Mouse LOX-1/OLR1 Antibody

Monoclonal Rat IgG_{2B} Clone # 214001

Catalog Number: MAB15641

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse LOX-1/OLR1 in ELISAs. In sandwich immunoassays, no cross-reactivity or interference with recombinant human LOX-1 or human LDL, HDL or VLDL is observed.
Source	Monoclonal Rat IgG_{2B} Clone # 214001
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse LOX-1/OLR1 Arg60-Ile363 Accession # AAG44998
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 either lyophilized or 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.*

Mouse LOX-1/OLR1 Sandwich Immunoassay		**Reagent**
ELISA Capture	2-8 µg/mL	Mouse LOX-1/OLR1 Antibody (Catalog # MAB15641)
ELISA Detection	0.1-0.4 µg/mL	Mouse LOX-1/OLR1 C-Terminus Biotinylated Antibody (Catalog # BAF1564)
Standard		Recombinant Mouse LOX-1/OLR1 (Catalog # 1564-LX)

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.** ● 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 mouse LOX-1 gene encodes a 363 amino acid (aa) protein with a short N-terminal intracellular domain, a transmembrane domain, triple repeats of an extracellular stalk/neck region followed by a C-type lectin-like domain (CTLD) (11). The CTLD, which is required for ligand recognition, contains the six conserved cysteine residues present in all C-type lectins, but lacks the Ca^{2+}-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. 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 up-regulates 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 down-regulation 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:

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