

LECT1 Antibody (N-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP2729a**Specification**

LECT1 Antibody (N-term) - Product Information

Application	WB,E
Primary Accession	O75829
Other Accession	O77770 , P17404
Reactivity	Human, Mouse, Rat
Predicted	Bovine, Rabbit
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	1-30

LECT1 Antibody (N-term) - Additional Information**Gene ID** 11061**Other Names**

Leukocyte cell-derived chemotaxin 1, Chondrosurfactant protein, CH-SP, Chondromodulin-1, Chondromodulin-I, ChM-I, LECT1, CHMI

Target/Specificity

This LECT1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1-30 amino acids from the N-terminal region of human LECT1.

Dilution

WB~~1:2000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

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

Precautions

LECT1 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

LECT1 Antibody (N-term) - Protein Information**Name** CNMD ([HGNC:17005](#))

Function Bifunctional growth regulator that stimulates the growth of cultured chondrocytes in the presence of basic fibroblast growth factor (FGF) but inhibits the growth of cultured vascular endothelial cells. May contribute to the rapid growth of cartilage and vascular invasion prior to the replacement of cartilage by bone during endochondral bone development. Inhibits in vitro tube formation and mobilization of endothelial cells. Plays a role as antiangiogenic factor in cardiac valves to suppress neovascularization.

Cellular Location

[Chondromodulin-1]: Secreted, extracellular space, extracellular matrix. Note=Accumulated in the inter-territorial matrix of cartilage

Tissue Location

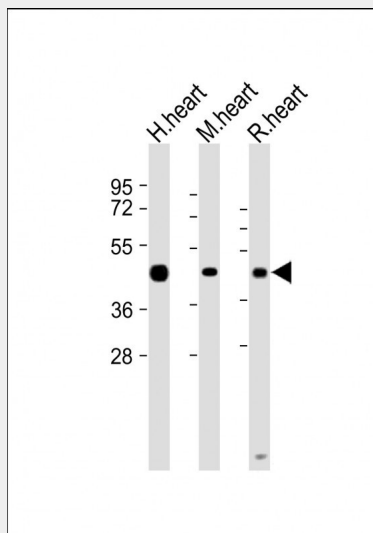
Detected in cartilage and cardiac valves (at protein level). Detected in the laminae fibrosa, spongiosa and ventricularis layers of normal cardiac valves (at protein level) Expression is decreased cardiac valves of patients with valvular heart disease (at protein level). Weakly expressed in chondrosarcoma

LECT1 Antibody (N-term) - 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](#)

LECT1 Antibody (N-term) - Images



All lanes : Anti-LECT1 Antibody (N-term) at 1:2000 dilution Lane 1: Human heart lysate Lane 2: Mouse heart lysate Lane 3: Rat heart lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 37 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

LECT1 Antibody (N-term) - Background

LECT1 is a glycosylated transmembrane protein that is cleaved to form a mature, secreted protein. The N-terminus of the precursor protein shares characteristics with other surfactant proteins and is sometimes called chondrosurfactant protein although no biological activity has yet been defined for it. The C-terminus of the precursor protein contains a 25 kDa mature protein called leukocyte cell-derived chemotaxin-1 or chondromodulin-1. The mature protein promotes chondrocyte growth and inhibits angiogenesis. This protein expressed in the avascular zone of prehypertrophic cartilage and its expression decreases during chondrocyte hypertrophy and vascular invasion. The mature protein likely plays a role in endochondral bone development by permitting cartilaginous anlagen to be vascularized and replaced by bone. It may be involved also in the broad control of tissue vascularization during development.

LECT1 Antibody (N-term) - References

Aoyama,T., Biochem. Biophys. Res. Commun. 365 (1), 124-130 (2008)
Yoshioka,M., Nat. Med. 12 (10), 1151-1159 (2006)
Aoyama,T., J. Biol. Chem. 279 (27), 28789-28797 (2004)