

CagA (A-10): sc-28368

BACKGROUND

Helicobacter pylori is a spiral shaped bacterium that accounts for 80 percent of stomach ulcers and more than 90 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 that of VacA. CagA induces morphological changes in the host, as well as inducing Actin reorganization, variations in the cell cycle and autocrine effects.

SOURCE

CagA (A-10) is a mouse monoclonal antibody raised against amino acids 1-300 of CagA of *H. pylori* origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

CagA (A-10) is available conjugated to agarose (sc-28368 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-28368 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-28368 PE), fluorescein (sc-28368 FITC), Alexa Fluor[®] 488 (sc-28368 AF488), Alexa Fluor[®] 546 (sc-28368 AF546), Alexa Fluor[®] 594 (sc-28368 AF594) or Alexa Fluor[®] 647 (sc-28368 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-28368 AF680) or Alexa Fluor[®] 790 (sc-28368 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

CagA (A-10) is recommended for detection of CagA of *H. pylori* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Molecular Weight of CagA: 120 kDa.

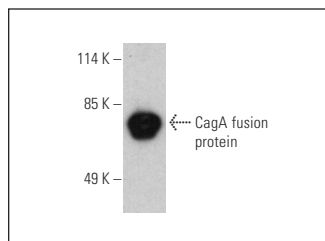
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker[™] Molecular Weight Standards: sc-2035, UltraCruz[®] Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

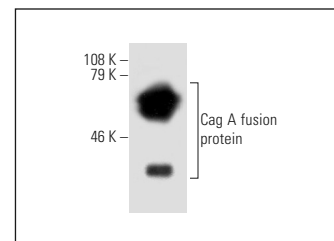
STORAGE

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

DATA



CagA (A-10): sc-28368. Western blot analysis of *H. pylori* recombinant CagA fusion protein. Detection reagent used: m-IgGκ BP-HRP: sc-516102.



CagA (A-10): sc-28368. Western blot analysis of *H. pylori* recombinant CagA fusion protein.

SELECT PRODUCT CITATIONS

- Lin, W.C., et al. 2010. Translocation of *Helicobacter pylori* CagA into human B lymphocytes, the origin of mucosa-associated lymphoid tissue lymphoma. *Cancer Res.* 70: 5740-5748.
- Kuo, S.H., et al. 2013. Detection of the *Helicobacter pylori* CagA protein in gastric mucosa-associated lymphoid tissue lymphoma cells: clinical and biological significance. *Blood Cancer J.* 3: e125.
- Kuo, S.H., et al. 2014. *Helicobacter pylori*-related diffuse large B-cell lymphoma of the stomach: a distinct entity with lower aggressiveness and higher chemosensitivity. *Blood Cancer J.* 4: e220.
- Hartung, M.L., et al. 2015. *H. pylori*-induced DNA strand breaks are introduced by nucleotide excision repair endonucleases and promote NFκB target gene expression. *Cell Rep.* 13: 70-79.
- Zhou, J., et al. 2016. Proteomics-based identification and analysis of proteins associated with *Helicobacter pylori* in gastric cancer. *PLoS ONE* 11: e0146521.
- Kuo, S.H., et al. 2017. First-line antibiotic therapy in *Helicobacter pylori*-negative low-grade gastric mucosa-associated lymphoid tissue lymphoma. *Sci. Rep.* 7: 14333.
- Kokate, S.B., et al. 2018. Acetylation-mediated Siah2 stabilization enhances PHD3 degradation in *Helicobacter pylori*-infected gastric epithelial cancer cells. *FASEB J.* 32: 5378-5389.
- Ohishi, T., et al. 2018. Monotherapy with a novel intervenolin derivative, AS-1934, is an effective treatment for *Helicobacter pylori* infection. *Helicobacter* 23: e12470.
- Zamperone, A., et al. 2019. Inhibition of polarity-regulating kinase PAR1b contributes to *Helicobacter pylori* inflicted DNA double strand breaks in gastric cells. *Cell Cycle* 18: 299-311.

RESEARCH USE

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