

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CXCL12/SDF-1 β in direct ELISAs and Western blots. Neutralizes 60-80% of the biological activity of CXCL12/SDF-1 β and does not neutralize the biological activity of SDF-1 α . In Western blots, less than 5% cross-reactivity with recomb
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human CXCL12/SDF-1 β Lys22-Met93 Accession # P48061
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 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.

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

CXCL12, also known as SCYB12, PBSF and SDF-1 β , is an 8.3 kDa, heparin-binding member of the CXC (or alpha-) family of chemokines (1, 2). Feline CXCL12(β) is synthesized as a 93 amino acid (aa) precursor that contains a 21 aa signal sequence and a 72 aa mature region (3). The mature molecule exhibits a typical three antiparallel β -strand chemokine-like fold. There are no potential N-linked glycosylation sites. N-terminal aa's 1 - 8 form a receptor binding site, while aa's 1 and 2 (Lys-Pro) are involved in receptor activation (4). The C-terminus is likely associated with heparin binding (5). SDF-1 β circulates and undergoes proteolytic processing. CD26 will remove the first two N-terminal amino acids, possibly creating a reduced-activity chemokine (5, 6). In addition to the β -isoform, alternate splicing of the feline SDF-1 gene generates an α -isoform. The alpha isoform is identical to SDF-1 β , but shorter by four aa's at the C-terminus (3). Although α - and β -isoforms show similar activity, SDF-1 α is differentially processed, and different cells secrete the two isoforms (5, 7). Mature feline SDF-1 β is 96%, 97% and 100% aa identical to rat, mouse and human SDF-1 β , respectively. Human (and by inference, feline) SDF-1 is active on mouse cells. SDF-1 α and β are reported to be monomers at neutral pH and physiologic ionic strength (4). SDF-1 α is also reported to form dimers in the presence of heparansulfate (8). On the cell surface, this may well facilitate SDF-1 interaction with its two receptors, CXCR4 and syndecan-4 (9). Heparin sulfate is known to protect SDF-1 from proteolysis, and CXCR4 exists constitutively as a dimer (9 - 11). Among its many functions, CXCL12 is known to influence lymphopoiesis, regulate patterning and cell number of neural progenitors, and promote angiogenesis (12, 13). It also enhances the survival of myeloid progenitor cells.

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