Interaction forces between the extracellular matrix and variants of borrelial decorin binding protein A probed by atomic force microscopy

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M. Strnad[I], Y. Oh[II], M. Vancova[I], P. Hinterdorfer[II], L. Grubhoffer[I], R.O. Rego[I]

[I]*Institute of Parasitology, Biology Centre of the Academy of Sciences of the Czech Republic, Ceske Budejovice, Czech Republic,* [II]*Johannes Kepler University in Linz, Linz, Austria*

The spirochetal bacterium *Borrelia burgdorferi*, the etiologic agent of Lyme disease (LD), infects mammalian hosts via the bite of *Ixodes* spp. ticks. Unlike in North America, where the infection is caused only by *B. burgdorferi sensu stricto*, in Europe there are a number of *B. burgdorferi* genospecies that are associated with human LD. Different genospecies display different patterns of host specialization and tissue tropism and are associated with distinct spectra of clinical manifestations.

It is well established that *B. burgdorferi* modulates its gene expression as it cycles between the tick vector and the mammalian host they infect. Upon host invasion, *B. burgdorferi* has avoid vigorous host immune response in order to colonize and persist in the host. *B. burgdorferi* binds various components of the host extracellular matrix (ECM). Arguably the most well studied borrelial adhesin is decorin-binding protein A (DbpA). DbpA was identified as the adhesin responsible for binding to the proteoglycan decorin. Interestingly, DbpA amino acid sequences are highly polymorphic between the genospecies, with sequence similarities as low as 40–60%. This sequence heterogeneity results in structural variations that contribute to large differences in the adhesion activities of the protein, which ultimately influences the tissue tropism and disease manifestation of the genospecies.

Using atomic force microscopy, we measured specific binding forces of single bio-molecular adhesive interactions between variants of borrelial DbpA and several ECM components, and localized the interactions on the surface of *Borrelia*.