granzyme A (3G8.5): sc-33692



The Power to Question

BACKGROUND

Granzyme A and granzyme B are serine proteases that mediate apoptotic signaling in cytotoxic T lymphocytes (CTL) and natural killer (NK) cells. Both granzyme A and granzyme B are synthesized as inactive proenzymes, and they are stored within cytolytic granules and released by effector cells during degranulation. In activated CTLs, granzyme A and granzyme B are processed and activated by cathepsin C, and they then function to induce apoptosis by two distinct pathways. Granzyme B proteolytically cleaves and activates members of the caspase family of cysteine proteases, including caspase-3, caspase-6, caspase-7 and caspase-9. When cleaved, these caspases assemble into active holoenzymes that then mediate apoptosis through a defined proteolytic cascade involving nuclear lamins and PARP (poly ADP ribose polymerase). Granzyme A mediates the activation of apoptosis by inducing single-strand DNA breaks, membrane perturbation and nuclear condensations in an alternative pathway that is independent from caspase activation or the caspase proteolytic cascade.

CHROMOSOMAL LOCATION

Genetic locus: Gzma (mouse) mapping to 13 D2.2.

SOURCE

granzyme A (3G8.5) is a mouse monoclonal antibody raised against full length recombinant granzyme A of mouse origin.

PRODUCT

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

granzyme A (3G8.5) is available conjugated to agarose (sc-33692 AC), 500 μ g/0.25 ml agarose in 1 ml, for IP; to HRP (sc-33692 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-33692 PE), fluorescein (sc-33692 FITC), Alexa Fluor* 488 (sc-33692 AF488), Alexa Fluor* 546 (sc-33692 AF546), Alexa Fluor* 594 (sc-33692 AF594) or Alexa Fluor* 647 (sc-33692 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor* 680 (sc-33692 AF680) or Alexa Fluor* 790 (sc-33692 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

granzyme A (3G8.5) is recommended for detection of granzyme A of mouse 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)], flow cytometry (1 μg per 1 x 106 cells) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for granzyme A siRNA (m): sc-37432, granzyme A shRNA Plasmid (m): sc-37432-SH and granzyme A shRNA (m) Lentiviral Particles: sc-37432-V.

Molecular Weight of granzyme A monomer: 28 kDa.

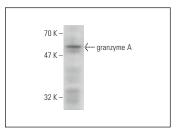
Molecular Weight of granzyme A homodimer: 43-65 kDa.

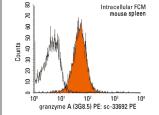
Positive Controls: WEHI-231 whole cell lysate: sc-2213.

STORAGE

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

DATA





granzyme A (3G8.5): sc-33692. Western blot analysis of granzyme A expression in WEHI-231 whole cell lysate.

granzyme A (3G8.5) PE: sc-33692 PE. Intracellular FCM analysis of fixed and permeabilized BALB/c splenocytes. Black line histogram represents the isotype control, normal mouse $\lg G_{2b}$ -PE: sc-2868.

SELECT PRODUCT CITATIONS

- 1. Cao, X., et al. 2007. Granzyme B and perforin are important for regulatory T cell-mediated suppression of tumor clearance. Immunity 27: 635-646.
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- 3. Lucas, M., et al. 2010. Studying NK cell/dendritic cell interactions. Methods Mol. Biol. 612: 97-126.
- Nguyen, M.L., et al. 2016. Dynamic regulation of permissive histone modifications and GATA3 binding underpin acquisition of granzyme A expression by virus-specific CD8+ T cells. Eur. J. Immunol. 46: 307-318.
- 5. Xu, A., et al. 2016. TGF-β-induced regulatory T cells directly suppress B cell responses through a noncytotoxic mechanism. J. Immunol. 196: 3631-3641.
- Aragoneses-Fenoll, L., et al. 2018. T-cell-specific loss of the Pl-3-kinase p110α catalytic subunit results in enhanced cytokine production and antitumor response. Front. Immunol. 9: 332.
- Pais Ferreira, D., et al. 2020. Central memory CD8+ T cells derive from stem-like Tcf7^{hi} effector cells in the absence of cytotoxic differentiation. Immunity 53: 985-1000.e11.
- 8. Mahmoudzadeh, S., et al. 2024. Toxoplasma gondii suppress human cord blood cell differentiation to the NK cell population. Immun. Inflamm. Dis. 12: e1329.

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.