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## VEGF-C Antibody

**For Research Use Only. Not for Use in Diagnostic Procedures.**

<b>Applications:</b> W	<b>Reactivity:</b> H	<b>Sensitivity:</b> Recombinant protein	<b>MW (kDa):</b> 21	<b>Source/Isotype:</b> Rabbit	<b>UniProt ID:</b> #P49767	<b>Entrez-Gene Id:</b> 7424
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<b>Product Usage Information</b>	<b>Application</b> Western Blotting	<b>Dilution</b> 1:1000
<b>Storage</b>	Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.	
<b>Specificity/Sensitivity</b>	VEGF-C Antibody detects recombinant human VEGF-C protein at various concentrations. This antibody does not cross-react with other VEGF family members.	
<b>Source / Purification</b>	Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Thr189 of human VEGF-C. Antibodies are purified by protein A and peptide affinity chromatography.	
<b>Background</b>	<p>Vascular endothelial growth factor (VEGF) is a highly specific mitogen for vascular endothelial cells. VEGF and its close relatives VEGF-B, -C and -D form a subfamily within PDGF family of growth factors, which belongs to the cysteine knot class of cytokines. Five VEGF isoforms of 121, 145, 165, 189 and 206 amino acids (VEGF121-206) are generated as a result of alternative splicing from a single VEGF gene (1). The various VEGF forms bind to three tyrosine-kinase receptors, VEGFR-1, VEGFR-2 and VEGFR-3 which are expressed almost exclusively in endothelial cells. VEGFR-2 is the main angiogenic signal transducer for VEGF, while VEGFR-3 is specific for VEGF-C and -D and is necessary and sufficient for lymphangiogenic signaling. However, upon proteolytic processing VEGF-C and -D gain the ability to also bind and activate VEGFR-2 (2). Guided by the binding properties of the ligands, the VEGFRs are able to form both homodimers and heterodimers. Receptor dimerization is accompanied by activation of receptor kinase activity leading to receptor autophosphorylation. Phosphorylated receptors recruit interacting proteins and induce downstream signaling (3). Recently, tumor therapies based on neutralizing anti-VEGF antibodies and small molecule tyrosine kinase inhibitors targeting VEGFRs have been developed. These new strategies for tumor treatment show the clinical relevance of inhibiting VEGF signal transduction pathways that are exaggerated in pathological angiogenesis (4).</p>	
<b>Background References</b>	<ol style="list-style-type: none"> <li>1. Olsson, A.K. et al. (2006) <i>Nat. Rev. Mol. Cell Biol.</i> 7, 359-371.</li> <li>2. Gluzman-Poltorak, Z. et al. (2001) <i>J. Biol. Chem.</i> 276, 18688-18694.</li> <li>3. Matsumoto, T. and Mugishima, H. (2006) <i>J. Atheroscler. Thromb.</i> 13, 130-135.</li> <li>4. Gatto, B. and Cavalli, M. (2006) <i>Anticancer Agents Med. Chem.</i> 6, 287-301.</li> </ol>	
<b>Species Reactivity</b>	Species reactivity is determined by testing in at least one approved application (e.g., western blot).	
<b>Western Blot Buffer</b>	IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.	
<b>Applications Key</b>	<b>W:</b> Western Blotting	
<b>Cross-Reactivity Key</b>	<b>H:</b> Human	
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