

Neutrophil Elastase (G-2): sc-55549

BACKGROUND

Neutrophil Elastase (NE) is a serine protease that is expressed in bone marrow precursor cells, stored in peripheral blood granulocytes and implicated in the progression of a variety of inflammatory diseases, including idiopathic pulmonary fibrosis, rheumatoid arthritis, adult respiratory distress syndrome and cystic fibrosis. In neutrophils, Neutrophil Elastase contributes largely to the proteolysis of phagocytosed proteins, the migration of neutrophils and the remodeling of tissues following injury. Neutrophil Elastase, which is also designated medullasin, is secreted into the extracellular matrix, where it is then capable of destroying connective tissue proteins, including elastin, proteoglycans and Type IV Collagens. Neutrophil Elastase also mediates proteolysis by cleaving proteins that are associated with the complement system, such as antithrombin and Fibrinogen. Additionally, Neutrophil Elastase functions in secretion and mobilization of calcium in response to cathepsin G binding to platelet surface receptors.

CHROMOSOMAL LOCATION

Genetic locus: ELANE (human) mapping to 19p13.3.

SOURCE

Neutrophil Elastase (G-2) is a mouse monoclonal antibody raised against amino acids 211-267 of Neutrophil Elastase of human origin.

PRODUCT

Each vial contains 200 µg IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Neutrophil Elastase (G-2) is available conjugated to agarose (sc-55549 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-55549 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-55549 PE), fluorescein (sc-55549 FITC), Alexa Fluor® 488 (sc-55549 AF488), Alexa Fluor® 546 (sc-55549 AF546), Alexa Fluor® 594 (sc-55549 AF594) or Alexa Fluor® 647 (sc-55549 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-55549 AF680) or Alexa Fluor® 790 (sc-55549 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

In addition, Neutrophil Elastase (G-2) is available conjugated to biotin (sc-55549 B), 200 µg/ml, for WB, IHC(P) and ELISA.

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APPLICATIONS

Neutrophil Elastase (G-2) is recommended for detection of Neutrophil Elastase of 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).

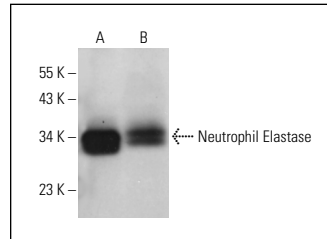
Suitable for use as control antibody for Neutrophil Elastase siRNA (h): sc-36042, Neutrophil Elastase shRNA Plasmid (h): sc-36042-SH and Neutrophil Elastase shRNA (h) Lentiviral Particles: sc-36042-V.

Molecular Weight of Neutrophil Elastase: 29 kDa.

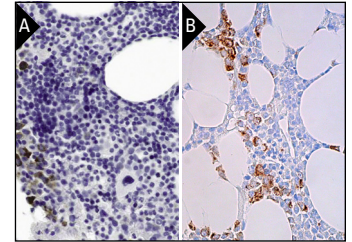
STORAGE

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

DATA



Neutrophil Elastase (G-2): sc-55549. Western blot analysis of Neutrophil Elastase expression in U-937 (A) and HL-60 (B) whole cell lysates.



Neutrophil Elastase (G-2): sc-55549. Immunoperoxidase staining of formalin fixed, paraffin-embedded human bone marrow tissue showing cytoplasmic staining of a subset of bone marrow cells. Kindly provided by The Swedish Human Protein Atlas (HPA) program (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human bone marrow tissue showing cytoplasmic staining of subset of hematopoietic cells (B).

SELECT PRODUCT CITATIONS

1. Franco-Pons, N., et al. 2010. Release of inflammatory mediators by adipose tissue during acute pancreatitis. *J. Pathol.* 221: 175-182.
2. Friedrich, V., et al. 2011. Reduction of neutrophil activity decreases early microvascular injury after subarachnoid haemorrhage. *J. Neuroinflammation* 8: 103.
3. Ueki, S., et al. 2016. Eosinophil extracellular trap cell death-derived DNA traps: their presence in secretions and functional attributes. *J. Allergy Clin. Immunol.* 137: 258-267.
4. Lefrançois, E., et al. 2018. Maladaptive role of neutrophil extracellular traps in pathogen-induced lung injury. *JCI Insight* 3: e98178.
5. Morizawa, Y., et al. 2018. Correlation of immune cells and cytokines in the tumor microenvironment with elevated neutrophil-to-lymphocyte ratio in blood: an analysis of muscle-invasive bladder cancer. *Cancer Invest.* 36: 395-405.
6. Dannenmann, B., et al. 2019. Human iPSC-based model of severe congenital neutropenia reveals elevated UPR and DNA damage in CD34⁺ cells preceding leukemic transformation. *Exp. Hematol.* 71: 51-60.
7. Mallavia, B., et al. 2019. Mitochondrial DNA stimulates TLR9-dependent NET formation in primary graft dysfunction. *Am. J. Respir. Cell Mol. Biol.* 62: 364-372.
8. Ortiz-Muñoz, G., et al. 2020. Cystic fibrosis transmembrane conductance regulator dysfunction in platelets drives lung hyperinflammation. *J. Clin. Invest.* 130: 2041-2053.
9. Cleary, S.J., et al. 2020. Complement activation on endothelium initiates antibody-mediated acute lung injury. *J. Clin. Invest.* E-published.

RESEARCH USE

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