

# **Human Oncostatin M/OSM Antibody**

Polyclonal Goat IgG Catalog Number: AB-295-NA

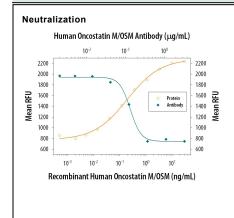
DESCRIPTION		
Species Reactivity	Human	
Specificity	Detects human Oncostatin M/OSM in direct ELISAs and Western blots. In direct ELISAs, less than 1% cross-reactivity with recombinant mouse OSM is observed.	
Source	Polyclonal Goat IgG	
Purification	Protein A or G purified	
Immunogen	E. coli-derived recombinant human Oncostatin M/OSM Ala26-Arg221 Accession # P13725	
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.	
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.	

### **APPLICATIONS**

Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

	Recommended Concentration	Sample	
Western Blot	1 μg/mL	Recombinant Human Oncostatin M/OSM (Catalog # 295-OM)	
Neutralization	Measured by its ability to neutralize Oncostatin M/OSM-induced proliferation in the TF-1 human erythroleukemic cell line. Kitamura, T. et al. (1989) J. Cell Physiol. <b>140</b> :323. The Neutralization Dose (ND <sub>50</sub> ) is typically		
	0.2-0.4 μg/mL in the	e presence of 2 ng/mL Recombinant Human Oncostatin M/OSM.	

#### DATA



Cell Proliferation Induced by Oncostatin M/OSM and Neutralization by Human Oncostatin M/OSM Antibody. Recombinant Human Oncostatin M/OSM (Catalog # 295-OM) stimulates proliferation in the TF-1 human erythroleukemic cell line in a dosedependent manner (orange line). Proliferation elicited by Recombinant Human Oncostatin M/OSM (2 ng/mL) is neutralized (green line) by increasing concentrations of Goat Anti-Human Oncostatin M/ OSM Polyclonal Antibody (Catalog # AB-295-NA). The ND<sub>so</sub> is typically 0.2-0.4 µg/mL

### PREPARATION AND STORAGE

Reconstitution Reconstitute at 1 mg/mL in sterile PBS.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below

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- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 6 months, -20 to -70 °C under sterile conditions after reconstitution

## BACKGROUND

OSM is a cytokine originally isolated from medium conditioned by PMA-treated U-937 human histiocytic leukemia cells based on its ability to inhibit growth of A375 melanoma cells. The human OSM cDNA encodes a 252 amino acid pre-pro-OSM polypeptide with a 25 residue hydrophobic signal peptide and a hydrophilic C-terminal domain that are proteolytically processed to generate the 196 residue mature form of OSM. Although both mature and pro-OSM are equally active in radio-receptor assays, the mature OSM is 5- to 60-fold more active in growth inhibition assays. Thus, proteolytic processing of the pro-OSM peptide may be important in regulating the *in vivo* activities of OSM.

OSM is a pleiotropic cytokine that initiates its biological activities by binding to specific cell surface receptors. The gp130, a signal transducing component ( $\beta$  subunit) of the IL-6, LIF and CNTF receptor complexes, was identified as a low-affinity OSM receptor that does not transduce OSM signals. The low affinity LIF receptor (LIF R, a gp130-related protein) has now been identified to be a component of a high-affinity OSM receptor that will transduce OSM signals. Since OSM is also active on cells that do not express LIF R, a specific OSM receptor that does not involve LIF R must also exist. Besides its growth inhibitory activities on human A375 melanoma and mouse M1 myeloid leukemic cells, as well as on other solid tumor cells, OSM also has growth stimulatory activities on normal fibroblasts,

AIDS-Kaposi's sarcoma cells, and a human erythroleukemia cell line, TF-1. Other OSM-mediated activities reported to date include: stimulation of plasminogen activator activity in cultured bovine aortic endothelial cells; regulation of IL-6 expression in human endothelial cells; and stimulation of LDL uptake and up-regulation of cell surface LDL receptors in HepG2 cells.

**K&D**SYSTEMS