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Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide

Synthetic peptide
Catalog # BP2016a

Specification

Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - Product Information

Primary Accession	Q86YL7
Other Accession	NP_006465

Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - Additional Information

Gene ID 10630

Other Names
Podoplanin, Aggrus, Glycoprotein 36, Gp36, PA226 antigen, T1-alpha, T1A, PDPN {ECO:0000312|EMBL:AAH146682}

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP2016a was selected from the N-term region of human T1A-2 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - Protein Information

Name PDPN {ECO:0000312|EMBL:AAH14668.2}

Function
Mediates effects on cell migration and adhesion through its different partners. During development plays a role in blood and lymphatic vessels separation by binding CLEC1B, triggering CLEC1B activation in platelets and leading to platelet activation and/or aggregation (PubMed:14522983, PubMed:15231832, PubMed:17616532, PubMed:18215137, PubMed:17222411). Interaction with CD9, on the contrary, attenuates platelet aggregation induced by PDPN (PubMed:18541721). Through

MSN or EZR interaction promotes epithelial- mesenchymal transition (EMT) leading to ERZ phosphorylation and triggering RHOA activation leading to cell migration increase and invasiveness (PubMed:17046996, PubMed:21376833). Interaction with CD44 promotes directional cell migration in epithelial and tumor cells (PubMed:20962267). In lymph nodes (LNs), controls fibroblastic reticular cells (FRCs) adhesion to the extracellular matrix (ECM) and contraction of the actomyosin by maintaining ERM proteins (EZR; MSN and RDX) and MYL9 activation through association with unknown transmembrane proteins. Engagement of CLEC1B by PDPN promotes FRCs relaxation by blocking lateral membrane interactions leading to reduction of ERM proteins (EZR; MSN and RDX) and MYL9 activation (By similarity). Through binding with LGALS8 may participate in connection of the lymphatic endothelium to the surrounding extracellular matrix (PubMed:19268462). In keratinocytes, induces changes in cell morphology showing an elongated shape, numerous membrane protrusions, major reorganization of the actin cytoskeleton, increased motility and decreased cell adhesion (PubMed:15515019). Controls invadopodia stability and maturation leading to efficient degradation of the extracellular matrix (ECM) in tumor cells through modulation of RHOC activity in order to activate ROCK1/ROCK2 and LIMK1/LIMK2 and inactivation of CFL1 (PubMed:25486435). Required for normal lung cell proliferation and alveolus formation at birth (By similarity). Does not function as a water channel or as a regulator of aquaporin-type water channels (PubMed:9651190). Does not have any effect on folic acid or amino acid transport (By similarity).

Cellular Location
[Podoplanin]: Membrane; Single-pass type I membrane protein {ECO:0000250|UniProtKB:Q62011}. Cell projection, lamellipodium membrane; Single-pass type I membrane protein {ECO:0000250|UniProtKB:Q62011}. Cell projection, filopodium membrane; Single- pass type I membrane protein {ECO:0000250|UniProtKB:Q62011}. Cell projection, microvillus membrane; Single- pass type I membrane protein {ECO:0000250|UniProtKB:Q62011}. Cell projection, ruffle membrane; Single-pass type I membrane protein {ECO:0000250|UniProtKB:Q62011}. Membrane raft. Apical cell membrane. Basolateral cell membrane. Cell projection, invadopodium. Note=Localized to actin-rich microvilli and plasma membrane projections such as filopodia, lamellipodia and ruffles (By similarity). Association to the lipid rafts is required for PDPN-induced epithelial to mesenchymal transition (EMT) (PubMed:21376833). Colocalizes with CD9 in tetraspanin microdomains (PubMed:18541721). Localized at invadopodium adhesion rings in tumor cell. Association to the lipid rafts is essential for PDPN recruitment to invadopodia and ECM degradation (PubMed:25486435) {ECO:0000250|UniProtKB:Q62011, ECO:0000269|PubMed:18541721, ECO:0000269|PubMed:21376833, ECO:0000269|PubMed:25486435}

Tissue Location
Highly expressed in placenta, lung, skeletal muscle and brain. Weakly expressed in brain, kidney and liver. In placenta, expressed on the apical plasma membrane of endothelium. In lung, expressed in alveolar epithelium. Up-regulated in colorectal tumors and expressed in 25% of early oral squamous cell carcinomas

Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - Images

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Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - Background

Expressed at the plasma membrane in epithelia involved in fluid transport, including type I alveolar epithelial cells, choroid plexus, and ciliary epithelium, the specific function of this protein has not been determined but it has been proposed as a marker of lung injury. T1A-2 has a broad tissue distribution with strong expression in lung, placenta and skeletal muscle. The physiological function of this protein may be related to its mucin-type character. The homologous protein in other species has been described as a differentiation antigen and influenza-virus receptor.

Podoplanin / GP36 /TIA-2 Antibody (N-term) Blocking peptide - References

Kato, Y., et al., J. Biol. Chem. 278(51):51599-51605 (2003).Ma, T., et al., Am. J. Respir. Cell Mol. Biol. 19(1):143-149 (1998).Zimmer, G., et al., Biochem. J. 341 (Pt 2), 277-284 (1999).