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MERTK Antibody (N-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7662a

Specification

MERTK Antibody (N-term) - Product Information

Application	**IHC-P,E**
Primary Accession	Q12866
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Antigen Region	**21-51**

MERTK Antibody (N-term) - Additional Information

Gene ID 10461

Other Names
Tyrosine-protein kinase Mer, Proto-oncogene c-Mer, Receptor tyrosine kinase MerTK, MERTK, MER

Target/Specificity
This MERTK antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 21-51 amino acids from the N-terminal region of human MERTK.

Dilution
IHC-P~~1:10~50

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
MERTK Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

MERTK Antibody (N-term) - Protein Information

Name MERTK

Synonyms MER

Function Receptor tyrosine kinase that transduces signals from the extracellular matrix into the cytoplasm by binding to several ligands including LGALS3, TUB, TULP1 or GAS6. Regulates many

physiological processes including cell survival, migration, differentiation, and phagocytosis of apoptotic cells (efferocytosis). Ligand binding at the cell surface induces autophosphorylation of MERTK on its intracellular domain that provides docking sites for downstream signaling molecules. Following activation by ligand, interacts with GRB2 or PLCG2 and induces phosphorylation of MAPK1, MAPK2, FAK/PTK2 or RAC1. MERTK signaling plays a role in various processes such as macrophage clearance of apoptotic cells, platelet aggregation, cytoskeleton reorganization and engulfment (PubMed:32640697). Functions in the retinal pigment epithelium (RPE) as a regulator of rod outer segments fragments phagocytosis. Also plays an important role in inhibition of Toll-like receptors (TLRs)-mediated innate immune response by activating STAT1, which selectively induces production of suppressors of cytokine signaling SOCS1 and SOCS3.

Cellular Location
Cell membrane; Single-pass type I membrane protein

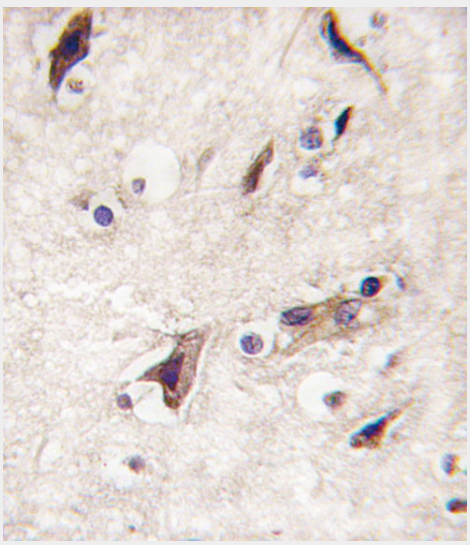
Tissue Location
Not expressed in normal B- and T-lymphocytes but is expressed in numerous neoplastic B- and T-cell lines. Highly expressed in testis, ovary, prostate, lung, and kidney, with lower expression in spleen, small intestine, colon, and liver

MERTK Antibody (N-term) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

MERTK Antibody (N-term) - Images



Formalin-fixed and paraffin-embedded human brain tissue reacted with MERK antibody (N-term), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been

evaluated.

MERTK Antibody (N-term) - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the g 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.

MERTK Antibody (N-term) - References

Thompson, D.A., et al., Am. J. Hum. Genet. 70(1):224-229 (2002).
Graham, D.K., et al., Cell Growth Differ. 5(6):647-657 (1994).
Weier, H.U., et al., Cytogenet. Cell Genet. 84 (1-2), 91-92 (1999).