SANTA CRUZ BIOTECHNOLOGY, INC.

Helicobacter pylori (51-13): sc-57778

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Helicobacter pylori is a bacterium that infects the mucus lining of mammalian stomach and duodenum and may cause peptic ulcers, gastritis and duodenitis. It is estimated that about 66% of the world population are infected by the bacterium, though most do not experience symptoms. This spiral-shaped Gram-negative bacterium is unique in that it can thrive in the highly acidic environment of the stomach. Helicobacter pylori can exist in a number of locations: in the mucus; attached to epithelial cells; or inside of vacuoles in epithelial cells, where it produces adhesins that bind to membrane-associated lipids and carbohydrates to help its adhesion to epithelial cells. Helicobacter pylori contains a hydrogenase enzyme and obtains energy by oxidizing molecular hydrogen produced by other intestinal bacteria. It also excretes urease in order to convert urea into ammonia and bicarbonate which neutralizes the acidic environment of the stomach.

REFERENCES

1. Marshall, B.J. and Warren, J.R. 1984. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet 1: 1311-1315.
2. Bode, G., et al. 1994. Ultrastructural localization of urease of *Helicobacter pylori*. Med. Microbiol. Immunol. 182: 233-242.
3. Logan, R.P. and Walker, M.M. 2001. ABC of the upper gastrointestinal tract: epidemiology and diagnosis of *Helicobacter pylori* infection. BMJ 323: 920-922.
4. Olson, J.W. and Maier, R.J. 2002. Molecular hydrogen as an energy source for *Helicobacter pylori*. Science 298: 1788-1790.
5. Tsuji, S., et al. 2003. Review article: inflammation-related promotion of gastrointestinal carcinogenesis—a perigenetic pathway. Aliment. Pharmacol. Ther. 18: 82-89.
6. Konturek, J.W. 2004. Discovery by Jaworski of *Helicobacter pylori* and its pathogenetic role in peptic ulcer, gastritis and gastric cancer. J. Physiol. Pharmacol. 54: 23-41.
7. Viala, J., et al. 2004. Nod1 responds to peptidoglycan delivered by the *Helicobacter pylori* cag pathogenicity island. Nat. Immunol. 5: 1166-1174.
8. Blaser, M.J. 2005. An endangered species in the stomach. Sci. Am. 292: 38-45.
9. Pietroiusti, A., et al. 2005. *Helicobacter pylori* duodenal colonization is a strong risk factor for the development of duodenal ulcer. Aliment. Pharmacol. Ther. 21: 909-915.

SOURCE

Helicobacter pylori (51-13) is a mouse monoclonal antibody raised against *Helicobacter pylori*.

PRODUCT

Each vial contains 100 µg IgG_1 in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

Helicobacter pylori (51-13) is recommended for detection of *Helicobacter Pylori* by immunofluorescence (starting dilution undiluted, dilution range 1:50-1:500); non cross-reactive with other members of the Enterobacteriaceae.

Molecular Weight of Helicobacter pylori precursor: 140 kDa.

Molecular Weight of mature Helicobacter pylori: 95 kDa.

Molecular Weight of Helicobacter pylori cytoxin fragments: 58/37 kDa.

Molecular Weight of Helicobacter pylori outermembrane antigen: 19 kDa.

SELECT PRODUCT CITATIONS

1. Valdmanis, P.N., et al. 2012. Expression determinants of mammalian argonaute proteins in mediating gene silencing. Nucleic Acids Res. 40: 3704-3713.

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.