

**RON Antibody (N-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7674a****Specification**

---

**RON Antibody (N-term) Blocking Peptide - Product Information**Primary Accession [Q04912](#)**RON Antibody (N-term) Blocking Peptide - Additional Information****Gene ID** 4486**Other Names**

Macrophage-stimulating protein receptor, MSP receptor, CDw136, Protein-tyrosine kinase 8, p185-Ron, CD136, Macrophage-stimulating protein receptor alpha chain, Macrophage-stimulating protein receptor beta chain, MST1R, PTK8, RON

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7674a](/product/products/AP7674a) was selected from the N-term region of human RON. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**RON Antibody (N-term) Blocking Peptide - Protein Information****Name** MST1R**Synonyms** PTK8, RON**Function**

Receptor tyrosine kinase that transduces signals from the extracellular matrix into the cytoplasm by binding to MST1 ligand. Regulates many physiological processes including cell survival, migration and differentiation. Ligand binding at the cell surface induces autophosphorylation of RON on its intracellular domain that provides docking sites for downstream signaling molecules. Following activation by ligand, interacts with the PI3-kinase subunit PIK3R1, PLCG1 or the adapter GAB1. Recruitment of these downstream effectors by RON leads to the activation of several signaling cascades including the RAS-ERK, PI3 kinase-AKT, or PLCgamma-PKC. RON signaling activates the wound healing response by promoting epithelial cell migration, proliferation as well

as survival at the wound site. Also plays a role in the innate immune response by regulating the migration and phagocytic activity of macrophages. Alternatively, RON can also promote signals such as cell migration and proliferation in response to growth factors other than MST1 ligand.

**Cellular Location**

Membrane; Single-pass type I membrane protein.

**Tissue Location**

Expressed in colon, skin, lung and bone marrow.

**RON Antibody (N-term) Blocking Peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

**RON Antibody (N-term) Blocking Peptide - Images****RON Antibody (N-term) Blocking Peptide - Background**

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the  $\gamma$  phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains. The tyrosine kinase (TK) group is mainly involved in the regulation of cell-cell interactions such as differentiation, adhesion, motility and death. There are currently about 90 TK genes sequenced, 58 are of receptor protein TK (e.g. EGFR, EPH, FGFR, PDGFR, TRK, and VEGFR families), and 32 of cytosolic TK (e.g. ABL, FAK, JAK, and SRC families).

**RON Antibody (N-term) Blocking Peptide - References**

Maggiore, P., et al., Exp. Cell Res. 288(2):382-389 (2003). Santoro, M.M., et al., Dev. Cell 5(2):257-271 (2003). Penengo, L., et al., Oncogene 22(24):3669-3679 (2003). Zhou, Y.Q., et al., Oncogene 22(2):186-197 (2003). Danilkovitch-Miagkova, A., et al., Proc. Natl. Acad. Sci. U.S.A. 100(8):4580-4585 (2003).