SANTA CRUZ BIOTECHNOLOGY, INC.

OSM (N-1): sc-129

BACKGROUND

Oncostatin M (OSM) is a glycoprotein that was originally isolated from the conditioned medium of U-937 human histiocytic leukemia cells that had been induced to differentiate into macrophage-like cells by treatment with phorbol 12-myristate 13-acetate. OSM inhibits the growth of a broad range of human tumor cell lines, but does not influence the growth of normal human fibroblasts. High-affinity binding sites for OSM have been detected on normal and tumor cells, and a receptor has been identified by chemical cross-linking studies. Expression of OSM is greatest in activated monocytic and lymphocytic cell lines and in normal adherent macrophages. Amino acid sequence analysis of OSM has revealed homology with leukemia inhibitory factor (LIF), granulocyte colony stimulating factor (G-CSF) and interleukin-6 (IL-6), all of which affect the growth and differentiation of a broad range of cell types, including those of hematopoietic origin.

REFERENCES

1. Zarling, J.M., et al. 1986. Oncostatin M: a growth regulator produced by differentiated histiocytic lymphoma cells. Proc. Natl. Acad. Sci. USA 83: 9739-9743.
2. Brown, T.J., et al. 1987. Purification and characterization of cytostatic lymphokines produced by activated human T lymphocytes. J. Immunol. 139: 2977-2983.

CHROMOSOMAL LOCATION

Genetic locus: OSM (human) mapping to 22q12.2.

SOURCE

OSM (N-1) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping within the N-terminus of OSM of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-129 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

OSM (N-1) is recommended for detection of OSM of human 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)], 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 OSM siRNA (h): sc-39689, OSM shRNA Plasmid (h): sc-39689-SH and OSM shRNA (h) Lentiviral Particles: sc-39689-V.

Molecular Weight of OSM: 28 kDa.

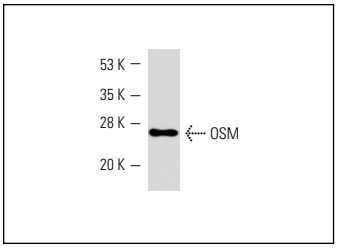
STORAGE

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

RESEARCH USE

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

DATA



OSM (N-1): sc-129. Western blot analysis of 20 ng purified human recombinant OSM using primary antibody at 0.1 µg/ml.

SELECT PRODUCT CITATIONS

1. Jung, M., et al. 1995. Correction of radiation sensitivity in ataxia telangiectasia cells by a truncated IkB-a. Science 268: 1619-1621.
2. García-Tuñón, I., et al. 2008. OSM, LIF, its receptors, and its relationship with the malignance in human breast carcinoma (*in situ* and in infiltrative). Cancer Invest. 26: 222-229.
3. Yu, M., et al. 2008. Interleukin-6 cytokine family member oncostatin M is a hair-follicle-expressed factor with hair growth inhibitory properties. Exp. Dermatol. 17: 12-19.
4. Tsai, C.H., et al. 2008. Immunohistochemical localization of oncostatin M in epithelialized apical periodontitis lesions. Int. Endod. J. 41: 772-776.
5. Kong, N., et al. 2009. Pilot-scale fermentation, purification, and characterization of recombinant human Oncostatin M in *Pichia pastoris*. Protein Expr. Purif. 63: 134-139.
6. Huang, F.M., et al. 2009. The upregulation of oncostatin M in inflamed human dental pulps. Int. Endod. J. 42: 627-631.
7. Kausar, T., et al. 2011. Clinical significance of GPR56, transglutaminase 2, and NFκB in esophageal squamous cell carcinoma. Cancer Invest. 29: 42-48.
8. Kausar, T., et al. 2011. Overexpression of a splice variant of oncostatin M receptor b in human esophageal squamous carcinoma. Cell. Oncol. 34: 177-187.
9. Wang, Y., et al. 2013. Transcription factor KLLN inhibits tumor growth by AR suppression, induces apoptosis by TP53/TP73 stimulation in prostate carcinomas, and correlates with cellular differentiation. J. Clin. Endocrinol. Metab. 98: E586-E594.