DIARECT
Part of BBI Solutions

Bartonella henselae Antigens

Bartonella spp. are causative agents of emerging human and veterinary diseases. The rod-shaped, gram-negative and facultative intracellular pathogen *Bartonella henselae* causes several forms of Bartonellosis including Cat Scratch Disease (CSD) and Bacillary Angiomatosis (BA), which are significant co-infections in Lyme Disease (Higgins *et al.* 1996; Anderson *et al.* 1995). *B. henselae* is the only species known to provoke CSD, while BA has also been associated with *B. guitana*. CSD was first identified as clinical entity in France (Debré *et al.* 1950). In 1992, Regnery *et al.* discovered an etiologic agent of CSD and the clinical syndrome BA. The bacteria were named *Rochalimaea henselae*, and after later phylogenetic analysis classified under the *Bartonella* genus (Brenner *et al.* 1991).

Cat Scratch Disease is caused by traumatic cat contact, mainly via scratches and bites or cat fleas (Anderson *et al.* 1996; Higgins *et al.* 1996). Cats are the natural reservoir for *B. henselae* (Higgins *et al.* 1996), which are most likely infected by fleas and usually asymptomatic. However, CSD is also considered a tick transmitted disease (Cotté *et al.* 2008). According to a US study infections caused by *B. henselae* are distributed worldwide, with an incidence of 3.7 per 100,000 (Prutsky *et al.* 2013).

Main manifestations of Bartonellosis at disease onset include the development of lymphadenopathy (Higgins *et al.* 1996), followed by persistent fever, abdominal pains and loss of weight. Encephalopathy occurs very frequently. Furthermore, fatigue and neuronal manifestations such as memory loss and disorientation are described (Berghoff *et al.* 2012).

Through a Type IV secretory system, *B. henselae* proteins are transported into the host cells, which provoke vascular proliferation in infected endothelial cells (Hoey *et al.* 2009). After induction through endothelial cells the microorganisms cause deformation on the outside of the erythrocyte membrane and with increasing duration of infection, the bacterium is primarily intracellular where it replicates (Berghoff *et al.* 2012).

Key factors used as antigens for diagnostics are highly immunoreactive proteins produced in *B. henselae*. The first antigen, found to be strongly reactive with sera from CSD patients, was p17 (Sweger *et al.* 2000), considered to be a species specific marker for *B. henselae* during early stages of infection. The gene encoding this protein lies in the virB-operon of the type IV secretion system (Hoey *et al.* 2009). Sequence analyses indicate that the protein is a bacterial membrane-associated protein (Anderson *et al.* 1995) similar to the outer membrane protein p26 that also contains dominant antigenic sites for CSD patient antibodies (Werner *et al.* 2008). The immunogenic protein dihydrolipoamide-succinyltransferase (SucB) is a component of the 2-oxoglutarate dehydrogenase complex and involved in catalysis of the overall conversion of 2-oxoglutarate to succinyl-CoA and CO_2 (Gilmore *et al.* 2003).

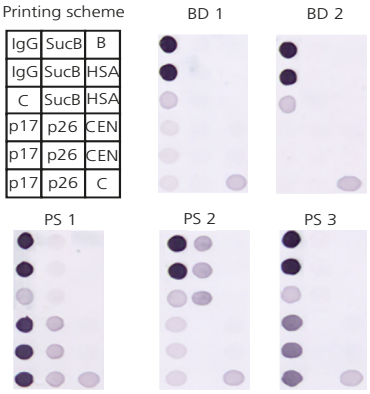

Figure: Immunodot analyses of negative (BD 1-2) and positive samples (PS 1-3) for the Bartonella henselae antigens p17, p26 and SucB. The presence of antibodies was determined spotting triplicates of recombinant DIARECT antigens on nitrocellulose membrane. Positive (anti-IgG (IgG) and anti-IgGMA (C)) and negative controls (Buffer (B), HSA and CENP-B antigen (CEN)) were spotted in the left and right columns.

DIARECT's *Bartonella henselae* antigens p17 (17 kDa protein), p26 (26 kDa protein), and SucB (dihydrolipoamide-succinyltransferase) are produced in *E. coli*.

Ordering Information		
45000	*Bartonella henselae* 17 kDa	0.1 mg
45001		1.0 mg
45100	*Bartonella henselae* 26 kDa	0.1 mg
45101		1.0 mg
45200	*Bartonella henselae* SucB	0.1 mg
45201		1.0 mg

References:
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Cotté *et al.* (2008) Emerg Infect Dis.14: 1074-1080
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Gilmore *et al.* (2003) Infect Immun. 71: 4818-4822
Higgins *et al.* (1996) J Med Entomol. 33: 490-495
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Prutsky *et al.* (2013) Int J Infect Dis. 17: e811-e819
Regnery *et al.* (1992) The Lancet. 339 (8807): 1443-1445
Sweger *et al.* (2000) Clin Diagn Lab Immunol. 7: 251-257
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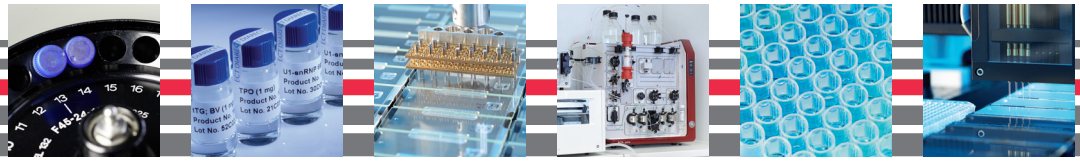

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