SANTA CRUZ BIOTECHNOLOGY, INC.

γ-GCSm (E-4): sc-55586

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

γ-glutamylcysteine synthetase (γ-GCS) is the rate limiting enzyme for glutathione (L-γ-glutamyl-L-cysteinylglycine, GSH) synthesis. GSH is ubiquitous in mammalian cells as a vital intra- and extracellular protective antioxidant. γ-GCS is a heterodimer of a heavy catalytic subunit and a light regulatory subunit that is responsive to inflammation, phenolic antioxidants, heat shock, oxidants and cytokines. The human γ-GCS gene encoding the 367 amino acid catalytic subunit maps to chromosome 6p12. The human γ-GCS gene encoding the regulatory subunit maps to chromosome 1p22.1. The two subunits of γ-GCS form a heterodimeric zinc metalloprotein that gains activity through formation of a reversible disulfide bond.

CHROMOSOMAL LOCATION

Genetic locus: GCLM (human) mapping to 1p22.1; Gclm (mouse) mapping to 3 G1.

SOURCE

γ-GCSm (E-4) is a mouse monoclonal antibody raised against amino acids 1-274 representing full length γ-GCSm of human origin.

PRODUCT

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

γ-GCSm (E-4) is available conjugated to agarose (sc-55586 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-55586 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-55586 PE), fluorescein (sc-55586 FITC), Alexa Fluor® 488 (sc-55586 AF488), Alexa Fluor® 546 (sc-55586 AF546), Alexa Fluor® 594 (sc-55586 AF594) or Alexa Fluor® 647 (sc-55586 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-55586 AF680) or Alexa Fluor® 790 (sc-55586 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

γ-GCSm (E-4) is recommended for detection of γ-GCSm of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for γ-GCSm siRNA (h): sc-40602, γ-GCSm siRNA (m): sc-40603, γ-GCSm shRNA Plasmid (h): sc-40602-SH, γ-GCSm shRNA Plasmid (m): sc-40603-SH, γ-GCSm shRNA (h) Lentiviral Particles: sc-40602-V and γ-GCSm shRNA (m) Lentiviral Particles: sc-40603-V.

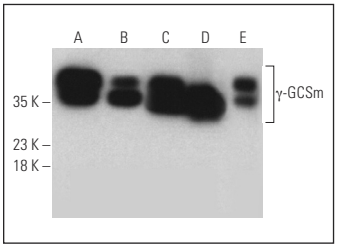
Molecular Weight of γ-GCSm: 31 kDa.

Positive Controls: A549 cell lysate: sc-2413, MOLT-4 cell lysate: sc-2233 or K-562 whole cell lysate: sc-2203.

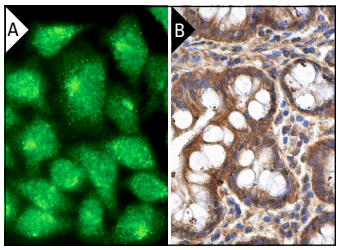
STORAGE

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

DATA



γ-GCSm (E-4): sc-55586. Western blot analysis of γ-GCSm expression in A549 (**A**), MOLT-4 (**B**), K-562 (**C**) and A-673 (**D**) whole cell lysates and K-562 nuclear extract (**E**).



γ-GCSm (E-4): sc-55586. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic and nuclear staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human small intestine tissue showing cytoplasmic staining of glandular cell (**B**).

SELECT PRODUCT CITATIONS

1. Paonessa, J.D., et al. 2009. 5,6-Dihydrocyclopenta[c][1,2]-dithiole-3(4H)-thione is a promising cancer chemopreventive agent in the urinary bladder. Chem. Biol. Interact. 180: 119-126.
2. Zheng, Y., et al. 2012. Sulforaphane prevents pulmonary damage in response to inhaled arsenic by activating the Nrf2-defense response. Toxicol. Appl. Pharmacol. 265: 292-299.
3. Tao, S., et al. 2013. Tanshinone I activates the Nrf2-dependent antioxidant response and protects against As(III)-induced lung inflammation *in vitro* and *in vivo*. Antioxid. Redox Signal. 19: 1647-1661.
4. Tao, S., et al. 2014. Oncogenic KRAS confers chemoresistance by upregulating NRF2. Cancer Res. 74: 7430-7441.
5. Duan, X., et al. 2015. Activation of NRF2 pathway in spleen, thymus as well as peripheral blood mononuclear cells by acute arsenic exposure in mice. Int. Immunopharmacol. 28: 1059-1067.
6. Cholanians, A.B., et al. 2016. From the cover: arsenic induces accumulation of α-synuclein: implications for synucleinopathies and neurodegeneration. Toxicol. Sci. 153: 271-281.
7. Lu, C., et al. 2017. Nrf2 activation is required for ligustrazine to inhibit hepatic steatosis in alcohol-preferring mice and hepatocytes. Toxicol. Sci. 155: 432-443.
8. Liu, P., et al. 2019. Non-covalent NRF2 activation confers greater cellular protection than covalent activation. Cell Chem. Biol. 26: 1427-1435.e5.
9. Liu, P., et al. 2020. NRF2 negatively regulates primary ciliogenesis and hedgehog signaling. PLoS Biol. 18: e3000620.

RESEARCH USE

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