

**DESCRIPTION**

<b>Source</b>	Mouse myeloma cell line, NS0-derived						
	<table border="1" style="width:100%; border-collapse: collapse;"> <tr> <td style="width:33%; text-align: center;">                     Mouse CD117/c-kit (Gln26-Thr523) Accession # P05532                 </td> <td style="width:33%; text-align: center;">IEGRMDP</td> <td style="width:33%; text-align: center;">                     Mouse IgG<sub>2A</sub> (Glu98-Lys330)                 </td> </tr> <tr> <td style="text-align: left;">N-terminus</td> <td></td> <td style="text-align: right;">C-terminus</td> </tr> </table>	Mouse CD117/c-kit (Gln26-Thr523) Accession # P05532	IEGRMDP	Mouse IgG <sub>2A</sub> (Glu98-Lys330)	N-terminus		C-terminus
Mouse CD117/c-kit (Gln26-Thr523) Accession # P05532	IEGRMDP	Mouse IgG <sub>2A</sub> (Glu98-Lys330)					
N-terminus		C-terminus					

**N-terminal Sequence Analysis** Gln26 predicted: No results obtained, sequencing might be blocked

**Structure / Form** Disulfide-linked homodimer

**Predicted Molecular Mass** 82.6 kDa (monomer)

**SPECIFICATIONS**

<b>SDS-PAGE</b>	100-125 kDa, reducing conditions
<b>Activity</b>	Measured by its binding ability in a functional ELISA. When Recombinant Mouse CD117/c-kit Fc Chimera is present at 0.5 µg/mL, the concentration of Recombinant Mouse SCF/c-kit Ligand (Catalog # 455-MC) that produces 50% of the optimal binding response is approximately 3-12 ng/mL.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 µg/mL in 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>● 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

Stem Cell Factor Receptor (SCF R), also known as c-Kit and CD117, is a widely expressed 145 kDa receptor tyrosine kinase. It is the cellular homolog of the feline sarcoma virus protein, v-Kit. Binding of SCF R to SCF, also known as Steel Factor and Kit Ligand, promotes the survival, differentiation, and mobilization of progenitor cells in multiple lineages (1-4). Mutations or deletions of SCF R cause a wide variety of malignancies as well as pigmentation disorders and sterility (5, 6). Mature mouse SCF R consists of a 503 amino acid (aa) extracellular domain (ECD) with five tandem immunoglobulin-like domains, a 21 aa transmembrane segment, and a 431 aa cytoplasmic domain with the split tyrosine kinase domain (7). Within the ECD, mouse SCF R shares 73% and 88% aa sequence identity with human and rat SCF R, respectively. Alternative splicing of mouse SCF R generates a truncated intracellular isoform that corresponds to the C-terminal half of the tyrosine kinase domain (8). SCF is expressed as transmembrane and soluble noncovalent homodimers (9). One SCF dimer binds to two molecules of SCF R, inducing receptor dimerization and activation (9). Transmembrane SCF induces more prolonged signaling through SCF R compared to soluble SCF (10). Rat SCF is active on mouse and human cells, but human SCF is only weakly active on mouse cells (11). A 100 kDa glycosylated ECD fragment of SCF R can be shed into the circulation by TACE/ADAM17, and this fragment inhibits the interaction of SCF with transmembrane SCF R (12, 13). SCF is a primary growth and activation factor for mast cells and eosinophils (14). SCF R expression on mast cells enables them to infiltrate SCF-secreting tumors where they promote tumor growth and induce local immune suppression (15). SCF R is up-regulated on dendritic cells by Th2- or Th17-biasing stimuli, and it is required for subsequent dendritic cell induction of Th2 and Th17 responses (16). SCF R protects vascular smooth muscle cells from apoptosis and assists in the recovery of cardiac function following myocardial infarction (17, 18).

**References:**

1. Kimura, Y. *et al.* (2011) PLoS ONE **6**:e26918.
2. Tallini, Y.N. *et al.* (2009) Proc. Natl. Acad. Sci. USA **106**:1808.
3. Bashamboo, A. *et al.* (2006) J. Cell Sci. **119**:3039.
4. Sun, L. *et al.* (2004) J. Clin. Invest. **113**:1364.
5. Pittoni, P. *et al.* (2011) Oncogene **30**:757.
6. Sartini, S. *et al.* (2011) Curr. Med. Chem. **18**:2893.
7. Qiu, F.H. *et al.* (1988) EMBO J. **7**:1003.
8. Zayas, J. *et al.* (2008) Stem Cells Dev. **17**:343.
9. Lemmon, M.A. *et al.* (1997) J. Biol. Chem. **272**:6311.
10. Miyazawa, K. *et al.* (1995) Blood **85**:641.
11. Martin, F.H. *et al.* (1990) Cell **63**:203.
12. Turner, A.M. *et al.* (1995) Blood **85**:2052.
13. Cruz, A.C. *et al.* (2004) J. Biol. Chem. **279**:5612.
14. Jamur, M.C. and C. Oliver (2011) Front. Biosci. (School Ed.) **3**:1390.
15. Huang, B. *et al.* (2008) Blood **112**:1269.
16. Krishnamoorthy, N. *et al.* (2008) Nat. Med. **14**:565.
17. Wang, C.-H. *et al.* (2007) Arterioscler. Thromb. Vasc. Biol. **27**:540.
18. Kanellakis, P. *et al.* (2006) Cardiovasc. Res. **70**:117.