

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 Sf 21-derived recombinant mouse HGF R/c-MET Glu25-Asn929 Accession # P16056
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *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.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Blockade of Receptor-ligand Interaction	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

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

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.

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