

**Phospho-TEK(Y1113) Antibody**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP3563a****Specification**

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**Phospho-TEK(Y1113) Antibody - Product Information**

Application	DB,E
Primary Accession	<a href="#">Q02763</a>
Other Accession	<a href="#">Q02858</a> , <a href="#">Q73791</a> , <a href="#">Q06807</a>
Reactivity	Human
Predicted	Bovine, Zebrafish, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	125830

**Phospho-TEK(Y1113) Antibody - Additional Information****Gene ID** 7010**Other 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, hTIE2, p140 TEK, CD202b, TEK, TIE2, VMCM, VMCM1

**Target/Specificity**

This TEK Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding Y1113 of human TEK.

**Dilution**

DB~~1:500

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**

Phospho-TEK(Y1113) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Phospho-TEK(Y1113) Antibody - Protein Information****Name** TEK ([HGNC:11724](#))

**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 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, ANGPT1 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 (PubMed:18425119, PubMed:18425120) 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 (PubMed:11806244).

#### **Tissue Location**

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

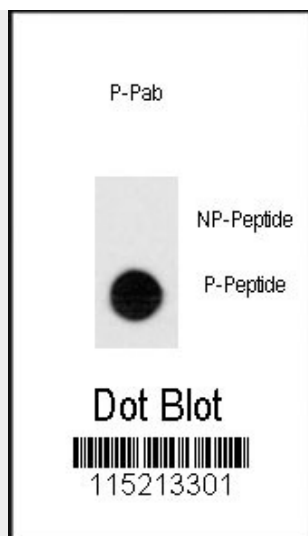
#### **Phospho-TEK(Y1113) Antibody - Protocols**

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](#)

#### **Phospho-TEK(Y1113) Antibody - Images**





Dot blot analysis of anti-Phospho-TEK-pY1113 Antibody (Cat.#AP3563a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.5ug per ml.

#### **Phospho-TEK(Y1113) Antibody - Background**

The TEK receptor tyrosine kinase is expressed almost exclusively in endothelial cells in mice, rats, and humans. This receptor possesses a unique extracellular domain containing 2 immunoglobulin-like loops separated by 3 epidermal growth factor-like repeats that are connected to 3 fibronectin type III-like repeats. The ligand for the receptor is angiopoietin-1. Defects in TEK are associated with inherited venous malformations; the TEK signaling pathway appears to be critical for endothelial cell-smooth muscle cell communication in venous morphogenesis. TEK is closely related to the TIE receptor tyrosine kinase.

#### **Phospho-TEK(Y1113) Antibody - References**

- Cascone, I., et al., Blood 102(7):2482-2490 (2003).
- DeBusk, L.M., et al., Arthritis Rheum. 48(9):2461-2471 (2003).
- Poncet, S., et al., Neuropathol Appl Neurobiol 29(4):361-369 (2003).
- Lee, H.J., et al., Biochem. Biophys. Res. Commun. 304(2):399-404 (2003).
- Sussman, L.K., et al., Cancer Biol. Ther. 2(3):255-256 (2003).