Catalog # TI2-M5253



Synonym

TIE2, Tie-2, TEK, VMCM, VMCM1, CD202b, Angiopoietin-1 receptor

Source

Mouse TIE2, Fc Tag(TI2-M5253) is expressed from human 293 cells (HEK293). It contains AA Ala 23 - Leu 746 (Accession # <u>Q02858-1</u>).

Molecular Characterization

TIE2(Ala 23 - Leu 746) Fc(Pro 100 - Lys 330) Q02858-1 P01857

This protein carries a human IgG1 Fc tag at the C-terminus.

The protein has a calculated MW of 107.4 kDa. The protein migrates as 120-140 kDa under reducing (R) condition (SDS-PAGE) due to glycosylation.

Endotoxin

Less than 1.0 EU per μ g by the LAL method.

Purity

>95% as determined by SDS-PAGE.

>90% as determined by SEC-MALS.

Formulation

Lyophilized from 0.22 μm filtered solution in PBS, pH7.4 with trehalose as protectant.

Contact us for customized product form or formulation.

Reconstitution

Please see Certificate of Analysis for specific instructions.

For best performance, we strongly recommend you to follow the reconstitution protocol provided in the CoA.

Storage

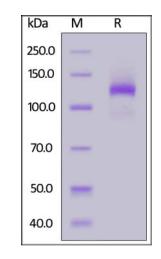
For long term storage, the product should be stored at lyophilized state at -20°C or lower.

Please avoid repeated freeze-thaw cycles.

This product is stable after storage at:

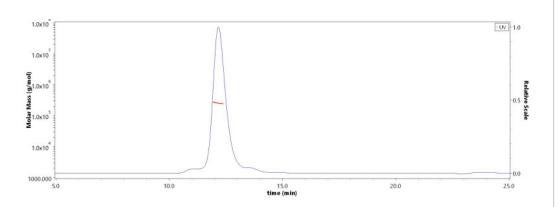
- -20°C to -70°C for 12 months in lyophilized state;
- -70°C for 3 months under sterile conditions after reconstitution.

SDS-PAGE



Mouse TIE2, Fc Tag on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 95%.

SEC-MALS



The purity of Mouse TIE2, Fc Tag (Cat. No. TI2-M5253) is more than 90% and the molecular weight of this protein is around 235-285kDa verified by SEC-MALS.

Report

Bioactivity-ELISA

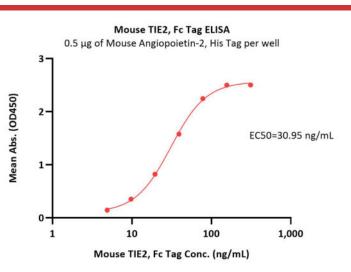
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9/22/2023

Mouse TIE2 Protein, Fc Tag (MALS verified)



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Immobilized Mouse Angiopoietin-2, His Tag (Cat. No. AN2-M52H3) at 5 μ g/mL (100 μ L/well) can bind Mouse TIE2, Fc Tag (Cat. No. TI2-M5253) with a linear range of 5-78 ng/mL (QC tested).

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.

Clinical and Translational Updates

Please contact us via TechSupport@acrobiosystems.com if you have any question on this product.



