

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus)
Purified Goat Polyclonal Antibody
Catalog # AF4254a

Specification

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) - Product Information

Application	WB
Primary Accession	P78380
Other Accession	NP_002534.1 , NP_001166103.1 , NP_001166104.1
Reactivity	Human
Predicted	Human
Host	Goat
Clonality	Polyclonal
Concentration	0.5
Calculated MW	30959

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) - Additional Information

Gene ID 4973

Other Names

OLR1; oxidized low density lipoprotein (lectin-like) receptor 1; CLEC8A; LOX1; LOXIN; SCARE1; SLOX1; C-type lectin domain family 8 member A; OTTHUMP00000238950; OTTHUMP00000238954; OTTHUMP00000238955; OTTHUMP00000238958; hLOX-1; lectin-type oxidized LDL r

Format

Supplied at 0.5 mg/ml in Tris saline, 0.02% sodium azide, pH7.3 with 0.5% bovine serum albumin. Aliquot and store at -20°C. Minimize freezing and thawing.

Immunogen

Peptide with sequence DLKIQTVKQDPDEK-C, from the N Terminus of the protein sequence according to [NP_002534.1](#); [NP_001166103.1](#); [NP_001166104.1](#).

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) - Protein Information

Name OLR1

Synonyms CLEC8A, LOX1

Function

Receptor that mediates the recognition, internalization and degradation of oxidatively modified low density lipoprotein (oxLDL) by vascular endothelial cells. OxLDL is a marker of atherosclerosis that induces vascular endothelial cell activation and dysfunction, resulting in pro-inflammatory responses, pro-oxidative conditions and apoptosis. Its association with oxLDL induces the activation of NF-kappa-B through an increased production of intracellular reactive oxygen and a variety of pro-atherogenic cellular responses including a reduction of nitric oxide (NO) release, monocyte adhesion and apoptosis. In addition to binding oxLDL, it acts as a receptor for the HSP70 protein involved in antigen cross-presentation to naive T-cells in dendritic cells, thereby participating in cell-mediated antigen cross-presentation. Also involved in inflammatory process, by acting as a leukocyte-adhesion molecule at the vascular interface in endotoxin-induced inflammation. Also acts as a receptor for advanced glycation end (AGE) products, activated platelets, monocytes, apoptotic cells and both Gram-negative and Gram-positive bacteria.

Cellular Location

Cell membrane; Lipid-anchor. Cell membrane; Single-pass type II membrane protein. Membrane raft. Secreted. Note=A secreted form also exists. Localization to membrane rafts requires palmitoylation

Tissue Location

Expressed at high level in endothelial cells and vascular-rich organs such as placenta, lung, liver and brain, aortic intima, bone marrow, spinal cord and substantia nigra. Also expressed at the surface of dendritic cells. Widely expressed at intermediate and low level.

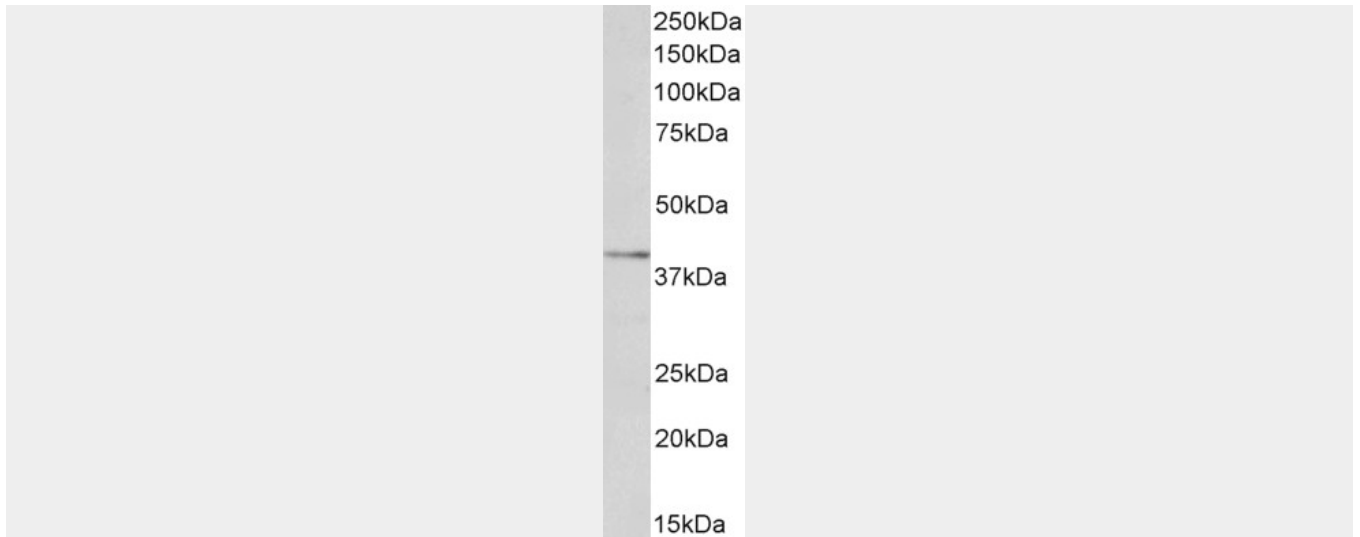
Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) - Images





AF4254a (0.1 µg/ml) staining of Human Liver lysate (35 µg protein in RIPA buffer). Primary incubation was 1 hour. Detected by chemiluminescence.

Goat Anti-ORL1 / LOX1 / LOXIN Antibody (N Terminus) - References

Oxidized low-density lipoprotein activates p66Shc via lectin-like oxidized low-density lipoprotein receptor-1, protein kinase C-beta, and c-Jun N-terminal kinase kinase in human endothelial cells. Shi Y, Cosentino F, Camici GG, Akhmedov A, Vanhoutte PM, Tanner FC, Lüscher TF. *Arterioscler Thromb Vasc Biol.* 2011 Sep;31(9):2090-7.