

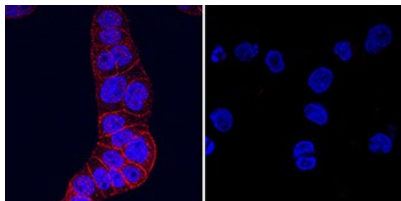
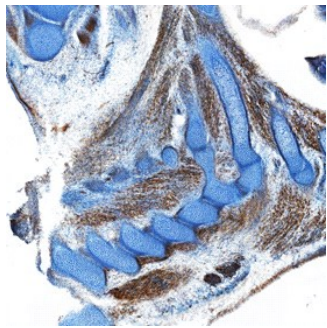
DESCRIPTION	
Species Reactivity	Mouse
Specificity	Detects mouse HGF R/c-MET in direct ELISAs and Western blots. In direct ELISAs, approximately 10% cross-reactivity with recombinant human (rh) HGF R and less than 1% cross-reactivity with rhMSP R is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf21-derived recombinant mouse HGF R/c-MET Glu25-Asn929 Accession # P16056
Endotoxin Level	<0.01 EU per 1 µg of the antibody by the LAL method.
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.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Mouse HGF R/c-MET Fc Chimera (Catalog # 527-ME)
Immunocytochemistry	5-15 µg/mL	See Below
Immunohistochemistry	5-15 µg/mL	See Below
Blockade of Receptor-ligand Interaction	In a functional ELISA, 0.3-1 µg/mL of this antibody will block 50% of the binding of 5 ng/mL of Recombinant Human HGF (Catalog # 256-GF) to immobilized Recombinant Mouse HGF R/c-MET Fc Chimera (Catalog # 527-ME) coated at 1 µg/mL (100 µL/well). At 20 µg/mL, this antibody will block >90% of the binding.	

DATA

<p>Immunocytochemistry</p>  <p>HGF R/c-MET in HT-29 and U937 Human Cell Lines. HGF R/c-MET was detected in immersion fixed HT-29 human colon adenocarcinoma cell line (positive control, left panel) and U937 human histiocytic lymphoma cell line (negative control, right panel) using Goat Anti-Mouse HGF R/c-MET Antigen Affinity-purified Polyclonal Antibody (Catalog # AF527) at 5 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (red; Catalog # NL001) and counterstained with DAPI (blue). Specific staining was localized to plasma membrane. View our protocol for Fluorescent ICC Staining of Cells on Coverslips.</p>	<p>Immunohistochemistry</p>  <p>HGF R/c-MET in Mouse Embryo. HGF R/c-MET was detected in immersion fixed frozen sections of mouse embryo (15 d.p.c.) using Goat Anti-Mouse HGF R/c-MET Antigen Affinity-purified Polyclonal Antibody (Catalog # AF527) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to cytoplasm in muscle cells. View our protocol for Chromogenic IHC Staining of Frozen Tissue Sections.</p>
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PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 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. <ul style="list-style-type: none"> ● 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). 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 α and β 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 downregulation *via* internalization and proteasome-dependent degradation (10). In the absence of ligand, HGF R forms non-covalent 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, over-expression, 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:

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. Chan, A.M. *et al.* (1988) *Oncogene* **2**:593.
5. Lee, C.-C. and K.M. Yamada (1994) *J. Biol. Chem.* **269**:19457.
6. Lee, C.-C., *et al.* (1995) *J. Biol. Chem.* **270**:507.
7. Kong-Beltran, M. *et al.* (2004) *Cancer Cell* **6**:75.
8. Naldini, L. *et al.* (1991) *Mol. Cell. Biol.* **11**:1793.
9. Ponzetto, C. *et al.* (1994) *Cell* **77**:261.
10. Jeffers, M. *et al.* (1997) *Mol. Cell. Biol.* **17**:799.
11. Orian-Rousseau, V. *et al.* (2002) *Genes Dev.* **16**:3074.
12. Klosek, S.K. *et al.* (2005) *Biochem. Biophys. Res. Commun.* **336**:408.
13. Jo, M. *et al.* (2000) *J. Biol. Chem.* **275**:8806.
14. Wang, X. *et al.* (2002) *Mol. Cell* **9**:411.
15. Trusolino, L. *et al.* (2001) *Cell* **107**:643.
16. Giordano, S. *et al.* (2002) *Nat. Cell Biol.* **4**:720.
17. Conrotto, P. *et al.* (2004) *Oncogene* **23**:5131.
18. Follenzi, A. *et al.* (2000) *Oncogene* **19**:3041.
19. Sonnenberg, E. *et al.* (1993) *J. Cell Biol.* **123**:223.