

<b>Cat. No:</b>	MAB-53309
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100 ug
<b>Clone:</b>	13DH2
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rb
<b>Isotype:</b>	IgG
<b>Reactivity:</b>	Hu, Ms, Rt
<b>Applications:</b>	WB 1:1000
<b>Molecular Weight:</b>	145 kDa

**Purification:** Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Tyr1349 of human Met.

**Background:** Met, a high affinity tyrosine kinase receptor for hepatocyte growth factor (HGF, also known as scatter factor) is a disulfide-linked heterodimer made of 45 kDa  $\alpha$ - and 145 kDa  $\beta$ -subunits (1,2). The  $\alpha$ -subunit and the amino-terminal region of the  $\beta$ -subunit form the extracellular domain. The remainder of the  $\beta$ -chain spans the plasma membrane and contains a cytoplasmic region with tyrosine kinase activity. Interaction of Met with HGF results in autophosphorylation at multiple tyrosines, which recruit several downstream signaling components, including Gab1, c-Cbl, and PI3 kinase (3). These fundamental events are important for all of the biological functions involving Met kinase activity. The addition of a phosphate at cytoplasmic Tyr1003 is essential for Met protein ubiquitination and degradation (4). Phosphorylation at Tyr1234/1235 in the Met kinase domain is critical for kinase activation. Phosphorylation at Tyr1349 in the Met cytoplasmic domain provides a direct binding site for Gab1 (5). Altered Met levels and/or tyrosine kinase activities are found in several types of tumors, including renal, colon, and breast. Thus, Met is an attractive cancer therapeutic and diagnostic target (6,7). Phospho-Met (Tyr1349) (13DH2) Rabbit mAb detects endogenous levels of Met only when phosphorylated at tyrosine 1349. This antibody may crossreact with other activated protein tyrosine kinases.

<b>Form:</b>	liquid
<b>Buffer:</b>	PBS with 0.02% sodium azide, 50% glycerol, pH7.3
<b>Storage:</b>	Store at -20°C, and avoid repeat freeze-thaw cycles

## References

(1) Cooper, C.S. et al. (1984) Nature 311, 29-33. (2) Bottaro, D.P. et al. (1991) Science 251, 802-4. (3) Bardelli, A. et al. (1997) Oncogene 15, 3103-11. (4) Taher, T.E. et al. (2002) J Immunol 169, 3793-800. (5) Schaeper, U. et al. (2000) J Cell Biol 149, 1419-32. (6) Eder, J.P. et al. (2009) Clin Cancer Res 15, 2207-14. (7) Sattler, M. and Salgia, R. (2009) Update Cancer Ther 3, 109-118..

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