

**DESCRIPTION**

<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects the $\alpha$ subunit of mouse HGF R/c-MET in direct ELISAs and Western blots.
<b>Source</b>	Monoclonal Rat IgG <sub>2A</sub> Clone # 118624
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant mouse HGF R/c-MET Glu25-Asn929 Accession # P16056
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in PBS with Trehalose.

**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>	1 $\mu$ g/mL	Recombinant Mouse HGF R/c-MET Fc Chimera (Catalog # 527-ME)

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 0.5 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**

HGF R, also known as Met (from *N*-methyl-*N*-nitro-*N*-nitrosoguanidine induced), is a glycosylated receptor tyrosine kinase that plays a central role in epithelial morphogenesis and cancer development. HGF R is synthesized as a single chain precursor which undergoes cotranslational proteolytic cleavage. This generates a mature HGF R that is a disulfide-linked dimer composed of a 50 kDa extracellular  $\alpha$  chain and a 145 kDa transmembrane  $\beta$  chain (1, 2). The extracellular domain (ECD) contains a seven bladed  $\beta$ -propeller sema domain, a cysteine-rich PSI/MRS, and four Ig-like E-set domains, while the cytoplasmic region includes the tyrosine kinase domain (3, 4). An alternately spliced form of mouse HGF R lacks a cytoplasmic juxtamembrane region important for regulation of signal transduction (5, 6). The sema domain, which is formed by both the  $\alpha$  and  $\beta$  chains of HGF R, mediates both ligand binding and receptor dimerization (3, 7). Ligand-induced tyrosine phosphorylation in the cytoplasmic region activates the kinase domain and provides docking sites for multiple SH2-containing molecules (8, 9). HGF stimulation induces HGF R down-regulation via internalization and proteasome-dependent degradation (10). In the absence of ligand, HGF R forms noncovalent complexes with a variety of membrane proteins including CD44v6, CD151, EGF R, Fas, integrin  $\alpha$ 6/ $\beta$ 4, plexins B1, 2, 3, and MSP R/Ron (11-18). Ligation of one complex component triggers activation of the other, followed by cooperative signaling effects (11-18). Formation of some of these heteromeric complexes is a requirement for epithelial cell morphogenesis and tumor cell invasion (11, 15, 16). Paracrine induction of epithelial cell scattering and branching tubulogenesis results from the stimulation of HGF R on undifferentiated epithelium by HGF released from neighboring mesenchymal cells (19). Genetic polymorphisms, chromosomal translocation, overexpression, and additional splicing and proteolytic cleavage of HGF R have been described in a wide range of cancers (1). Within the ECD, mouse HGF R shares 87%, 87%, and 94% amino acid sequence identity with canine, human, and rat HGF R, respectively.

**References:**

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