Recombinant Mouse Endogenous Soluble VEGFR-2/Flk-1

FOR RESEARCH ONLY! NOT FOR HUMAN USE!

Cat.-no:	**S01-M04**
Size:	20 µg
Lot. No.:	According to product label
Country of origin:	Germany

Scientific Background

Gene:	*Kdr, Flk-1*
Synonyms:	Vascular endothelial growth factor receptor 2, Protein-tyrosine kinase receptor flk-1

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 mouse esKDR generated by alternative splicing consist of the first 6 Ig-like loops followed by the unique C-terminal end: *GMEASLGDRIAMP*.

References

1. Shibata et al, BMC Medicine 8 (2010)
2. Albaquerque et al, Nature Med 2009
3. Ebos et al, Mol Cancer Res 2 (2004)
4. Ebos et al, Cancer res 68 (2008).

Sequence

ASVGLTGDFLHPPKLSTQKDILTILANTTLQITCRGQRDLDWLWPNAQRDSE
ERVLVTECGGGDSIFCKTLTIPRVVGNDTGAYKCSYRDVDIASTVYVYVRDY
RSPFIASVSDQHGIVYITENKNKTVVIPCRGSISNLNVSLCARYPEKRFVPD
GNRISWDSEIGFTLPSYMISYAGMVFCEAKINDETYQSIMYIVVVVGYRIYD
VILSPPHEIELSAGEKLVLNCTARTELNVGLDFTWHSPPSKSHHKKIVNRDV
KPFPGTVAKMFLSTLTIESVTKSDQGEYTCVASSGRMIKRNRTFVRVHTKPF
IAFGSGMKSLVEATVGSQVRIPVKYLSYPAPDIKWYRNGRPIESNYTMIVGD
ELTIMEVTERDAGNYTVILTNPISMEKQSHMVSLVVNVPPQIGEKALISPMD
SYQYGTMQTLTCTVYANPPLHHIQWYWQLEEACSYRPGQTSPYACKEWRHVE
DFQGGNKIEVTKNQYALIEGKNKTVSTLVIQAANVSALYKCEAINKAGRGER
VISFHVIRGPEITVQPAAQPTEQESVSLLCTADRNTFENLTWYKLGSQATSV
HMGESLTPVCKNLDALWKLNGTMFSNSTNDILIVAFQNASLQDQGDYVCSAQ
DKKTKKRHCLVKQLIILGMEASLGDRIAMP

Database References

Protein RefSeq:	ACJ66293.1
Uniprot ID:	P35918
mRNA RefSeq:	EU884114

Product Specifications

Expressed in	Insect cells
Purity	> 95% by SDS-PAGE
Buffer	25 mM MES, 100 mM NaCl; pH 5.5
Stabilizer	None
Formulation	lyophilized
Length (aa):	654
MW:	105 kDa (Monomer)
Result by N-terminal sequencing	ASVGLPGDFL

Stability: The material is stable for greater than six months at -20°C to -70°C. After the first thawing it is recommended to aliquote the material, because repeated freeze-thaw cycles will decrease the activity. Store at 4°C not longer than 2 days.

Reconstitution: The lyophilized mouse sFlk-1 is soluble in water and most aqueous buffers; it should be reconstituted in water or PBS to a concentration of not lower than 100µg/ml.

AVOID REPEATED FREEZE AND THAW CYCLES!

Biological Activity: Measured by its ability to inhibit the VEGF165-induced proliferation in human umbilical vein endothelial (HUVE) cells.

ReliaTech GmbH
Phone: +49 (0)5331 8586 987 • Fax: +49 (0)5331 8586 989 • E-mail: orders@reliatech.de • web: www.reliatech.de
Location: Lindener Str. 15, 38300 Wolfenbüttel, Germany

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Handling/Application

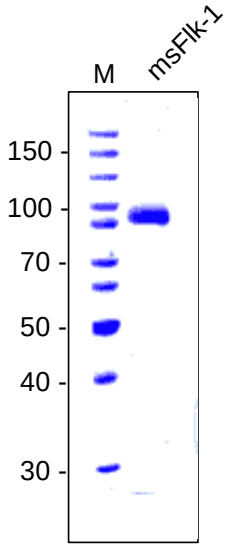

Fig. 1: SDS-PAGE analysis of recombinant mouse soluble VEGFR-2 produced in insect cells. Sample was loaded in 10% SDS-polyacrylamide gel under reducing condition and stained with Coomassie blue.

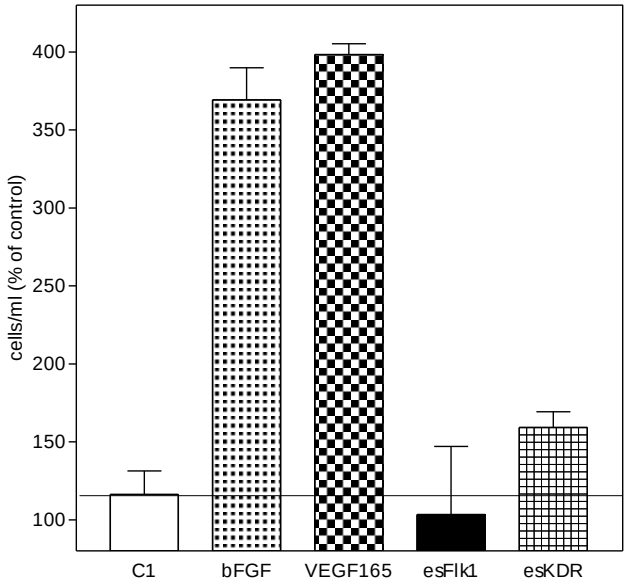

Figure 2. Inhibition of the VEGF165-induced proliferation of HUVE cells by recombinant human and mouse endogenous sKDR and sFlk-1 HUVECs were stimulated with 10 ng/ml VEGF165, both soluble receptors were added with a 100X excess.

ReliaTech GmbH
Phone: +49 (0)5331 8586 987 • Fax: +49 (0)5331 8586 989 • E-mail: orders@reliatech.de • web: www.reliatech.de
Location: Lindener Str. 15, 38300 Wolfenbüttel, Germany