

DESCRIPTION

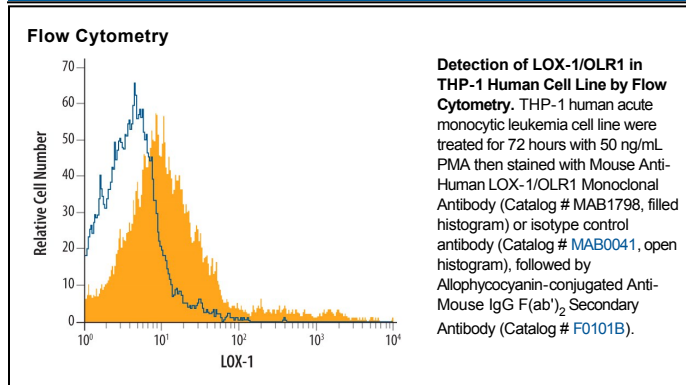
Species Reactivity	Human
Specificity	Detects human LOX-1 in direct ELISAs and Western blots. In direct ELISAs and Western blots, this antibody does not cross-react with recombinant mouse LOX-1.
Source	Monoclonal Mouse IgG _{2B} Clone # 331212
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human LOX-1/OLR1 Ser61-Gln273 Accession # P78380
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. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

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 LOX-1/OLR1 (Catalog # 1798-LX)
Flow Cytometry	2.5 µg/10 ⁶ cells	See Below
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	
Blockade of Receptor-ligand Interaction	In a functional ELISA, 0.3-1.2 µg/mL of this antibody will block 50% of the binding of 1 µg/mL of biotinylated AGE-BSA to immobilized Recombinant Human LOX-1/OLR1 (Catalog # 1798-LX) coated at 5 µg/mL (100 µL/well). At 10 µg/mL, this antibody will block >90% of the binding.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 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

Lectin-like oxidized low-density-lipoprotein receptor-1 (LOX-1), also known as oxidized low-density-lipoprotein receptor-1 (OLR-1), is a type II transmembrane receptor belonging to the C-type lectin family (1). It also belongs to the functionally defined scavenger receptor (SR) superfamily, whose members share the common ability to bind and internalize modified forms of Low Density Lipoproteins (LDL) (2-4). LOX-1 is the first member of the class E scavenger receptor subfamily (SR-E). It binds and supports the internalization of multiple structurally unrelated macromolecules including oxidized LDL, advanced glycation end products (AGE), activated platelets, bacteria, apoptotic or aged cells, and heat shock proteins (5-7). LOX-1 has also been implicated as an intestinal receptor involved in the transcytosis of pancreatic bile salt-dependent lipase (8). The human LOX-1 gene encodes a 273 amino acid (aa) residue protein with a short N-terminal intracellular domain, a transmembrane domain, an extracellular stalk/neck region followed by a C-type lectin-like domain (CTLD) (1, 6). The CTLD, which is required for ligand recognition, contains the six conserved cysteine residues present in all C-type lectins, but lacks the Ca²⁺-binding residues found in classical C-type lectins. LOX-1 can be detected on activated endothelial cells, vascular smooth muscle cells, macrophages, intestinal cells, and dendritic cells (6-8). The expression of LOX-1 is induced by proinflammatory or proatherogenic stimuli, as well as by oxidized LDL itself and hemodynamic or oxidative stress. Human LOX-1 exists on the cell surface as covalent homodimers, which can further associate into non-covalent-linked oligomers (9). Cell surface LOX-1 can also be cleaved by yet unidentified proteases to release the soluble LOX-1 extracellular domain (6). Binding and endocytosis of oxidized LDL by LOX-1 induces oxidative stress, activates NFκB, and upregulates the expression of monocyte chemoattractant protein-1 and matrix metalloproteases (5-9). LOX-1-dependent oxidized LDL uptake also induces apoptosis by inducing the expression of the pro-apoptotic Bax and downregulation of the anti-apoptotic Bcl-2 (10). Oxidized LDL plays a key role in the pathogenesis of atherosclerosis and endothelial dysfunction. Blockade of LOX-1 functions may turn out to be a suitable target for the therapeutic intervention of atherosclerosis.

References:

1. Sawamura, T. *et al.* (1997) *Nature* **386**:73.
2. Daugherty, A. *et al.* (2000) *Curr. Opin. Cardiovasc. Pulm. Ren. Invest. Drugs.* **2**:223.
3. Platt, N. and S. Gordon (2001) *J. Clin. Invest.* **108**:649.
4. Platt, N. and S. Gordon (1998) *Chem. Biol.* **5**:R193.
5. Jono, T. *et al.* (2002) *FEBS Lett.* **511**:170.
6. Kume, N. *et al.* (2001) *Curr. Opin. Lipidol.* **12**:419.
7. Delneste, Y. *et al.* (2002) *Immunity* **17**:353.
8. Bruneau, N. *et al.* (2003) *Mol. Biol. Cell* **14**:2861.
9. Xie, Q. *et al.* (2004) *DNA and Cell Biol.* **23**:111.
10. Chen, J. *et al.* (2003) *Circ. Res.* **94**:370.