10320 Camino Santa Fe, Suite G
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MLST8 / GBL Antibody (aa150-200)

Rabbit Polyclonal Antibody
Catalog # ALS12073

Specification

MLST8 / GBL Antibody (aa150-200) - Product Information

Application	**WB, IHC**
Primary Accession	Q9BVC4
Reactivity	**Human, Mouse, Rat, Monkey, Chicken, Bovine, Dog**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**36kDa KDa**

MLST8 / GBL Antibody (aa150-200) - Additional Information

Gene ID 64223

Other Names
Target of rapamycin complex subunit LST8, TORC subunit LST8, G protein beta subunit-like, Gable, Protein GbetaL, Mammalian lethal with SEC13 protein 8, mLST8, MLST8, GBL, LST8

Target/Specificity
A portion of amino acids 150-200 of human LST8 was used as the immunogen.

Reconstitution & Storage
Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles.

Precautions
MLST8 / GBL Antibody (aa150-200) is for research use only and not for use in diagnostic or therapeutic procedures.

MLST8 / GBL Antibody (aa150-200) - Protein Information

Name MLST8 {ECO:0000303|PubMed:34741373, ECO:0000312|HGNC:HGNC:24825}

Function
Subunit of both mTORC1 and mTORC2, which regulates cell growth and survival in response to nutrient and hormonal signals (PubMed:12718876, PubMed:15268862, PubMed:15467718, PubMed:24403073). mTORC1 is activated in response to growth factors or amino acids (PubMed:12718876, PubMed:15268862, PubMed:15467718, PubMed:24403073). In response to nutrients, mTORC1 is recruited to the lysosome

membrane and promotes protein, lipid and nucleotide synthesis by phosphorylating several substrates, such as ribosomal protein S6 kinase (RPS6KB1 and RPS6KB2) and EIF4EBP1 (4E-BP1) (PubMed:12718876, PubMed:15268862, PubMed:15467718, PubMed:24403073). In the same time, it inhibits catabolic pathways by phosphorylating the autophagy initiation components ULK1 and ATG13, as well as transcription factor TFEB, a master regulators of lysosomal biogenesis and autophagy (PubMed:24403073). The mTORC1 complex is inhibited in response to starvation and amino acid depletion (PubMed:24403073). Within mTORC1, LST8 interacts directly with MTOR and enhances its kinase activity (PubMed:12718876). In nutrient-poor conditions, stabilizes the MTOR- RPTOR interaction and favors RPTOR-mediated inhibition of MTOR activity (PubMed:12718876). mTORC2 is also activated by growth factors, but seems to be nutrient-insensitive (PubMed:15467718). mTORC2 seems to function upstream of Rho GTPases to regulate the actin cytoskeleton, probably by activating one or more Rho-type guanine nucleotide exchange factors (PubMed:15467718). mTORC2 promotes the serum-induced formation of stress-fibers or F-actin (PubMed:15467718). mTORC2 plays a critical role in AKT1 'Ser-473' phosphorylation, which may facilitate the phosphorylation of the activation loop of AKT1 on 'Thr-308' by PDK1 which is a prerequisite for full activation (PubMed:15467718). mTORC2 regulates the phosphorylation of SGK1 at 'Ser-422' (PubMed:15467718). mTORC2 also modulates the phosphorylation of PRKCA on 'Ser-657' (PubMed:15467718).

Cellular Location
Lysosome membrane. Cytoplasm {ECO:0000250|UniProtKB:Q9Z2K5}. Note=Targeting to lysosomal membrane depends on amino acid availability: mTORC1 is recruited to lysosome membranes via interaction with GTP-bound form of RagA/RRAGA (or RagB/RRAGB) in complex with the GDP-bound form of RagC/RRAGC (or RagD/RRAGD), promoting its mTORC1 recruitment to the lysosomes

Tissue Location
Broadly expressed, with highest levels in skeletal muscle, heart and kidney.

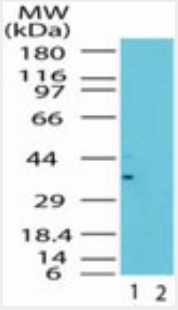
MLST8 / GBL Antibody (aa150-200) - Protocols

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

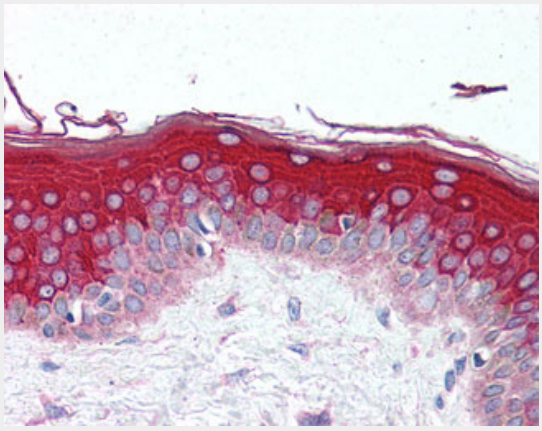
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

MLST8 / GBL Antibody (aa150-200) - Images

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Western blot of LST8 in the 1) absence and 2) presence of immunizing peptide in human brain...



Anti-GBL antibody IHC of human skin.

MLST8 / GBL Antibody (aa150-200) - Background

Subunit of both mTORC1 and mTORC2, which regulates cell growth and survival in response to nutrient and hormonal signals. mTORC1 is activated in response to growth factors or amino acids. Growth factor-stimulated mTORC1 activation involves a AKT1- mediated phosphorylation of TSC1-TSC2, which leads to the activation of the RHEB GTPase that potently activates the protein kinase activity of mTORC1. Amino acid-signaling to mTORC1 requires its relocalization to the lysosomes mediated by the Ragulator complex and the Rag GTPases. Activated mTORC1 up-regulates protein synthesis by phosphorylating key regulators of mRNA translation and ribosome synthesis. mTORC1 phosphorylates EIF4EBP1 and releases it from inhibiting the elongation initiation factor 4E (eiF4E). mTORC1 phosphorylates and activates S6K1 at 'Thr-389', which then promotes protein synthesis by phosphorylating PDCD4 and targeting it for degradation. Within mTORC1, LST8 interacts directly with MTOR and enhances its kinase activity. In nutrient- poor conditions, stabilizes the MTOR-RPTOR interaction and favors RPTOR-mediated inhibition of MTOR activity. mTORC2 is also activated by growth factors, but seems to be nutrient-insensitive. mTORC2 seems to function upstream of Rho GTPases to regulate the actin cytoskeleton, probably by activating one or more Rho-type guanine nucleotide exchange factors. mTORC2 promotes the serum- induced formation of stress-fibers or F-actin. mTORC2 plays a critical role in AKT1 'Ser-473' phosphorylation, which may facilitate the phosphorylation of the activation loop of AKT1 on 'Thr-308' by PDK1 which is a prerequisite for full activation. mTORC2 regulates the phosphorylation of SGK1 at 'Ser-422'. mTORC2 also modulates the phosphorylation of PRKCA on 'Ser-657'.

MLST8 / GBL Antibody (aa150-200) - References

Mao Y.,et al.Submitted (JAN-2003) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Martin J.,et al.Nature 432:988-994(2004).

Mural R.J.,et al.Submitted (SEP-2005) to the EMBL/GenBank/DDBJ databases.
Ramachandiran S.,et al.Submitted (OCT-1999) to the EMBL/GenBank/DDBJ databases.