

**MST1R / RON Antibody (Internal)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ALS10890****Specification**

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**MST1R / RON Antibody (Internal) - Product Information**

Application	IHC-P, E
Primary Accession	<a href="#">Q04912</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	152kDa KDa
Dilution	IHC-P~~N/A E~~N/A

**MST1R / RON Antibody (Internal) - Additional Information****Gene ID** 4486**Other Names**

Macrophage-stimulating protein receptor, MSP receptor, 2.7.10.1, 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**

Human MST1R / RON. BLAST analysis of the peptide immunogen showed no homology with other human proteins, except FANCE (50%).

**Reconstitution & Storage**

Long term: -70°C; Short term: +4°C

**Precautions**

MST1R / RON Antibody (Internal) is for research use only and not for use in diagnostic or therapeutic procedures.

**MST1R / RON Antibody (Internal) - 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.

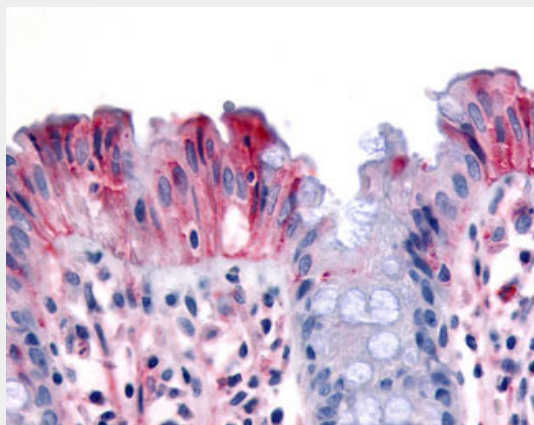
**Volume**

50 µl

**MST1R / RON Antibody (Internal) - Protocols**

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

**MST1R / RON Antibody (Internal) - Images**

Anti-MST1R / RON antibody ALS10890 IHC of human colon, surface epithelium.

**MST1R / RON Antibody (Internal) - Background**

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. Plays also 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.

#### **MST1R / RON Antibody (Internal) - References**

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Collesi C.,et al.Mol. Cell. Biol. 16:5518-5526(1996).  
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Muzny D.M.,et al.Nature 440:1194-1198(2006).  
Ponzetto C.,et al.Mol. Cell. Biol. 13:4600-4608(1993).