



Anti-human endogenous soluble VEGFR-2/KDR **„Vascular Endothelial Growth Factor Receptor-2“**

Catalog Number 102-PA19
Lot Number (See product label)
Size 200µg

Species Reactivity Human
Isotype Polyclonal Rabbit IgG
Immunogen Recombinant human esKDR (RT# S01-003)
Accession codes:
P22333
NM 002253.2

Preparation: Produced from sera of rabbits pre-immunized with highly pure (>95%) recombinant human esKDR (Ala20-Pro678) derived from insect cells.

Purification: Protein-A purified

Endotoxin level: < 0.1 EU/1µg of the antibody (LAL)

Formulation: Lyophilized from PBS, pH 7.2

Reconstitution: Centrifuge vial prior to opening. Reconstitute in sterile water to a concentration of 0.1-1.0 mg/ml.

Storage/Stability: The lyophilized antibody is stable at room temperature for up to 1 month. The reconstituted antibody is stable for at least two weeks at 2-8°C. Frozen aliquots are stable for at least 6 months when stored at -20°C. **Avoid repeated freeze-thaw cycles!**

Country of Origin: Germany

APPLICATIONS

Western Blot: 2-5µg/mL

NOTE: Optimal dilutions should be determined by each laboratory for each application!

For Research use only
Not for human use.

Product Information

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*.

Reference

Shibata et al, BMC Medicine 8 (2010); Albuquerque et al, Nature Med 2009; Ebos et al, Mol Cancer Res 2 (2004); Ebos et al, Cancer res 68 (2008).



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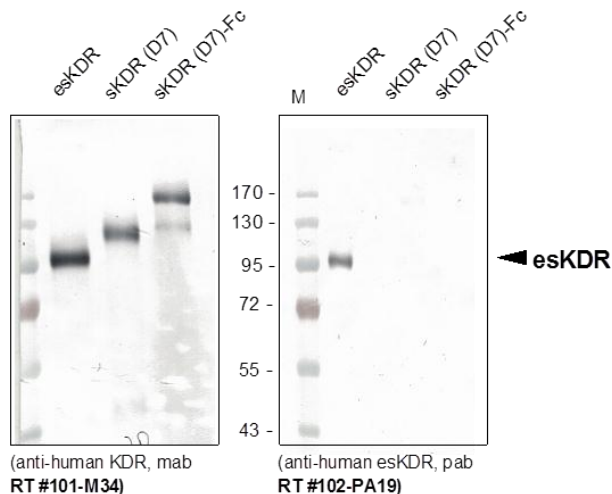


Figure 1. Western Analysis of anti-human esKDR. Samples were loaded in 10% SDS-polyacrylamide gel under reducing conditions. Left panel: monoclonal antibody against the soluble KDR D1-7); Right panel: polyclonal antibody (peptide) against against the unique C-terminal end of esKDR.

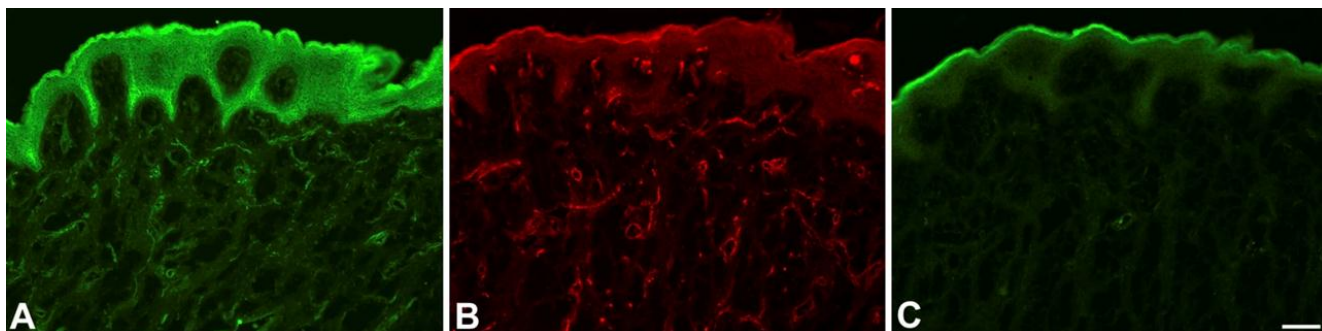


Figure 2: Immunofluorescence staining with consecutive sections of unfixated, human foreskin. A) Staining with anti-sVEGFR2/KDR antibodies (#102-PA19). Note signal in epidermis and vessels. B) Staining with anti-membrane-bound VEGFR-2/KDR (#101-M32). Note staining in vessels. C) Negative control. Note non-specific fluorescence in the hornified layer of the epithelium.

Provided by Prof. J. Wilting, Göttingen, Germany.