

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human VEGF-C in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human VEGF-C
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
ELISA	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Vascular endothelial growth factor C (VEGF-C) and VEGF-D constitute a VEGF sub-family that share the conserved VEGF homology domain (VHD) with other VEGF family members but are distinguished by their preferential formation of non-covalent homodimers. Both VEGF-C and -D have long N- and C-terminal propeptide extensions. The VEGF-C propeptide undergoes stepwise proteolytic processing to generate ligands with increasing affinity for VEGF-R3. However, only the fully processed VEGF-C containing just the VHD can bind VEGF-R2. None of the VEGF-C forms have appreciable affinity for VEGF-R1. VEGF-C is expressed in multiple adult human tissues, most prominently in lymph nodes, heart, placenta, ovary, and small intestine. Traces of VEGF-C are also detected in brain, liver, thymus, skeletal muscles, spleen, prostate, testis and colon. Unlike other VEGF family members, VEGF-C expression is not regulated by hypoxia. VEGF-C is a lymphangiogenic growth factor and the VEGF-C/VEGF-R3 signaling pathway has been shown to be crucial for lymphangiogenesis. VEGF-C and VEGF-R3 are usually co-expressed at sites with lymphatic vessel sprouting, in the embryo, and in various pathological conditions. VEGF-C stimulates lymphangiogenesis in the avian chorioallantoic membrane model. Over-expression of VEGF-C in breast cancer cells has been shown to increase intratumoral lymphangiogenesis, resulting in enhanced metastasis to regional lymph nodes and to the lungs. Mouse tumors expressing elevated levels of VEGF-C have increased lymphatic metastasis and increased lymphatic surface area in the tumor margin. VEGF-C is also associated with lymph node metastasis of colorectal carcinoma. Besides lymphangiogenesis, VEGF-C can have potent effects on physiological angiogenesis through its interaction with VEGF R2. The protein can stimulate migration and proliferation of endothelial cells *in vitro* and *in vivo* and has been shown to stimulate angiogenesis in the mouse cornea and in rabbit hind limb ischaemia.

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