Genetics of <i>Borrelia burgdorferi</i>

Abstract:

The spirochetes in the Borrelia burgdorferi sensu lato genospecies group cycle in nature between tick vectors and vertebrate hosts. The current assemblage of B. burgdorferi sensu lato, of which three species cause Lyme disease in humans, originated from a rapid species radiation that occurred near the origin of the clade. All of these species share a unique genome structure that is highly segmented and predominantly com- posed of linear replicons. One of the circular plasmids is a prophage that exists as several isoforms in each cell and can be transduced to other cells, likely contributing to an otherwise relatively anemic level of horizontal gene transfer, which nevertheless appears to be adequate to permit strong natural selection and adaptation in populations of B. burgdorferi. Although the molecular genetic toolbox is meager, sev- eral antibiotic-resistant mutants have been isolated, and the resistance alleles, as well as some exogenous genes, have been fashioned into mark- ers to dissect gene function. Genetic studies have probed the role of the outer membrane lipoprotein OspC, which is maintained in nature by multiple niche polymorphisms and negative frequency-dependent se- lection. One of the most intriguing genetic systems in B. burgdorferi is vls recombination, which generates antigenic variation during infection of mammalian hosts.