

## Mouse HGF R/c-MET Biotinylated Antibody

Antigen Affinity-purified Polyclonal Goat IgG Catalog Number: BAF527

DESCRIPTION			
Species Reactivity	Mouse		
Specificity	Detects mouse HGF R/c-MET in ELISAs and Western blots. In sandwich immunoassays, less than 0.2% cross-reactivity with recombinant human HGF R, recombinant mouse (rm) HGF A, and rmMSP R is observed.		
Source	Polyclonal Goat IgG		
Purification	Antigen Affinity-purified		
Immunogen	S. frugiperda insect ovarian cell line Sf 21-derived recombinant mouse HGF R/c-MET Glu25-Asn929 Accession # P16056		
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.		
APPLICATIONS			
Please Note: Optimal diluti	ons should be determined by each laborate	ory for each applica	ation. General Protocols are available in the Technical Information section on our website.
		mmended centration	Sample
Western Blot	0.1 μ	g/mL	Recombinant Mouse HGF R/c-MET Fc Chimera (Catalog # 527-ME)
Immunohistochemis	try 5-15	μg/mL	Immersion fixed frozen sections of mouse embryo (E13)
Mouse HGF R/c-MET Sandwich Immunoassay			Reagent
ELISA Capture	2-8 μ	g/mL	Mouse HGF R/c-MET Antibody (Catalog # MAB5271)
ELISA Detection	0.1-0	.4 μg/mL	Mouse HGF R/c-MET Biotinylated Antibody (Catalog # BAF527)
Standard			Recombinant Mouse HGF R/c-MET Fc Chimera (Catalog # 527-ME)
PREPARATION AND S	TORAGE		
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.		
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.		





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## BACKGROUND

HGF R, also known as Met (from *N*-methyl-*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 downregulation 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

## References:

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