SANTA CRUZ BIOTECHNOLOGY, INC.

IcsA (bL-20): sc-17389

BACKGROUND

Infection by the gram-negative bacterium *Shigella flexneri* results in dysentery, an acute inflammatory disease of the colon. Essential events in the pathogenesis of *Shigella* infections include bacterial invasion of epithelial cells, escape from the phagosome, and induction of apoptosis in macrophages. The *Shigella* outer membrane protein IcsA belongs to the family of type V secreted (autotransported) virulence factors. Members of this family mediate their own translocation across the bacterial outer membrane: the carboxy-terminal β domain forms a β barrel channel in the outer membrane through which the amino-terminal α domain passes. IcsA, which is localized at one pole of the bacterium, mediates actin assembly by *Shigella*, which is essential for bacterial intracellular movement. IcsA, a 120 kDa protein, directly binds two proteins, vinculin and neural-Wiskott-Aldrich Syndrome protein (N-WASP). In turn, VASP (vasodilator stimulated phosphoprotein) can recruit profilin to the bacterial surface, which can provide actin for tail construction. N-WASP binding of IcsA can also recruit profilin to the bacterial surface and may be another means of obtaining monomeric actin for tail formation and subsequent bacterial motility.

REFERENCES

1. Egile, C., Loisel, T.P., Laurent, V., Li, R., Pantaloni, D., Sansonetti, P.J., and Carlier, M.F. 1999. Activation of the Cdc42 effector N-WASP by the *Shigella flexneri* IcsA protein promotes actin nucleation by Arp2/3 complex and bacterial actin-based motility. J. Cell. Biol. 6: 1319-1332.
2. Charles, M., Magdalena, J., Theriot, J.A., and Goldberg, M.B. 1999. Functional analysis of a rickettsial OmpA homology domain of *Shigella flexneri* IcsA. J. Bacteriol. 3: 869-878.
3. Brandon, L.D. and Goldberg, M.B. 2001. Periplasmic transit and disulfide bond formation of the autotransported *Shigella* protein IcsA. J. Bacteriol. 3: 951-958.
4. Guichon, A., Hersh, D., Smith, M.R., and Zychlinsky, A. 2001. Structure-function analysis of the *Shigella* virulence factor IpaB. J. Bacteriol. 4: 1269-1276.
5. Sansonetti, P.J. 2001. Microbes and microbial toxins: paradigms for microbial-mucosal interactions III. *Shigellosis*: from symptoms to molecular pathogenesis. Am. J. Physiol. Gastrointest. Liver Physiol. 3: G319-323.

SOURCE

IcsA (bL-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of IcsA of *S. flexneri* origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-17389 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

IcsA (bL-20) is recommended for detection of IcsA of *S. flexneri* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.