Histophilus somni (formerly known as Haemophilus somnus) was first identified as a bovine pathogen and cause of thrombotic meningoencephalitis (TME) only 55 years ago, and Haemophilus agni (now also recognized as H. somni) a cause of septicemic disease in lambs two years earlier. The only known habitats of H. somni are the mucosal surfaces of ruminants, making this bacterium an opportunistic pathogen. H. somni is responsible for a wide variety of systemic diseases in addition to TME, including respiratory disease syndromes, myocarditis, reproductive disease syndromes, polyarthritis, mastitis, ocular disease, and septicemia. Nonetheless, some H. somni isolates from genital sites may not be pathogenic at all. Although this bacterium is capable of causing inflammation at systemic sites (vasculitis is a hallmark of infection due to H. somni) and is toxic to epithelial and phagocytic cells, the bacterium’s wide array of virulence factors act primarily as a defense against, or to escape recognition from, host innate and adaptive immunity. Although no longer recognized as a member of the genus Haemophilus due to genetic dissimilarity, many genes responsible for virulence attributes in H. somni are conserved in Haemophilus influenzae, the type species of the genus, and homologs are common in other members of the Pasteurellaceae and Neisseria spp. Although antibodies to certain membrane or surface proteins have been shown to be protective, particularly of the IgG2 allotype, correlates of protective immunity are incomplete. The role of cellular immunity in protection against histophilosis has not been adequately examined, and therefore the specificity and nature of the broader host immune response required to provide optimal protection against systemic diseases due to H. somni remains unclear. Further complicating matters is that H. somni is capable of forming a biofilm in host tissues. The biofilm likely interferes with the susceptibility of the bacterium to host immunity and complicates measuring protective immunity in model systems that utilize planktonic cells. Furthermore, the genomes of only two strains of H. somni, a pathogenic pneumonia isolate and a nonpathogenic preputial genital isolate, have been sequenced. Therefore, unlike most other bacterial species of disease and economic importance, there is a woeful deficiency in the number of isolates whose genomes have been
sequenced. Essential information regarding whether isolates from various geographic regions share or lack genes responsible for virulence factors, or may have important virulence traits not yet recognized, is lacking.

In this volume experts who have contributed to the *H. somni* literature have reviewed what is currently known regarding the taxonomy, disease syndromes, genetics, biology, and pathogenic factors of *H. somni*, and the host immune response to this pathogen. We gratefully recognize that much of this work could not have been accomplished without the financial support of the United States Department of Agriculture-National Institute of Food and Agriculture, and other federal agencies worldwide.

Thomas J. Inzana