SANTA CRUZ BIOTECHNOLOGY, INC.

Urease α (bI-17): sc-22444

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Helicobacter pylori is present in the stomachs of at least half of the world's population. Although gastic adenocarcinoma is associated with the presence of *H. pylori* in the stomach, only a small fraction of colonized individuals develop this common malignancy. *H. pylori* urease, an enzyme that generates ammonia and is present within the lamina propria of colonized individuals, binds to class II major histocompatability complex (MHC) molecules on the surfaces of gastric epithelial cells *in vitro*. In addition, Urease, which may be toxic to gastic epithelial cells, may also induce apoptosis. Specifically, Urease plays a crucial role in the development of ulcers in the duodenum by accelerating apoptosis in the antral mucosa. Ammonia accelerates TNFα cytokine-induced apoptosis, while ammonia or urease alone are unable to induce apoptosis. Urease exists as two forms, Urease α (UreA) and Urease β (UreB).

REFERENCES

1. Smoot, D.T. 1997. How does *Helicobacter pylori* cause mucosal damage? Direct mechanisms. Gastroenterology 113: S31-34; discussion S50.
2. Kohda, K., Tanaka, K., Aiba, Y., Yasuda, M., Miwa, T. and Koga, Y. 1999. Role of apoptosis induced by *Helicobacter pylori* infection in the development of duodenal ulcer. Gut 44: 456-462.
3. Fan, X., Gunasena, H., Cheng, Z., Espejo, R., Crowe, S.E., Ernst, P.B. and Reyes, V.E. 2000. *Helicobacter pylori* urease binds to class II MHC on gastric epithelial cells and induces their apoptosis. J. Immunol. 165: 1918-1924.
4. Igarashi, M., Kitada, Y., Yoshiyama, H., Takagi, A., Miwa, T. and Koga, Y. 2001. Ammonia as an accelerator of tumor necrosis factor α-induced apoptosis of gastric epithelial cells in *Helicobacter pylori* infection. Infect. Immunol. 69: 816-821.
5. Kumagai, T., Yan, J., Graham, D.Y., Tozuka, M., Okimura, Y., Ikeno, T., Sugiyama, A., Katsuyama, T. and Ota, H. 2001. Serum immunoglobulin G immune response to *Helicobacter pylori* antigens in Mongolian gerbils. J. Clin. Microbiol. 39: 1283-1288.
6. Peek, R.M., Jr. and Blaser, M.J. 2002. *Helicobacter pylori* and gastrointestinal tract adenocarcinomas. Nat. Rev. Cancer 2: 28-37.
7. Lock, R.A., Coombs, G.W., McWilliams, T.M., Pearman, J.W., Grubb, W.B., Melrose, G.J. and Forbes, G.M. 2002. Proteome analysis of highly immunoreactive proteins of *Helicobacter pylori*. Helicobacter 7: 175-182.

SOURCE

Urease α (bI-17) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of Urease α 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-22444 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

RESEARCH USE

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

APPLICATIONS

Urease α (bI-17) is recommended for detection of Urease α 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 Urease α: 26 kDa.

Positive Controls: *E. coli* extract.

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.

STORAGE

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

PROTOCOLS

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