Antibodies against *Borreliella spp.* Antigens

Lyme disease is the most common tick-borne disease in the northern hemisphere, caused by *Borreliella spp*. Even though Lyme disease was originally identified in the town of Lyme, Connecticut (USA) in the mid 1970s, prospective studies revealed an earlier history in untreated European patients (Borchers *et al.* 2015). The various clinical manifestations include flu-like symptoms and a characteristic circular skin rash called erythema migrans (EM).

Even though more abundant in the United States the total number of annual cases of Lyme Disease in Europe is estimated to be approximately 3-fold higher than the number of reported cases. Climate changes with a subsequent expansion of vector tick territory, along with changes in land use might affect changes in epidemiology of Lyme Disease and other tick-borne infections in the future (Andersen and Davis 2016).

The complex endozoonotic cycle of *Borreliella spp*. in Europe usually involves transmission to a broad range of vertebrates by infected ticks of the species *Ixodes ricinus* (Rizzoli *et al.* 2011). Due to the presence of different ecotypes within the species and utilization of different host reservoirs, Margos *et al.* (2009) analysed intra- and interspecific relationships by multilocus sequence analysis (MLSA). Apart from *B. garinii* and *B. bavariensis*, cases in Europe can be caused by *B. afzelii*, *B. spielmanii,* and *B. burgdorferi* sensu stricto. Similar immunogenic proteins of *Borreliella* species are associated in US and European cases. Especially outer surface proteins A and C (OspA, OspC), and variable major protein-like sequence Expressed 1 (VlsE1) appear to be important for infection and immune evasion. Basic membrane protein A (BmpA) serves as adhesin for binding to the host cell. Further immunogens are p58 and p100, whose cellular functions and pathogenic implications have not been clarified yet. The most sensitive protein for IgG antibody detection in all stages of Lyme disease was found to be VLsE1 followed by p100 and p58 (Goettner *et al.* 2005).

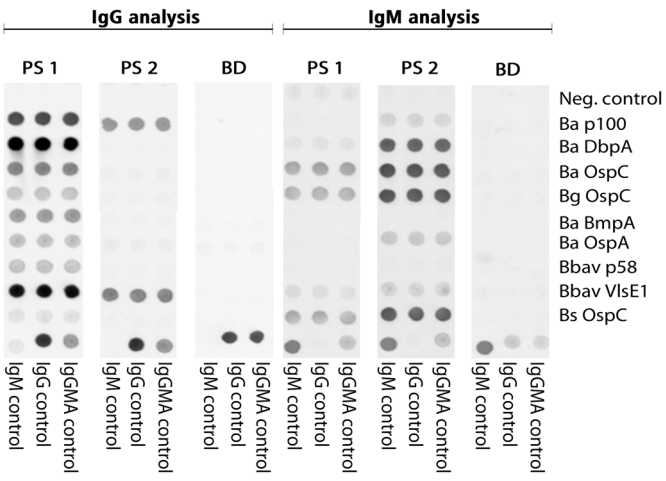

Figure: Immunodot analyses of positive (PS1, PS2) and negative (BD) samples for Borreliella afzelii (Ba), B. bavariensis (Bbav), B. garinii (Bg), B. spielmanii (Bs). The presence of IgG (left) and IgM (right) antibodies was determined spotting triplicates of recombinant DIARECT antigens derived from Borreliella spp. on nitrocellulose membrane.

The DIARECT's recombinant *Borreliella spp.* antigens are expressed in either *E. coli* or the baculovirus/insect cell expression system.

Ordering Information			
41100 41101	*Borreliella afzelii* BmpA		0.1 mg 1.0 mg
40900 40901	*Borreliella afzelii* DbpA		0.1 mg 1.0 mg
41000 41001	*Borreliella afzelii* OspA		0.1 mg 1.0 mg
41800 41801	*Borreliella afzelii* OspC		0.1 mg 1.0 mg
42300 42301	*Borreliella afzelii* p100		0.1 mg 1.0 mg
42700 42701	*Borreliella bavariensis* DbpA	NEW!	0.1 mg 1.0 mg
40700 40701	*Borreliella bavariensis* p58		0.1 mg 1.0 mg
41400 41401	*Borreliella bavariensis* VlsE1		0.1 mg 1.0 mg
42800 42801	*Borreliella garinii* DbpA	NEW!	0.1 mg 1.0 mg
41900 41901	*Borreliella garinii* OspC		0.1 mg 1.0 mg
42900 42901	*Borreliella spielmanii* DbpA	NEW!	0.1 mg 1.0 mg
40800 40801	*Borreliella spielmanii* OspC		0.1 mg 1.0 mg

References:
Andersen and Davis (2016) Int J Dermatol. 33: 2358-2365
Borchers *et al.* (2015) J Autoimmun. 57:82-115
Goettner *et al.* (2005) J Clinical Microbio. 43: 3602-3609
Magros *et al.* (2009) Appl Environ Microb. 75: 5410-5416
Rizzoli *et al.* (2011) Euro Surveill. 16:pii 19906

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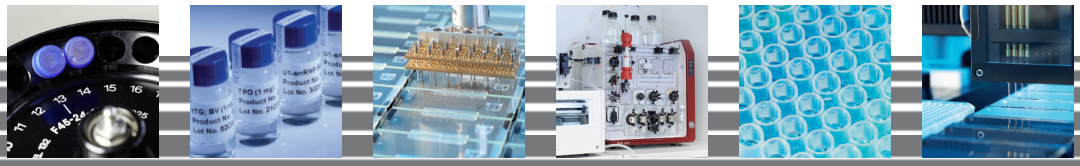

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