SANTA CRUZ BIOTECHNOLOGY, INC.

VacA (bN-20): sc-17447

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

Helicobacter pylori is a spiral shaped bacterium that accounts for eighty percent of stomach ulcers and more than ninety percent of duodenal ulcers. Infection with *H. pylori* is also associated with the development of gastric cancer. The vacuolating toxin VacA is a major determinant of *H. pylori*-associated gastric disease. In non-polarized cells, VacA alters the endocytic pathway, resulting in the release of acid hydrolases and the reduction of both extracellular ligand degradation and antigen processing. The toxin forms transmembrane anion-specific channels and reduces the transepithelial electrical resistance of polarized monolayers. Localization of the VacA channels in acidic intracellular compartments causes osmotic swelling, which, together with membrane fusion, leads to vacuole formation. This protein has recently been shown to be an important antigen in the human immune response to *H. pylori* infection. Cytotoxin associated gene A, otherwise known as CagA, is closely associated with VacA. CagA, a 120 kDa protein, induces morphological changes in the host, as well as inducing actin reorganization, variations in the cell cycle and autocrine effects.

REFERENCES

1. Konturek, P.C., Bielanski, W., Konturek, S.J. and Hahn, E.G. 1999. *Helicobacter pylori* associated gastric pathology. J. Physiol. Pharmacol. 5: 695-710.
2. McGee, D.J. and Mobley, H.L. 1999. Mechanisms of *Helicobacter pylori* infection: bacterial factors. Curr. Top. Microbiol. Immunol. 241: 155-180.
3. Graham, D.Y. and Yamaoka, Y. 2000. Disease-specific *Helicobacter pylori* virulence factors: the unfulfilled promise. Helicobacter Suppl. 1: S3-9; discussion S27-31.
4. Dundon, W.G., de Bernard, M. and Montecucco, C. 2001. Virulence factors of *Helicobacter pylori*. Int. J. Med. Microbiol. 8: 647-658.
5. Censini, S., Stein, M. and Covacci, A. 2001. Cellular responses induced after contact with *Helicobacter pylori*. Curr. Opin. Microbiol. 1: 41-46.
6. Sande, N., Nikulin, M., Nilsson, I., Wadstrom, T., Laxen, F., Harkonen, M., Suovaniemi, O. and Sipponen, P. 2001. Increased risk of developing atrophic gastritis in patients infected with CagA+ *Helicobacter pylori*. Scand. J. Gastroenterol. 9: 928-933.

SOURCE

VacA (bN-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of VacA of *H. pylori* 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-17447 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

VacA (bN-20) is recommended for detection of VacA of *H. pylori* 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).

Molecular Weight of VacA: 87 kDa.

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.