Human/Mouse/Rat VEGF-C Antibody

Antigen Affinity-purified Polyclonal Sheep IgG
Catalog Number: AF2179

DESCRIPTION

Species Reactivity	Human/Mouse/Rat
Specificity	Detects VEGF-C in direct ELISAs and Western blots. In direct ELISAs, less than 1% cross-reactivity with recombinant human (rh) VEGF-206, rhVEGF-B186, rhVEGF-165, rhVEGF-121, and rhVEGF-D is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human VEGF-C Thr103-Arg227 Accession # P49767
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μm filtered solution in PBS.

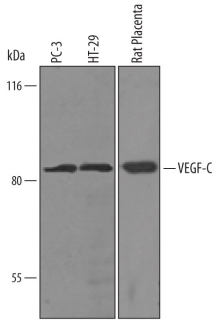
APPLICATIONS

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.*

	Recommended Concentration	Sample
Western Blot	1 µg/mL	See Below

DATA

Western Blot



Detection of Human and Rat VEGF-C by Western Blot. Western blot shows lysates of PC-3 human prostate cancer cell line, HT-29 human colon adenocarcinoma cell line, and rat placenta tissue. PVDF Membrane was probed with 1 µg/mL of Human VEGF-C Antigen Affinity-purified Polyclonal Antibody (Catalog # AF2179) followed by HRP-conjugated Anti-Sheep IgG Secondary Antibody (Catalog # HAF016). A specific band was detected for VEGF-C at approximately 85 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 8.

PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.2 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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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 (1-10).

References:

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