SANTA CRUZ BIOTECHNOLOGY, INC.

VacA (5E4): sc-32746

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Helicobacter pylori is a spiral shaped bacterium that accounts for 80% of stomach ulcers and more than 90% of duodenal ulcers. Infection with *Helicobacter pylori* is also associated with the development of gastric cancer. The vacuolating toxin VacA is a major determinant of *Helicobacter 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 *Helicobacter pylori* infection. Cytotoxin associated gene A, also known as CagA, is closely associated with VacA. CagA 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., et al. 1999. *Helicobacter pylori* associated gastric pathology. J. Physiol. Pharmacol. 50: 695-710.
2. McGee, D.J., et al. 1999. Mechanisms of *Helicobacter pylori* infection: bacterial factors. Curr. Top. Microbiol. Immunol. 241: 155-180.
3. Graham, D.Y., et al. 2000. Disease-specific *Helicobacter pylori* virulence factors: the unfulfilled promise. Helicobacter 5: S3-S9.
4. Dundon, W.G., et al. 2001. Virulence factors of *Helicobacter pylori.* Int. J. Med. Microbiol. 290: 647-658.
5. Censini, S., et al. 2001. Cellular responses induced after contact with *Helicobacter pylori.* Curr. Opin. Microbiol. 4: 41-46.
6. Sande, N., et al. 2001. Increased risk of developing atrophic gastritis in patients infected with CagA$^+$ *Helicobacter pylori.* Scand. J. Gastroenterol. 36: 928-933.

SOURCE

VacA (5E4) is a mouse monoclonal antibody detects amino acids 685-821 of purified native s1-m1 VacA.

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available azide-free for VacA toxin neutralizing activity, sc-32746 L, 200 µg/0.1 ml.

VacA (5E4) is available conjugated to agarose (sc-32746 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-32746 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-32746 PE), fluorescein (sc-32746 FITC), Alexa Fluor® 488 (sc-32746 AF488), Alexa Fluor® 546 (sc-32746 AF546), Alexa Fluor® 594 (sc-32746 AF594) or Alexa Fluor® 647 (sc-32746 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-32746 AF680) or Alexa Fluor® 790 (sc-32746 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

VacA (5E4) is recommended for detection of VacA of *H. pylori* origin by immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500); non cross-reactive with type s2-m2 VacA or denatured type s1-m1 VacA.

Molecular Weight of VacA: 87 kDa.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 2) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

SELECT PRODUCT CITATIONS

1. Chang, H., et al. 2016. Cortactin mediates apoptosis of gastric epithelial cells induced by VacA protein of *Helicobacter pylori.* Dig. Dis. Sci. 61: 80-90.
2. Zhu, P., et al. 2017. *Helicobacter pylori* VacA induces autophagic cell death in gastric epithelial cells via the endoplasmic reticulum stress pathway. Cell Death Dis. 8: 3207.
3. Valenzuela-Valderrama, M., et al. 2019. The *Helicobacter pylori* urease virulence factor is required for the induction of hypoxia-induced factor-1α in gastric cells. Cancers 11: 799.
4. Luo, J., et al. 2021. Autophagy induced by *H. pylori* VacA regulated the survival mechanism of the SGC7901 human gastric cancer cell line. Genes Genomics 43: 1223-1230.
5. Do, A.D., et al. 2022. Antagonistic activities of *Lactobacillus rhamnosus* JB3 against *Helicobacter pylori* infection through lipid raft formation. Front. Immunol. 12: 796177.
6. Li, X.H., et al. 2023. BanXiaXieXin decoction treating gastritis mice with drug-resistant *Helicobacter pylori* and its mechanism. World J. Gastroenterol. 29: 2818-2835.

STORAGE

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

RESEARCH USE

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

PROTOCOLS

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