to serious pulmonary disease, but this remains unproven. In their recent report, Buller et al suggest that the clinical features of E ewingii infection mimic those caused by E chaffeensis or the agent of HGE. Interestingly, three of their four cases were receiving immunosuppressive therapy, suggesting that host factors also play a role in the clinical expression of disease. In addition, subjects at risk for tick-borne disease may be co-infected with other tick-borne pathogens, making any interpretation of causality more complex. Chest physicians should be aware of the broad range of pathogens that may lead to chest disease after tick bites. We hope that a greater awareness of tick-borne infections among chest physicians might lead to increased reporting and understanding of these treatable diseases.