Human HGF R/c-MET
Alexa Fluor® 750-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 95106

Catalog Number: FAB3582S

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human HGF R/c-MET.
Source	Monoclonal Mouse IgG_1 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
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.*

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10^6 cells	MDA-MB-231 human breast cancer cell line

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.

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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:

1. Birchmeier, C. *et al.* (2003) Nat. Rev. Mol. Cell Biol. **4**:915.
2. Corso, S. *et al.* (2005) Trends Mol. Med. **11**:284.
3. Gherardi, E. *et al.* (2003) Proc. Natl. Acad. Sci. USA **100**:12039.
4. Park, M. *et al.* (1987) Proc. Natl. Acad. Sci. USA **84**:6379.
5. Crepaldi, T. *et al.* (1994) J. Biol. Chem. **269**:1750.
6. Prat, M. *et al.* (1991) Mol. Cell. Biol. **12**:5954.
7. Rodrigues, G.A. *et al.* (1991) Mol. Cell. Biol. **11**:2962.
8. Kong-Beltran, M. *et al.* (2004) Cancer Cell **6**:75.
9. Naldini, L. *et al.* (1991) Mol. Cell. Biol. **11**:1793.
10. Ponzetto, C. *et al.* (1994) Cell **77**:261.
11. Jeffers, M. *et al.* (1997) Mol. Cell. Biol. **17**:799.
12. Orian-Rousseau, V. *et al.* (2002) Genes Dev. **16**:3074.
13. Klosek, S.K. *et al.* (2005) Biochem. Biophys. Res. Commun. **336**:408.
14. Jo, M. *et al.* (2000) J. Biol. Chem. **275**:8806.
15. Wang, X. *et al.* (2002) Mol. Cell **9**:411.
16. Trusolino, L. *et al.* (2001) Cell **107**:643.
17. Giordano, S. *et al.* (2002) Nat. Cell Biol. **4**:720.
18. Conrotto, P. *et al.* (2004) Oncogene **23**:5131.
19. Follenzi, A. *et al.* (2000) Oncogene **19**:3041.
20. Sonnenberg, E. *et al.* (1993) J. Cell Biol. **123**:223.

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