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Product datasheet for AP26034PU-N

VEGF Receptor 2 (KDR) (esKDR) Rabbit Polyclonal Antibody

Product data:

Product Type:	Primary Antibodies
Applications:	IF, WB
Recommended Dilution:	**Western blot:** 1-5 µg/ml. **Immunofluorescence/Immunohistochemistry:** 1-5 µg/ml.
Reactivity:	Human
Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Immunogen:	Peptide consisting of the unique C-terminal end of esKDR: CGRETILDHSAEAVGMP
Specificity:	This antibody detects CD309 / VEGFR-2 / Flk-1. This poyclonal antibody directed against the unique C-terminal end of the endogenous sKDR (CGRETILDHSAEAVGMP) recognizing solely the endogenous form but not sKDR (D7) and sKDR (D7)-Fc consisting of the full extraplasmatic domain. The endogenous sKDR generated by alternative splicing consists of the first 6 Ig-like loops with a unique c-terminal end.
Formulation:	PBS, pH 7.2 State: Purified State: Lyophilized purified IgG fraction
Reconstitution Method:	Restore in sterile water to a concentration of 0.1-1.0 mg/ml.
Purification:	Protein A Chromatography
Conjugation:	Unconjugated
Storage:	Store lyophilized at 2-8°C for 6 months or at -20°C long term. After reconstitution store the antibody undiluted at 2-8°C for one month or (in aliquots) at -20°C long term. Avoid repeated freezing and thawing.
Stability:	Shelf life: one year from despatch.
Gene Name:	kinase insert domain receptor
Database Link:	Entrez Gene 3791 Human P35968

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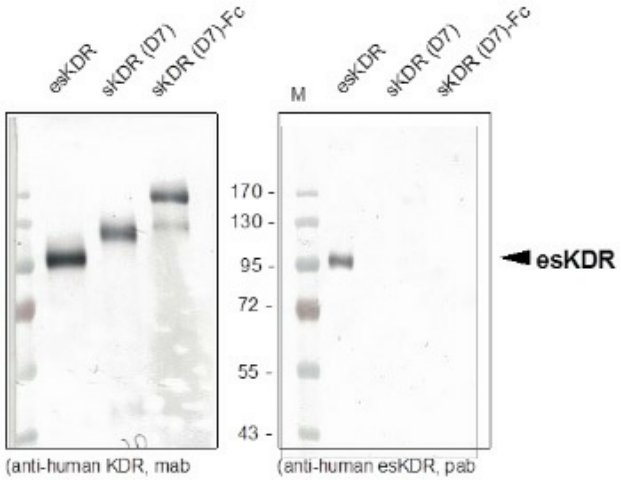
Background: Disruption of the precise balance of positive and negative molecular regulators of blood and lymphatic vessel growth can lead to myriad diseases. Although dozens of natural inhibitors of hemangiogenesis have been identified, an endogenous selective inhibitor of lymphatic vessel growth has not to our knowledge been previously described. A splice variant of the gene encoding vascular endothelial growth factor receptor-2 (VEGFR-2) that encodes a secreted form of the protein, designated endogenous soluble VEGFR-2 (esVEGFR-2/KDR) has been described. The endogenous soluble esKDR inhibits developmental and reparative lymphangiogenesis by blocking VEGF-C function. Tissue-specific loss of esKDR in mice induced, at birth, spontaneous lymphatic invasion of the normally alymphatic cornea and hyperplasia of skin lymphatics without affecting blood vasculature. Administration of esKDR inhibited lymphangiogenesis but not hemangiogenesis induced by corneal suture injury or transplantation, enhanced corneal allograft survival and suppressed lymphangioma cellular proliferation. Naturally occurring esKDR thus acts as a molecular uncoupler of blood and lymphatic vessels; modulation of esKDR might have therapeutic effects in treating lymphatic vascular malformations, transplantation rejection and, potentially, tumor lymphangiogenesis and lymphedema.
Recombinant human esKDR generated by alternative splicing consist of the first 6 Ig-like loops followed by the unique C-terminal end: CGRETILDHSAEAVGMP.

Synonyms: VEGFR2, FLK1, KDR, VEGF Receptor 2

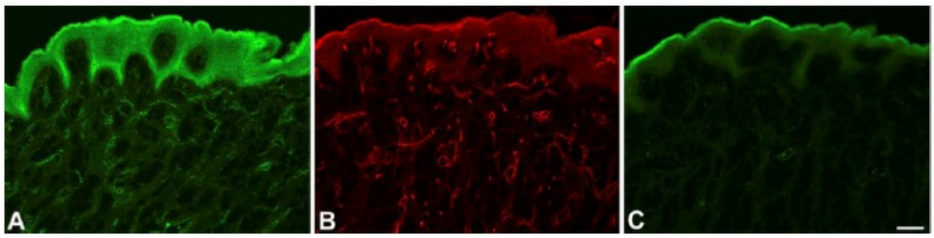
Protein Families: Druggable Genome, ES Cell Differentiation/IPS, Protein Kinase, Transmembrane

Protein Pathways: Cytokine-cytokine receptor interaction, Endocytosis, Focal adhesion, VEGF signaling pathway

Product images:



Western Analysis of anti-human esKDR. Samples were loaded in 10% SDS-polyacrylamide gel under reducing conditions. Left panel: monoclonal antibody against the soluble KDR D1-7); Right panel: polyclonal antibody (peptide) against against the unique C-terminal end of esKDR.



Immunofluorescence staining with consecutive sections of unfixed, human foreskin. A) Staining with anti-sVEGFR2/KDR antibodies. Note signal in epidermis and vessels. B) Staining with anti-membrane-bound VEGFR-2/KDR. Note staining in vessels. C) Negative control. Note non-specific fluorescence in the hornified layer of the epithelium. Provided by Prof. J. Wilting, GÃ¶ttingen, Germany.