

# N-SMase2 (G-6): sc-166637

## BACKGROUND

N-SMase2 (neutral sphingomyelinase 2), also known as NSMASE2 or SMPD3 (sphingomyelin phosphodiesterase 3), is a ubiquitously expressed 655 amino acid member of the magnesium-dependent phosphohydrolase protein family. Localized to the membrane of the Golgi apparatus, N-SMase2 functions to catalyze the hydrolysis of sphingomyelin to form ceramide and phosphocholine—two proteins that mediate cell growth arrest and apoptosis. N-SMase2 is enzymatically activated by unsaturated fatty acids and phosphatidylserine and, through regulation of ceramide synthesis, is involved in growth suppression and postnatal development. Expression of N-SMase2 is upregulated during the G<sub>0</sub>/G<sub>1</sub> phases of the cell cycle and optimal N-SMase2 activity occurs at a slightly basic pH of 7.5. N-SMase2 deficiency is the cause of chondrodysplasia, a genetic disorder characterized by impaired bone growth that leads to short stature, bowlegs and underdeveloped joints.

## CHROMOSOMAL LOCATION

Genetic locus: SMPD3 (human) mapping to 16q22.1; Smpd3 (mouse) mapping to 8 D3.

## SOURCE

N-SMase2 (G-6) is a mouse monoclonal antibody raised against amino acids 461-655 mapping at the C-terminus of N-SMase2 of human origin.

## PRODUCT

Each vial contains 200 µg IgG<sub>2a</sub> kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

N-SMase2 (G-6) is available conjugated to agarose (sc-166637 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-166637 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-166637 PE), fluorescein (sc-166637 FITC), Alexa Fluor<sup>®</sup> 488 (sc-166637 AF488), Alexa Fluor<sup>®</sup> 546 (sc-166637 AF546), Alexa Fluor<sup>®</sup> 594 (sc-166637 AF594) or Alexa Fluor<sup>®</sup> 647 (sc-166637 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor<sup>®</sup> 680 (sc-166637 AF680) or Alexa Fluor<sup>®</sup> 790 (sc-166637 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

## APPLICATIONS

N-SMase2 (G-6) is recommended for detection of sphingomyelin phosphodiesterase 3 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 N-SMase2 siRNA (h): sc-62655, N-SMase2 siRNA (m): sc-62656, N-SMase2 shRNA Plasmid (h): sc-62655-SH, N-SMase2 shRNA Plasmid (m): sc-62656-SH, N-SMase2 shRNA (h) Lentiviral Particles: sc-62655-V and N-SMase2 shRNA (m) Lentiviral Particles: sc-62656-V.

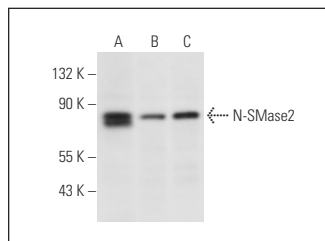
Molecular Weight of N-SMase2: 70 kDa.

Positive Controls: CCRF-CEM cell lysate: sc-2225, Jurkat whole cell lysate: sc-2204 or K-562 whole cell lysate: sc-2203.

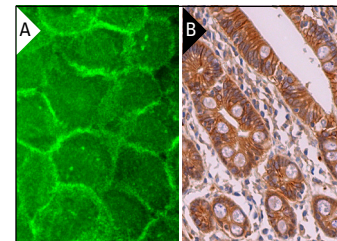
## STORAGE

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

## DATA



N-SMase2 (G-6): sc-166637. Western blot analysis of N-SMase2 expression in CCRF-CEM (A), Jurkat (B) and K-562 (C) whole cell lysates.



N-SMase2 (G-6): sc-166637. Immunofluorescence staining of formalin-fixed A-431 cells showing membrane localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human duodenum tissue showing membrane and cytoplasmic staining of glandular cells (B).

## SELECT PRODUCT CITATIONS

- Cubí, R., et al. 2013. Tetanus toxin Hc fragment induces the formation of ceramide platforms and protects neuronal cells against oxidative stress. *PLoS ONE* 8: e68055.
- Chen, R., et al. 2017. Hepatitis B virus X protein is capable of down-regulating protein level of host antiviral protein APOBEC3G. *Sci. Rep.* 7: 40783.
- Back, M.J., et al. 2018. Activation of neutral sphingomyelinase 2 by starvation induces cell-protective autophagy via an increase in Golgi-localized ceramide. *Cell Death Dis.* 9: 670.
- Poggio, M., et al. 2019. Suppression of exosomal PD-L1 induces systemic anti-tumor immunity and memory. *Cell* 177: 414-427.
- Hitomi, K., et al. 2020. DNA damage regulates senescence-associated extracellular vesicle release via the ceramide pathway to prevent excessive inflammatory responses. *Int. J. Mol. Sci.* 21: 3720.
- Ding, H., et al. 2021. Extracellular vesicles and exosomes generated from cystic renal epithelial cells promote cyst growth in autosomal dominant polycystic kidney disease. *Nat. Commun.* 12: 4548.
- Niekamp, P., et al. 2022. Ca<sup>2+</sup>-activated sphingomyelin scrambling and turnover mediate ESCRT-independent lysosomal repair. *Nat. Commun.* 13: 1875.
- Andreu, Z., et al. 2023. A rapid, convergent approach to the identification of exosome inhibitors in breast cancer models. *Nanotheranostics* 7: 1-21.

## RESEARCH USE

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

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