Introduction

This supplement is composed of presentations at the Ninth Annual International Conference on Lyme Borreliosis and Other Tick-borne Diseases held on 19–20 April 1996 in Boston. This conference, which was sponsored by the Lyme Disease Foundation in Hartford, Connecticut, focused on "Chronic Lyme Disease: Basic and Clinical Approaches." The conference was divided into sessions on animal models, pathogenesis, laboratory confirmation, and prevention and treatment of chronic Lyme disease as well as a session on emerging tick-borne diseases. The term "chronic Lyme disease" is used to describe the long-term, frequently ongoing symptoms and sequelae of infection with *Borrelia burgdorferi*. Patients with chronic Lyme disease may manifest objective findings (e.g., arthritis) but more commonly have subjective symptoms of encephalopathy, peripheral and radicular neuropathy, arthralgias or myalgias, and fatigue.

Investigators attempting to identify suitable animal models for Lyme disease have made significant advances within the past several years. Rabbits, hamsters, mice, and monkeys currently serve as models for specific aspects of this multisystemic disease. Stephen Barthold (Yale University) reviewed his investigations with use of the mouse model. This model is unparalleled by any other animal model because of the availability of numerous inbred strains with defined genetics and different susceptibilities to Lyme disease.

Several presentations focused on the mechanisms that may be involved in the persistence of *B. burgdorferi* infection. Janis Weis (University of Utah) associated tissue invasion with persistence of infection and used tissue levels of spirochetal DNA to explain clinical differences between two inbred species of mice. David Dorward (National Institutes of Health [NIH], Rocky Mountain Laboratories) detailed the interactions of *B. burgdorferi* and human B and T lymphocytes in vitro. He stated that spirochetes can invade the lymphocytes and emerge surrounded by host cell membrane, suggesting a possible mechanism to avoid recognition. Elizabeth Aberer (University of Graz, Austria) examined the persistence of *Borrelia* organisms in chronic skin lesions and determined that Langerhans' cells are heavily damaged; she suggested that this finding might explain a patient's impaired capacity to eliminate *B. burgdorferi* from skin sites.

Various serological tests and the role of humoral immunity in preventing chronic disease were examined in two presentations. Charles Pavia (New York Medical College) explained that in a mouse model, sera from patients with late Lyme disease uniformly exhibited antiborrelial activity, whereas sera from patients with early disease demonstrated significantly less activity. Richard Tilton (BBI–North American Laboratory Groups) reported that the Centers for Disease Control and Prevention criteria for standardization of interpretations of western blot testing for Lyme disease result in underdiagnosis.

Two recombinant vaccines for Lyme disease are currently undergoing human clinical trials. François Meurice (Smith-Kline Beecham) discussed the issues involved with the design of an efficacy study and the current status of one trial.

It has been recently recognized that other newly emerging or reemerging pathogens can be transmitted by *Ixodes* ticks. David Persing (Mayo Clinic) reported on coinfection with *B. burgdorferi* and *Babesia* in patients and speculated that infection by the latter species may lead to immunosuppression and a decreased immune response to Lyme disease. He further indicated that cases of human ehrlichiosis have been reported from the Midwest since the 1970s. Stephen Dumler (Johns Hopkins) detailed the clinicopathological features of human ehrlichiosis and compared them with those of Lyme disease.

Brian Fallon (Columbia University) reviewed the neuropsychiatric manifestations of Lyme disease, noting that depressive and cognitive disorders predominate and can be difficult to diagnose as Lyme disease. He reported that single photon emission CT scans are proving helpful in diagnosing CNS Lyme disease.

Claude Garon (NIH, Rocky Mountain Laboratories) described the in vitro inhibitory effects of melittin, a polypeptide found in bee venom, on *B. burgdorferi*. Sam Donta (Boston University) reported on his experiences treating clinically defined chronic Lyme disease with tetracycline.

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