

**MCT-1 Antibody (N-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP6655a****Specification**

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**MCT-1 Antibody (N-term) Blocking Peptide - Product Information**Primary Accession [Q9ULC4](#)**MCT-1 Antibody (N-term) Blocking Peptide - Additional Information****Gene ID** 28985**Other Names**

Malignant T-cell-amplified sequence 1, MCT-1, Multiple copies T-cell malignancies, MCTS1, MCT1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP6655a](/products/AP6655a) was selected from the N-term region of human MCT-1. 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.

**MCT-1 Antibody (N-term) Blocking Peptide - Protein Information****Name** MCTS1**Synonyms** MCT1**Function**

Anti-oncogene that plays a role in cell cycle regulation; decreases cell doubling time and anchorage-dependent growth; shortens the duration of G1 transit time and G1/S transition. When constitutively expressed, increases CDK4 and CDK6 kinases activity and CCND1/cyclin D1 protein level, as well as G1 cyclin/CDK complex formation. Involved in translation initiation; promotes recruitment of aminoacylated initiator tRNA to P site of 40S ribosomes. Can promote release of deacylated tRNA and mRNA from recycled 40S subunits following ABCE1-mediated dissociation of post-termination ribosomal complexes into subunits. Plays a role as translation enhancer; recruits the density-regulated protein/DENR and binds to the cap complex of the 5'-terminus of mRNAs, subsequently altering the mRNA translation profile; up-regulates protein levels of BCL2L2, TFDP1, MRE11, CCND1 and E2F1, while mRNA levels remains constant. Hyperactivates DNA damage

signaling pathway; increased gamma-irradiation-induced phosphorylation of histone H2AX, and induces damage foci formation. Increases the overall number of chromosomal abnormalities such as larger chromosomes formation and multiple chromosomal fusions when overexpressed in gamma- irradiated cells. May play a role in promoting lymphoid tumor development: lymphoid cell lines overexpressing MCTS1 exhibit increased growth rates and display increased protection against apoptosis. May contribute to the pathogenesis and progression of breast cancer via promotion of angiogenesis through the decline of inhibitory THBS1/thrombospondin-1, and inhibition of apoptosis. Involved in the process of proteasome degradation to down-regulate Tumor suppressor p53/TP53 in breast cancer cell; Positively regulates phosphorylation of MAPK1 and MAPK3. Involved in translation initiation; promotes aminoacylated initiator tRNA to P site of 40S ribosomes. Can promote release of deacylated tRNA and mRNA from recycled 40S subunits following ABCE1-mediated dissociation of post-termination ribosomal complexes into subunits.

**Cellular Location**

Cytoplasm. Note=Nuclear relocalization after DNA damage

**Tissue Location**

Ubiquitous. Over-expressed in T-cell lymphoid cell lines and in non-Hodgkin lymphoma cell lines as well as in a subset of primary large B-cell lymphomas.

**MCT-1 Antibody (N-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

**MCT-1 Antibody (N-term) Blocking Peptide - Images****MCT-1 Antibody (N-term) Blocking Peptide - Background**

MCTS1 play a role in cell cycle regulation; decreases cell doubling time and anchorage-dependent growth; shortens the duration of G1 transit time and G1/S transition.

**MCT-1 Antibody (N-term) Blocking Peptide - References**

Kasiappan,R., Mol. Cancer Res. 7 (4), 536-548 (2009) Mazan-Mamczarz,K., Leuk. Res. 33 (3), 474-482 (2009) Shi,B., J. Cell. Biochem. 90 (1), 68-79 (2003)