3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

Monoclonal Anti-Oncostatin M
clone 17001
produced in mouse, purified immunoglobulin

Catalog Number **O0884**

Synonym: Anti-OSM

Product Description
Monoclonal Anti-Oncostatin M (mouse IgG2a isotype) is purified from a mouse hybridoma elicited from an immunized mouse. Recombinant human OSM (rhOSM), expressed in *E. coli*, was used as immunogen. The antibody is purified by Protein A affinity chromatography.

Anti-Oncostatin M will recognize recombinant human OSM by various immunochemical techniques including neutralization, immunoblotting and ELISA. By ELISA, the antibody does not cross-react with recombinant human IL-6, IL-11, CNTF, LIF and recombinant mouse OSM.

Oncostatin M (OSM) is a growth-regulating cytokine that affects a number of tumor and normal cells. OSM was first identified by its ability to inhibit the growth of A375 melanoma cells and other human tumor cells, but not inhibit the growth of normal human fibroblasts.[1] Recombinant human OSM is produced from a DNA sequence encoding the mature 196 amino acid residue of human OSM. Oncostatin M acts synergistically with TGF-β1 to inhibit the proliferation of A375 melanoma cells.[1] Oncostatin M is secreted by macrophages and activated T lymphocytes. It affects a wide variety of normal and tumor cells. It induces an increase in LDL receptor expression and LDL uptake by hepatoma cells.[2] OSM will induce cultured human endothelial cells to increase IL-6 production.[3] It activates synovial fibroblast-like cells to produce urokinase type plasminogen activator.[4] Oncostatin M, LIF, G-CSF, IL-6 and ciliary neurotrophic factor (CNTF) are structurally related members of the same cytokine family sharing similarities in their primary amino acid sequences, predicted secondary structure, and receptor components.[6]

Reagent
Supplied as a lyophilized powder from phosphate buffered saline (PBS), pH 7.4, with 5% trehalose.

Preparation Instructions
To one vial of lyophilized powder, add 1 ml of 0.2 μm-filtered PBS to produce a 500 μg/ml stock solution.

Storage/Stability
Prior to reconstitution, store at –20 °C. Reconstituted product may be stored at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots at –20 °C. Avoid repeated freezing and thawing.

Product Profile
Neutralization: Monoclonal Anti- Oncostatin M is tested for its ability to neutralize the biological activity of rhOSM on the human TF-1 cell line.[7] In this bioassay, rhOSM was pre-mixed with various dilutions of the antibody for 1 hour at 37 °C.

The antigen-antibody mixture was then added to cultures TF-1 cells in a 96-well plate. The assay mixture was incubated at 37 °C for 48 hours in a humidified CO_2 incubator. ^{3}H-thymidine was added during the final 4 hour incubation. The cells were harvested and ^{3}H-thymidine incorporated into DNA was measured.

The Neutralization $Dose_{50}$ (ND_{50}) is 0.5-2 μg/mL in the presence of 2 ng/ml of recombinant human OSM, using the human TF-1 cell line.

The ND_{50} of the antibody is defined as the concentration of antibody resulting in a one-half maximal inhibition of bioactivity of rhOSM that is present at a concentration just high enough to elicit a maximum response.

The exact concentration of antibody required to neutralize recombinant human OSM activity is dependent on the cytokine concentration, cell type, growth conditions, and the type of activity studied.

Immunoblotting: a working concentration of 1 µg/mL antibody is recommended.

Capture ELISA: Monoclonal Anti-OSM can be used as the capture antibody. Use at a working concentration of 2-8 µg/mL in combination with a biotinylated human Oncostatin M/OSM detection antibody (0.1-0.4 µg/mL).

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working dilutions by titration test.

Endotoxin level is < 0.1 EU/µg antibody as determined by the LAL (Limulus amebocyte lysate) method.

References

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5. Rose, T., et al., *Proc. Natl. Acad. Sci. USA*, **88**, 8641 (1991).
6. Bazan, J., et al., *Neuron*, **7**, 197 (1991).
7. Kitamura, T., et al., *J. Cell Physiol*., **140**, 323 (1989).

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