10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

# Mouse Tek Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP21675b

## Product Information

| | |
|---|---|
| **Application** | WB, E |
| **Primary Accession** | Q02858 |
| **Reactivity** | Human, Mouse |
| **Predicted** | Rat, Bovine, Canine, Rabbit, Chicken |
| **Host** | Rabbit |
| **Clonality** | polyclonal |
| **Isotype** | Rabbit IgG |
| **Clone Names** | RB50277 |
| **Calculated MW** | 125701 |

## Additional Information

| | |
|---|---|
| **Gene ID** | 21687 |
| **Other Names** | Angiopoietin-1 receptor, Endothelial tyrosine kinase, HYK, STK1, 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, mTIE2, p140 TEK, CD202b, Tek, Hyk, Tie-2, Tie2 |
| **Target/Specificity** | This Mouse Tek antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between782-815 amino acids from the C-terminal region of Mouse Tek. |
| **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** | Mouse Tek Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures. |

## Protein Information

| | |
|---|---|
| **Name** | Tek |
| **Synonyms** | Hyk, Tie-2, Tie2 |

| | |
|---|---|
| **Function** | Tyrosine-protein kinase that acts as a 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 pro-inflammatory plasma proteins and leukocytes from blood vessels. Required for normal angiogenesis and heart development during embryogenesis. Required for postnatal 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. |
| **Cellular Location** | Cell membrane; Single-pass type I membrane protein. Cell junction. Cell junction, focal adhesion. Cytoplasm, cytoskeleton. Secreted. Note=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 (By similarity). |
| **Tissue Location** | Specifically expressed in developing vascular endothelial cells. Abundantly expressed in lung and heart, moderately in brain, liver and kidney, and weakly in thymus, spleen and testis |

## Background

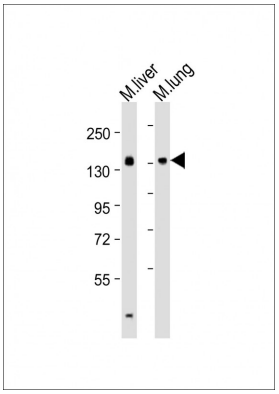
Tyrosine-protein kinase that acts as cell-surface receptor for ANGPT1, ANGPT2 and ANGPT4 and regulates angiogenesis, endothelial cell survival, proliferation, migration, adhesion and cell spreading, reorganization of the actin cytoskeleton, but also maintenance of vascular quiescence. Has anti-inflammatory 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.

## References


Sato T.N.,et al.Proc. Natl. Acad. Sci. U.S.A. 90:9355-9358(1993).
Dumont D.J.,et al.Oncogene 8:1293-1301(1993).
Horita K.,et al.Biochem. Biophys. Res. Commun. 189:1747-1753(1992).
Runting A.S.,et al.Growth Factors 9:99-105(1993).
Schnuerch H.,et al.Development 119:957-968(1993).


## Images




All lanes : Anti-Tek Antibody (C-term) at 1:2000 dilution Lane 1: mouse liver lysate Lane 2: mouse lung lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 126 kDa Blocking/Dilution buffer: 5% NFDM/TBST.


Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.