

Anti-Tie-2 antibody



Description Rabbit polyclonal to Tie-2.

Model STJ96015

Host Rabbit

Reactivity Human, Mouse

Applications ELISA, IHC

Immunogen Synthesized peptide derived from human Tie-2 around the non-

phosphorylation site of Y1102.

Immunogen Region 1040-1120 aa

Gene ID <u>7010</u>

Gene Symbol <u>TEK</u>

Dilution range IHC 1:100-1:300ELISA 1:5000

Specificity Tie-2 Polyclonal Antibody detects endogenous levels of Tie-2 protein.

Tissue Specificity Detected in umbilical vein endothelial cells. Proteolytic processing gives rise

to a soluble extracellular domain that is detected in blood plasma (at protein level). Predominantly expressed in endothelial cells and their progenitors, the angioblasts. Has been directly found in placenta and lung, with a lower level

in umbilical vein endothelial cells, brain and kidney.

Purification The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.

Note For Research Use Only (RUO).

Protein Name Angiopoietin-1 receptor Endothelial tyrosine kinase Tunica interna endothelial

cell kinase Tyrosine kinase with Ig and EGF homology domains-2 Tyrosine-protein kinase receptor TEK Tyrosine-protein kinase receptor TIE-2 h

Molecular Weight 120 kDa

Clonality Polyclonal

Conjugation Unconjugated

Isotype IgG

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Concentration 1 mg/ml

Storage Instruction Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links <u>HGNC:11724OMIM:600195</u>

Alternative Names Angiopoietin-1 receptor Endothelial tyrosine kinase Tunica interna endothelial

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Function Tyrosine-protein kinase that acts as cell-surface receptor for ANGPT1,

ANGPT2 and ANGPT4 and regulates angiogenesis, endothelial cell survival, proliferation, migration, adhesion and cell spreading, reorganization of the actin cytoskeleton, but also maintenance of vascular quiescence. Has antiinflammatory effects by preventing the leakage of proinflammatory plasma proteins and leukocytes from blood vessels. Required for normal angiogenesis and heart development during embryogenesis. Required for post-natal hematopoiesis. After birth, activates or inhibits angiogenesis, depending on the context. Inhibits angiogenesis and promotes vascular stability in quiescent vessels, where endothelial cells have tight contacts. In quiescent vessels, ANGPT1 oligomers recruit TEK to cell-cell contacts, forming complexes with TEK molecules from adjoining cells, and this leads to preferential activation of phosphatidylinositol 3-kinase and the AKT1 signaling cascades. In migrating endothelial cells that lack cell-cell adhesions, ANGT1 recruits TEK to contacts with the extracellular matrix, leading to the formation of focal adhesion complexes, activation of PTK2/FAK and of the downstream kinases MAPK1/ERK2 and MAPK3/ERK1, and ultimately to the stimulation of sprouting angiogenesis. ANGPT1 signaling triggers receptor dimerization and autophosphorylation at specific tyrosine residues that then serve as binding sites for scaffold proteins and effectors. Signaling is modulated by ANGPT2 that has lower affinity for TEK, can promote TEK autophosphorylation in the absence of ANGPT1, but inhibits ANGPT1-mediated signaling by competing for the same binding site. Signaling is also modulated by formation of heterodimers with TIE1, and by proteolytic processing that gives rise to a soluble TEK extracellular domain. The soluble extracellular domain modulates signaling by functioning as decoy receptor for angiopoietins. TEK phosphorylates DOK2, GRB7, GRB14, PIK3R1; SHC1 and TIE1.

Sequence and Domain Family

The soluble extracellular domain is functionally active in angiopoietin binding and can modulate the activity of the membrane-bound form by competing for angiopoietins.

Cellular Localization

Cell membrane Cell junction, focal adhesion Cytoplasm, cytoskeleton. Secreted. Recruited to cell-cell contacts in quiescent endothelial cells. Colocalizes with the actin cytoskeleton and at actin stress fibers during cell

Post-translational Modifications

spreading. Recruited to the lower surface of migrating cells, especially the rear end of the cell. Proteolytic processing gives rise to a soluble extracellular domain that is secreted.

Proteolytic processing leads to the shedding of the extracellular domain (soluble TIE-2 alias sTIE-2). Autophosphorylated on tyrosine residues in response to ligand binding. Autophosphorylation occurs in trans, i.e. one subunit of the dimeric receptor phosphorylates tyrosine residues on the other subunit. Autophosphorylation occurs in a sequential manner, where Tyr-992 in the kinase activation loop is phosphorylated first, followed by autophosphorylation at Tyr-1108 and at additional tyrosine residues. ANGPT1-induced phosphorylation is impaired during hypoxia, due to increased expression of ANGPT2. Phosphorylation is important for interaction with GRB14, PIK3R1 and PTPN11. Phosphorylation at Tyr-1102 is important for interaction with SHC1, GRB2 and GRB7. Phosphorylation at Tyr-1108 is important for interaction with DOK2 and for coupling to downstream signal transduction pathways in endothelial cells. Dephosphorylated by PTPRB. Ubiquitinated. The phosphorylated receptor is ubiquitinated and internalized, leading to its degradation.

St John's Laboratory Ltd

F +44 (0)207 681 2580

T +44 (0)208 223 3081

W http://www.stjohnslabs.com/ E info@stjohnslabs.com