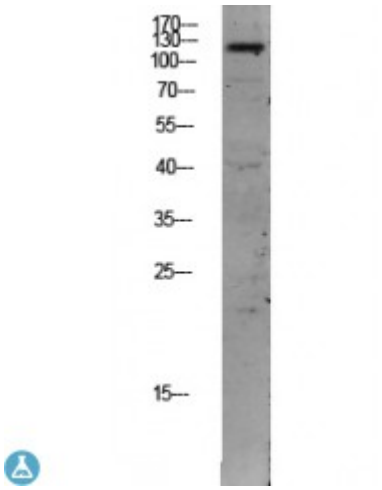
Anti-TIE2 antibody



Description	Rabbit polyclonal to TIE2.

Model	STJ99610
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, WB
Immunogen	Synthesized peptide derived from human TIE2.
Gene ID	7010
Gene Symbol	TEK
Dilution range	WB 1:500-2000ELISA 1:10000-20000
Specificity	This antibody detects endogenous levels of TIE2.
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	HGNC:11724OMIM:600195
Alternative Names	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
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 anti-inflammatory 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 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 .

Post-translational Modifications	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.