

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CXCL5/ENA-78 in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human CXCL5/ENA-78 Ala37-Asn114 Accession # P42830
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 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.

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

CXCL5, also known as epithelial cell-derived neutrophil-activating peptide (ENA-78), is an 8 kDa proinflammatory member of the CXC subfamily of chemokines. Its Glu-Leu-Arg (ELR) motif confers angiogenic properties and distinguishes it from ELR-CXC chemokines which are angiostatic (1-3). Human CXCL5 shares 57% amino acid (aa) sequence identity with mouse and rat CXCL5. Among other human ELR+ chemokines, it shares 77% aa sequence identity with CXCL6/GCP-2 and 35%-51% with CXCL1/GRO alpha, CXCL2/GRO beta, CXCL3/GRO gamma, CXCL7/NAP-2, and CXCL8/IL-8. Inflammatory stimulation up-regulates CXCL5 production in multiple hematopoietic cell types, fibroblasts, endothelial cells, and vascular smooth muscle cells. *In vivo*, CXCL5 is elevated at sites of inflammation and pulmonary fibrosis where it promotes neutrophil infiltration and activation as well as angiogenesis (3-6). Its upregulation contributes to increased vascularization, tumor growth, and metastasis in many cancers (6-9). Full length CXCL5 (78 aa) is trimmed at the N-terminus by cathepsin G and chymotrypsin to ENA-74 (74 aa) and ENA-70 (70 aa), with the shortened forms showing increased potency relative to full length CXCL5 (10, 11). CXCL5 exerts its effects primarily through interactions with CXCR2 (6, 12). It also binds duffy antigen receptor for chemokines (DARC), which can limit CXCR2-mediated responses (13, 14).

PRODUCT SPECIFIC NOTICES

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