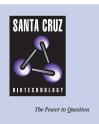
# SANTA CRUZ BIOTECHNOLOGY, INC.

# Urease α (bI-17): sc-22444



# 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 TNF $\alpha$  cytokine-induced apoptosis, while ammonia or urease alone are unable to induce apoptosis. Urease exists as two forms, Urease  $\alpha$  (UreA) and Urease  $\beta$  (UreB).

# REFERENCES

- Smoot, D.T. 1997. How does *Helicobacter pylori* cause mucosal damage? Direct mechanisms. Gastroenterology 113: S31-34; discussion S50.
- 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.
- 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.
- 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.
- 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.
- 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  $\alpha$  (bl-17) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of Urease  $\alpha$  of *H. pylori* origin.

#### PRODUCT

Each vial contains 200  $\mu$ g lgG 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  $\mu$ g 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  $\alpha$  (bl-17) is recommended for detection of Urease  $\alpha$  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  $\alpha$ : 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.