SANTA CRUZ BIOTECHNOLOGY, INC.

IpaC (bI-15): sc-17387

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* virulence factor invasion plasmid antigen B (IpaB) is required for all of these processes. Induction of apoptosis is dependent on IpaB binding to the cysteine protease caspase-1 (Casp-1). The activation of this enzyme triggers both apoptosis and release of the proinflammatory cytokine interleukin-1β. The N-terminal portion of IpaB is necessary for stable expression of IpaB. The effectors of *Shigella flexneri* invasion are the Ipa proteins, particularly IpaB and IpaC, which are secreted at the host-pathogen interface following bacterial contact with a host cell. Of the Ipa proteins, only IpaC has been shown to possess quantifiable *in vitro* activities that are related to cellular invasion. Entry of *S. flexneri* into epithelial cells and lysis of the phagosome involve the IpaB, IpaC, and IpaD proteins, which are secreted by type III secretion machinery.

REFERENCES

1. Turbyfill, K.R., Hartman, A.B., and Oaks, E.V. 2000. Isolation and characterization of a *Shigella flexneri* invasin complex subunit vaccine. Infect. Immun. 68: 6624-6632.
2. De Geyter, C., Wattiez, R., Sansonetti, P., Falmagne, P., Ruysschaert, J.M., Parsot, C., and Cabiaux, V. 2000. Characterization of the interaction of IpaB and IpaD, proteins required for entry of *Shigella flexneri* into epithelial cells, with a lipid membrane. Eur. J. Biochem. 267: 5769-5776.
3. Guichon, A., Hersh, D., Smith, M.R., and Zychlinsky, A. 2001. Structure-function analysis of the *Shigella* virulence factor IpaB. J. Bacteriol. 183: 1269-1276.
4. Picking, W.L., Coye, L., Osiecki, J.C., Barnoski-Serfis, A., Schaper, E., and Picking, W.D. 2001. Identification of functional regions within invasion plasmid antigen C (IpaC) of *Shigella flexneri.* Mol. Microbiol. 39: 100-111.
5. Szakal, D., Gado, I., and Pal, T. 2001. A colony blot immunoassay to detect enteroinvasive *Escherichia coli* and *Shigella* in water samples. J. Appl. Microbiol. 90: 229-236.

SOURCE

IpaC (bI-15) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of IpaC 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-17387 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

IpaC (bI-15) is recommended for detection of IpaC of *S. flexneri*, *S. dysenteriae* 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.