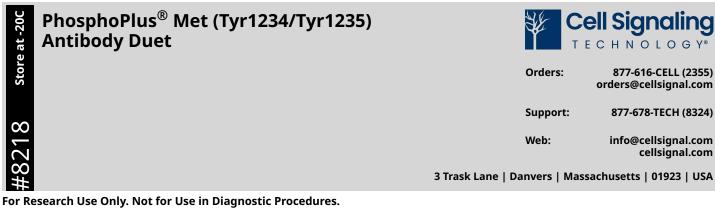
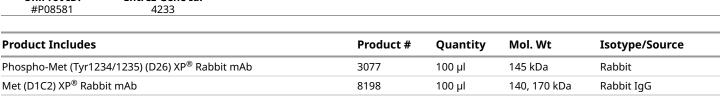
UniProt ID:



**Entrez-Gene Id:** 



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Description	PhosphoPlus <sup>®</sup> Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.
Storage	Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.
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). Research studies have shown that altered Met levels and/or tyrosine kinase activities are found in several types of tumors, including renal, colon, and breast. Thus, investigators have concluded that Met is an attractive potential cancer therapeutic and diagnostic target (6,7).
Background References	1. Cooper, C.S. et al. (1984) <i>Nature</i> 311, 29-33. 2. Bottaro, D.P. et al. (1991) <i>Science</i> 251, 802-4. 3. Bardelli, A. et al. (1997) <i>Oncogene</i> 15, 3103-11. 4. Taher, T.E. et al. (2002) <i>J Immunol</i> 169, 3793-800. 5. Schaeper, U. et al. (2000) <i>J Cell Biol</i> 149, 1419-32. 6. Eder, J.P. et al. (2009) <i>Clin Cancer Res</i> 15, 2207-14. 7. Sattler, M. and Salgia, R. (2009) <i>Update Cancer Ther</i> 3, 109-118.
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