

DESCRIPTION

Species Reactivity	Canine
Specificity	Detects canine HGF R/c-MET in ELISAs and Western blots. In sandwich immunoassays, less than 0.2% cross-reactivity with recombinant human HGF R and remombinant mouse HGF R is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant canine HGF R/c-MET Glu25-Leu935 Accession # Q75ZY9
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 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.

ELISA Capture (Matched Antibody Pair)	Optimal dilution of this antibody should be experimentally determined.
ELISA Detection (Matched Antibody Pair)	Optimal dilution of this antibody should be experimentally determined.
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.

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 posttranslational 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 region, and four Ig-like E-set domains, while the cytoplasmic region includes a tyrosine kinase domain (3). The sema domain, which is formed by both the α and β chains of HGF R, mediates both ligand binding and receptor dimerization (3, 4). Ligand-induced tyrosine phosphorylation in the cytoplasmic region activates the kinase domain and provides docking sites for multiple SH2-containing molecules (5, 6). HGF stimulation induces HGF R downregulation *via* internalization and proteasome-dependent degradation (7). 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, B2, and B3, and MSP R/Ron (8-15). Ligation of one complex component triggers activation of the other, followed by cooperative signaling effects (8-15). Formation of some of these heteromeric complexes is a requirement for epithelial cell morphogenesis and tumor cell invasion (8, 12, 13). HGF released from neighboring mesenchymal cells stimulates HGF R on undifferentiated epithelium and induces epithelial cell scattering and branching tubulogenesis (16). Genetic polymorphisms, chromosomal translocation, overexpression, and additional splicing and proteolytic cleavage of HGF R have been described in a wide range of cancers (1). Within the ECD, canine HGF R shares 85%-88% amino acid sequence identity with human, mouse and rat HGF R.

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