

## DESCRIPTION

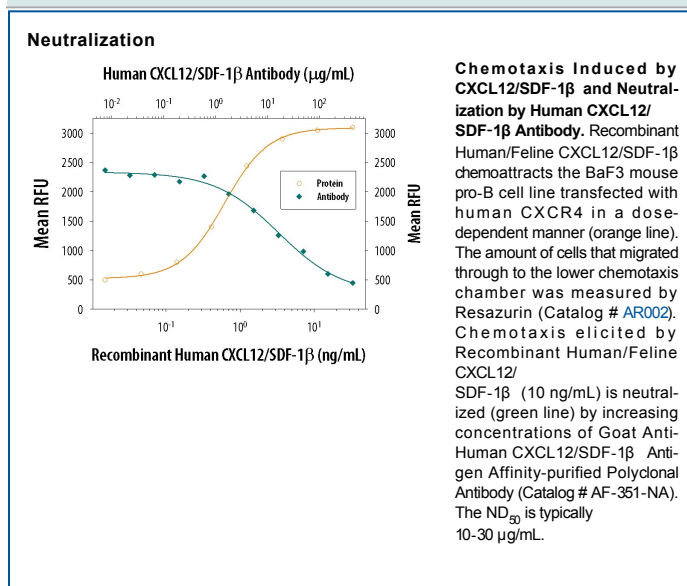
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human CXCL12/SDF-1 $\beta$ in direct ELISAs and Western blots. Neutralizes 60-80% of the biological activity of CXCL12/SDF-1 $\beta$ and does not neutralize the biological activity of SDF-1 $\alpha$ . In Western blots, less than 5% cross-reactivity with recombinant human SDF-1 $\alpha$ is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	<i>E. coli</i> -derived recombinant human CXCL12/SDF-1 $\beta$ Lys22-Met93 Accession # P48061
<b>Endotoxin Level</b>	<0.10 EU per 1 $\mu$ g of the antibody by the LAL method.
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

## 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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	0.1 $\mu$ g/mL	Recombinant Human/Feline CXCL12/SDF-1 $\beta$ aa 19-93 (Catalog # <a href="#">2716-SD</a> )
<b>Neutralization</b>	Measured by its ability to neutralize CXCL12/SDF-1 $\beta$ -induced chemotaxis in the BaF3 mouse pro-B cell line transfected with human CXCR4. The Neutralization Dose (ND <sub>50</sub> ) is typically 10-30 $\mu$ g/mL in the presence of 10 ng/mL Recombinant Human/Feline CXCL12/SDF-1 $\beta$ .	

## DATA



## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

## BACKGROUND

CXCL12, also known as SCYB12, PBSF and SDF-1 $\beta$ , is an 8.3 kDa, heparin-binding member of the CXC (or alpha-) family of chemokines (1, 2). Feline CXCL12( $\beta$ ) 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  $\beta$ -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 $\beta$  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  $\beta$ -isoform, alternate splicing of the feline SDF-1 gene generates an  $\alpha$ -isoform. The alpha isoform is identical to SDF-1 $\beta$ , but shorter by four aa's at the C-terminus (3). Although  $\alpha$ - and  $\beta$ -isoforms show similar activity, SDF-1 $\alpha$  is differentially processed, and different cells secrete the two isoforms (5, 7). Mature feline SDF-1 $\beta$  is 96%, 97% and 100% aa identical to rat, mouse and human SDF-1 $\beta$ , respectively. Human (and by inference, feline) SDF-1 is active on mouse cells. SDF-1 $\alpha$  and  $\beta$  are reported to be monomers at neutral pH and physiologic ionic strength (4). SDF-1 $\alpha$  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.

## References:

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