**DESCRIPTION**

Species Reactivity: Human  
Specificity: Detects human HGF R/c-MET.  
Source: Monoclonal Mouse IgG1, Clone # 95106  
Purification: Protein A or G purified from hybridoma culture supernatant  
Immunogen: Mouse myeloma cell line NS0-derived recombinant human HGF R/c-MET  
Glu25-Thr932  
Accession # P08581  
Formulation: Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.  
*Small pack size (SP) is supplied either lyophilized or as a 0.2 μm filtered solution in PBS.

**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.

<table>
<thead>
<tr>
<th>Recommended Concentration</th>
<th>Sample</th>
</tr>
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<tbody>
<tr>
<td>Flow Cytometry 2.5 μg/10⁶ cells</td>
<td>See Below</td>
</tr>
<tr>
<td>CyTOF-ready</td>
<td>Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.</td>
</tr>
</tbody>
</table>

**DATA**

Detection of HGF R/c-MET in MDA-MB-231 Human Cell Line by Flow Cytometry. MDA-MB-231 human breast cancer cell line was stained with Human HGF R/c-MET Monoclonal Antibody (Catalog # MAB3582, filled histogram) or isotype control antibody (Catalog # MAB002, open histogram), followed by Allophycocyanin-conjugated Anti-Mouse IgG F(ab’)₂ Secondary Antibody (Catalog # F0101B).

**PREPARATION AND STORAGE**

Reconstitution: Reconstitute at 0.5 mg/mL in sterile PBS.  
Shipping: The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.  
*Small pack size (SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C.  
Stability & Storage: Use a manual defrost freezer and avoid repeated freeze-thaw cycles.  
- 12 months from date of receipt, -20 to -70 °C as supplied.  
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.  
- 6 months, -20 to -70 °C under sterile conditions after reconstitution.
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 α chain and a 145 kDa transmembrane β chain (1, 2). The extracellular domain (ECD) contains a seven bladed β-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). Proteolysis and alternate splicing generate additional forms of human HGF R which either lack of the kinase domain, consist of secreted extracellular domains, or are deficient in proteolytic separation of the α and β chains (5-7). The sema domain, which is formed by both the α and β chains of HGF R, mediates both ligand binding and receptor dimerization (3, 8). Ligand-induced tyrosine phosphorylation in the cytoplasmic region activates the kinase domain and provides docking sites for multiple SH2-containing molecules (9, 10). HGF stimulation induces HGF R downregulation via internalization and proteasome-dependent degradation (11). In the absence of ligand, HGF R forms non-covalent complexes with a variety of membrane proteins including CD44v6, CD151, EGF R, Fas, Integrin α6/β4, Plexins B1, 2, 3, and MSP R/Ron (12-19). Ligation of one complex component triggers activation of the other, followed by cooperative signaling effects (12-19). Formation of some of these heteromeric complexes is a requirement for epithelial cell morphogenesis and tumor cell invasion (12, 16, 17). 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 (20). Genetic polymorphisms, chromosomal translocation, over-expression, and additional splicing and proteolytic cleavage of HGF R have been described in a wide range of cancers (1). Within the ECD, human HGF R shares 86-88% amino acid sequence identity with canine, mouse, and rat HGF R.

References: