

PRODUCT INFORMATION



Tie2/CD202b Extracellular Domain (human, recombinant)

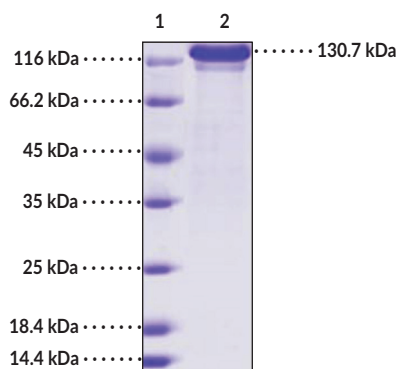
Item No. 33988

Overview and Properties

Synonyms:	Angiotensin-1 Receptor, Endothelial Tyrosine Kinase, Tunica Interna Endothelial Cell Kinase 2, Tyrosine-Protein Kinase Receptor TEK
Source:	Active recombinant human C-terminal IgG1 Fc-His-tagged Tie2/CD202b expressed in HEK293 cells
Amino Acids:	23-745
Uniprot No.:	Q02763
Molecular Weight:	108.5 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥90% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	<1.0 EU/μg, determined by the LAL endotoxin assay
Protein Concentration:	<i>batch specific</i> mg/ml
Bioactivity:	See figures for details

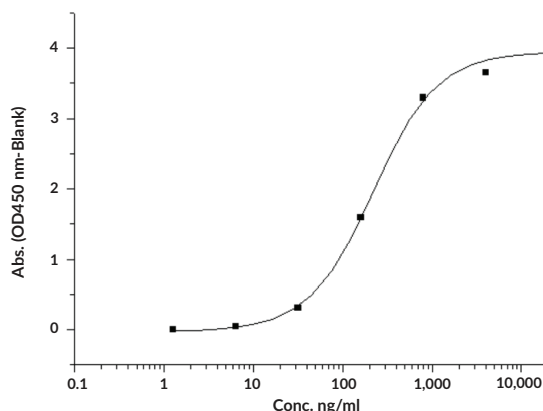
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: Tie2/CD202b Extracellular Domain

SDS-PAGE Analysis of Tie2/CD202b Extracellular Domain.
This protein has a calculated molecular weight of 108.5 kDa. It has an apparent molecular weight of approximately 130.7 kDa by SDS-PAGE under reducing conditions due to glycosylation.



Measured by its binding ability in a functional ELISA.
Immobilized Tie2/CD202b Extracellular Domain at 10 μg/ml (100 μl/well) can bind human Tie2/Fc chimera with a range of 0.2-20 μg/ml.

WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the [complete](#) Safety Data Sheet, which has been sent via email to your institution.

WARRANTY AND LIMITATION OF REMEDY
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Description

Tunica interna endothelial cell kinase 2 (Tie2), also known as CD202b, is a receptor tyrosine kinase that is encoded by the *TEK* gene in humans.^{1,2} It is composed of an extracellular ligand-binding domain containing immunoglobulin, EGF-like, and fibronectin-like repeats, a transmembrane domain, and an intracellular tyrosine kinase domain.¹ Tie2 is expressed in embryonic and adult endothelial cells, as well as hematopoietic stem cells (HSCs), where it is bound by the growth factors angiopoietin-1, -2, and -4. Upon ligand binding, Tie2 forms homodimers or heterodimers with Tie1, resulting in Tie2 phosphorylation and activation of various signaling pathways, including the PI3K pathway, to promote angiogenesis, endothelial cell survival, and hematopoiesis.^{1,3} *TEK* knockout in mice is embryonic lethal with embryos lacking cardiovascular system development and exhibiting vascular hemorrhaging.¹ Tie2 is upregulated in the vascular endothelium of several cancers, including lung, breast, and prostate cancers, and somatic mutations in *TEK* result in the development of blue rubber bleb nevus syndrome, a disease characterized by venous malformations and gastrointestinal lesions.^{1,2} Cayman's Tie2/CD202b Extracellular Domain (human, recombinant) protein can be used for ELISA and Western blot (WB) applications. This protein is a disulfide-linked homodimer. The reduced monomer, composed of Tie2 (amino acids 23-745) fused to His-tagged human IgG1 Fc at its C-terminus, consists of 970 amino acids, has a calculated molecular weight of 108.5 kDa, and a predicted N-terminus of Ala23 after signal peptide cleavage. As a result of glycosylation, the monomer migrates at approximately 130.7 kDa by SDS-PAGE under reducing conditions.

References

1. Martin, V., Liu, D., Fueyo, J., *et al.* Tie2: A journey from normal angiogenesis to cancer and beyond. *Histol. Histopathol.* **23(6)**, 773-780 (2008).
2. Soblet, J., Kangas, J., Nätyнки, M., *et al.* Blue rubber bleb nevus (BRBN) syndrome is caused by somatic *TEK* (*TIE2*) mutations. *J. Invest. Dermatol.* **137(1)**, 207-216 (2017).
3. Leppänen, V.-M., Saharinen, P., and Alitalo, K. Structural basis of Tie2 activation and Tie2/Tie1 heterodimerization. *Proc. Natl. Acad. Sci. USA* **114(17)**, 4376-4381 (2017).

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