

Gfi-1B (B-7): sc-28356

BACKGROUND

Growth factor independent 1 (Gfi-1) is a transcriptional repressor that specifically binds to the DNA consensus sequence TAAATCAC(A/T)GCA. The carboxy-terminus of Gfi-1 contains six C₂H₂-type zinc finger motifs, and zinc fingers 3, 4 and 5 are required for the binding of Gfi-1 to its DNA binding site. Gfi-1 also contains a 20 amino acid SNAG domain which mediates transcriptional repression. It represses Bax at the mRNA and protein levels, resulting in the inhibition of cell death. Gfi-1 is expressed outside the lymphoid system in granulocytes and activated macrophages. Gfi-1B, a related protein, is a transcriptional repressor primarily expressed in bone marrow and spleen. Gfi-1B is a direct repressor of the p21 promoter and the SOCS-1 and -3 promoters. The genes encoding human Gfi-1 and Gfi-1B map to chromosome 1p22 and 9q34.13, respectively.

CHROMOSOMAL LOCATION

Genetic locus: GFI1B (human) mapping to 9q34.13; Gfi1b (mouse) mapping to 2 A3.

SOURCE

Gfi-1B (B-7) is a mouse monoclonal antibody raised against amino acids 21-170 of Gfi-1B of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available as TransCruz reagent for Gel Supershift and ChIP applications, sc-28356 X, 200 µg/0.1 ml.

Gfi-1B (B-7) is available conjugated to agarose (sc-28356 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-28356 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-28356 PE), fluorescein (sc-28356 FITC), Alexa Fluor® 488 (sc-28356 AF488), Alexa Fluor® 546 (sc-28356 AF546), Alexa Fluor® 594 (sc-28356 AF594) or Alexa Fluor® 647 (sc-28356 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-28356 AF680) or Alexa Fluor® 790 (sc-28356 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

Gfi-1B (B-7) is recommended for detection of Gfi-1B 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Gfi-1B siRNA (h): sc-62374, Gfi-1B siRNA (m): sc-62375, Gfi-1B shRNA Plasmid (h): sc-62374-SH, Gfi-1B shRNA Plasmid (m): sc-62375-SH, Gfi-1B shRNA (h) Lentiviral Particles: sc-62374-V and Gfi-1B shRNA (m) Lentiviral Particles: sc-62375-V.

Gfi-1B (B-7) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

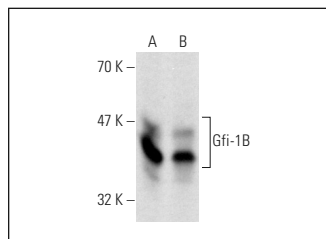
Molecular Weight of Gfi-1B: 41 kDa.

Positive Controls: TF-1 cell lysate: sc-2412 or HEL 92.1.7 cell lysate: sc-2270.

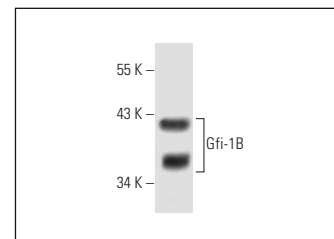
STORAGE

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

DATA



Gfi-1B (B-7): sc-28356. Western blot analysis of Gfi-1B expression in HEL 92.1.7 (A) and TF-1 (B) whole cell lysates.



Gfi-1B (B-7): sc-28356. Western blot analysis of Gfi-1B expression in RBL-1 whole cell lysates.

SELECT PRODUCT CITATIONS

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- Lee, M.C., et al. 2014. Gfi-1 is the transcriptional repressor of SOCS1 in acute myeloid leukemia cells. *J. Leukoc. Biol.* 95: 105-115.
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- Christophersen, M.K., et al. 2017. SMIM1 variants rs1175550 and rs143702418 independently modulate vel blood group antigen expression. *Sci. Rep.* 7: 40451.
- Yamamoto, R., et al. 2018. Selective dissociation between LSD1 and Gfi1B by a LSD1 inhibitor NCD38 induces the activation of ERG super-enhancer in erythroleukemia cells. *Oncotarget* 9: 21007-21021.
- Chen, Y., et al. 2019. Oxymatrine can attenuate pathological deficits of Alzheimer's disease mice through regulation of neuroinflammation. *J. Neuroimmunol.* 334: 576978.
- Tatsumi, G., et al. 2019. LSD1-mediated repression of GFI1 super-enhancer plays an essential role in erythroleukemia. *Leukemia*. E-published.
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RESEARCH USE

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

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