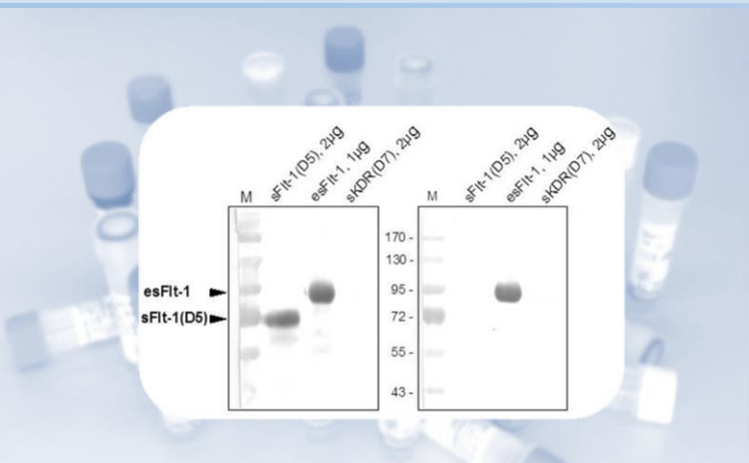


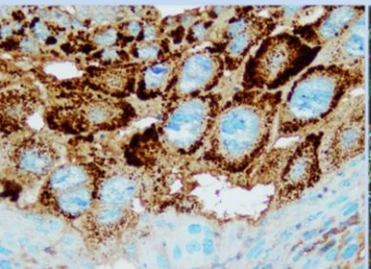
Western Blot Analysis with recombinant human sFlt-1(D5) **(#S01-011)**, sFlt-1 **(#S01-010)** and sKDR(D7) **(#S01-001)**. Our rabbit anti-human sFlt-1 **(#102-PA21,** right) only detects endogenous sFlt-1 whereas mouse anti-human Flt-1 (**#101-M30,** left) also detects sFlt-1(D5). There is no cross reactivity with sKDR(D7).

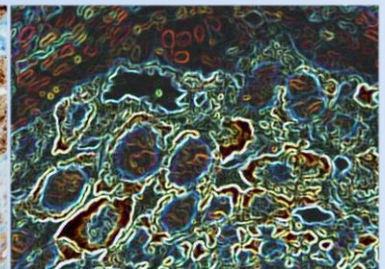
Service options

You are looking for something individual? Check, if you can find a solution in our contract work program:

NEW

- Activity Assays
  → send your protein of interest – we check the activity e.g. in primary endothelial cells, fibroblasts as well as cell lines.
- Production of recombinant proteins in E.coli and insect cells
- IXpress & Antibody
  → your own polyclonal antibody for your individual antigen.
- Reagent Formulation Service
  → design your own reagent conditions for your individual application.





Is it possible?

- You don't know us?

We are focused on the in-house production of new high-quality reagents for (lymph-)angiogenic research. However, biology is made to overcome traditions, isn't it? - Factors from our product palette pop up everywhere in biological sciences. As a result customers from varying fields in biology and medical sciences have discovered our reagents for their research in the meantime and - rely on them.

ReliaTech was founded in 1999 by Dr. Herbert Weich (HZI Braunschweig), Dr. Bernhard Barleon (Clinic for tumor biology (KTB), Freiburg) and Dr. Avner Yayon (Weizmann Institute of Science (WIS), Israel). In 2007 Dr. Volker Jaeger (HZI Braunschweig) joined the board.

A consistent and sophisticated dialog between leading scientists in lymph-/angiogenesis and our in-house experts combined with a fast supply of reagents is the secret that shapes the quality of our reagents and services. Find out yourself what we can do for you and visit our webpages!

ReliaTech

Receptor Ligand Technologies GmbH

Your certified partner in biotechnology

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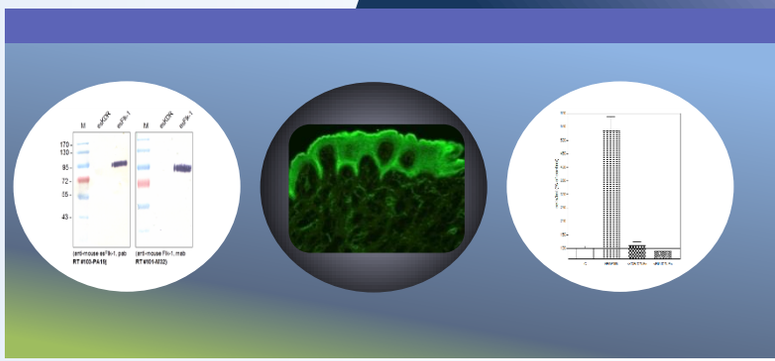
Release 08/2016

Code generated by ZXing Project

ReliaTech presents



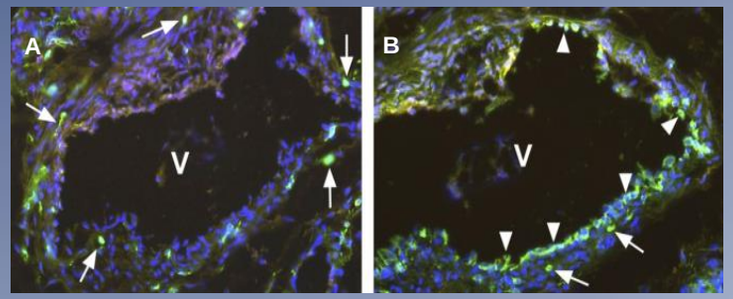
Endogenous Treasures

sVEGFR-1/sFlt-1

sVEGFR-2/KDR

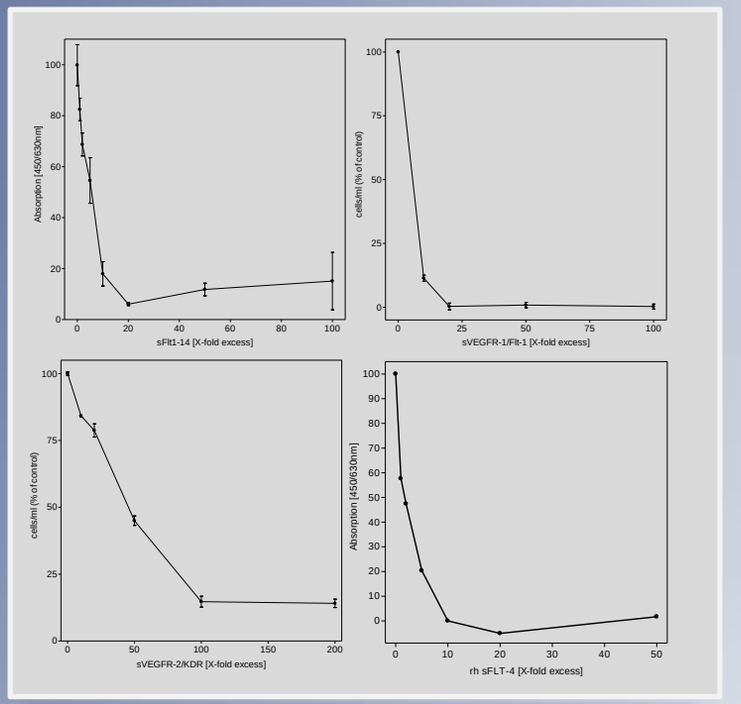
Human VEGFR-1/ Flt-1

The existence of a naturally occurring soluble Flt-1 (sFlt-1) is known since 1993. In 2008 a second soluble Flt-1 form was identified, sFlt1-14. Both are differentially expressed and distributed in human tissues. sFlt1-14 is highly placenta-specific and seems to play a role in preeclampsia, whereas sFlt-1 is expressed in several tissues. The therapeutic potential of sFlt-1 as an anti-angiogenic agent has been validated by an increasing number of preclinical studies. Anti-angiogenesis therapy changes the concentration of circulating VEGF, PIGF, sFlt-1, sKDR and even sFLT-4.



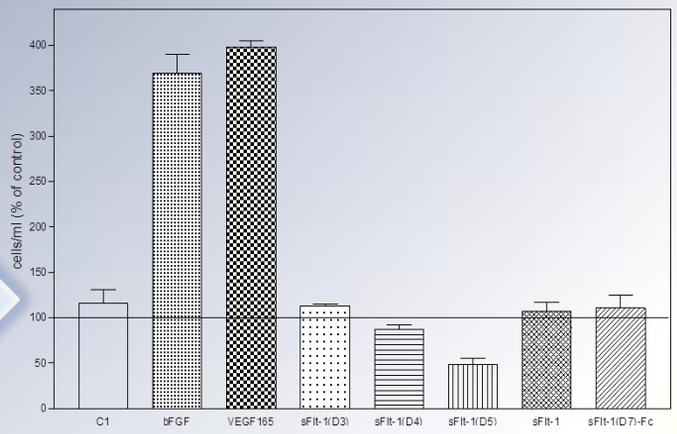
Immunofluorescence staining using ReliaTech's polyclonal antibody (**#102-PA21**, (A), green) directed against the C terminal end of endogenous sFlt-1 [Kendall & Thomas, PNAS 1993]. The antibody was generated against the unique C terminal end of sFlt-1 (*CGEHCNKKAVFSRISKFKSTRNDSTTQSNVKH*). B) Monoclonal antibody directed against the extracellular domain of the membrane-bound Flt-1 (**#101-M30**, green). Shown are two neighboring sections of a human vein (V), located near a hemangioma. The antibody against the soluble VEGFR-1/Flt-1 marks single cells (arrows) within media and adventitia of the vein. The antibody against membrane-bound VEGFR-1/Flt-1 marks single cells (arrows) and the endothelium (arrowhead) of the vein. Cell nuclei are counter-stained with DAPI (blue).
The experiment was performed by the group of Dr. K. Butler and Prof. Dr. J. Wilting, University of Göttingen, Germany.

Blocking activity of different forms of the soluble Flt-1 receptor (all available under www.reliatech.de) as shown by the inhibition of $VEGF_{165}$-induced proliferation of primary HUVECs.

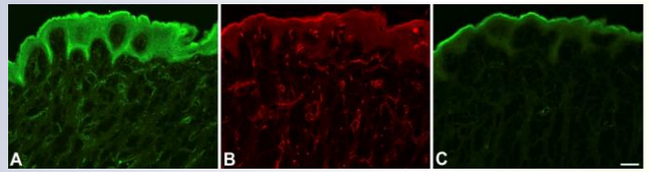


Blocking activity of soluble VEGF receptors was shown by the inhibition of $VEGF_{165}$-induced proliferation (VEGF-C induced respectively) of primary HDLECs for sFlt1-14 (**#S01-072**), sFlt-1 (**#S01-010**) and sKDR (**#S01-004**). For monitoring sFLT-4 (**#S01-017**) inhibition VEGF-C was used for proliferation induction.

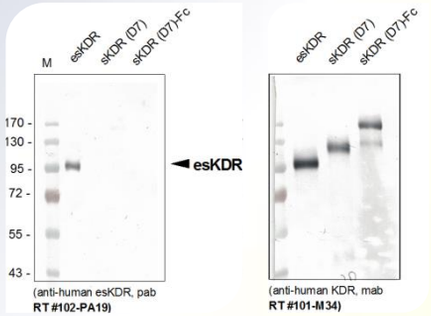
Soluble & endogenous



The existence of an endogenous soluble KDR was long discussed but first described in 2004. Soluble KDR inhibits developmental and reparative lymphangiogenesis by blocking VEGF-C function. Administration of esKDR inhibited lymphangiogenesis but not hemangiogenesis. 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.



Immunofluorescence staining of consecutive sections of unfixed, human foreskin. A) Staining with rabbit anti-human sKDR **(#102-PA19)** - this antibody was generated against a peptide of the unique C terminal end of esKDR (*CGRETILDHSAEAVGMP*) [Albuquerque et al., Nature Medicine 2009]). Note signal in epidermis and vessels. B) Staining with monoclonal anti-membrane-bound KDR (**#101-M32**). Note staining in vessels. C) Negative control. Note nonspecific fluorescence in the hornified layer of the epithelium.
The experiment was performed by the group of Dr. K. Butler and Prof. Dr. J. Wilting, University of Göttingen, Germany.



Western Blot Analysis with recombinant human esKDR, sKDR(D7) and sKDR(D7)-Fc (**#**). The anti-human sKDR (**#102-PA19**) only recognizes the endogenous sKDR whereas the anti-human KDR (**#101-M34**) also recognizes sKDR(D7) and sKDR(D7)-Fc.

Human VEGFR-2/ KDR