

GBL Antibody (ascites)
Mouse Monoclonal Antibody (Mab)
Catalog # AM1904A**Specification**

GBL Antibody (ascites) - Product Information

Application	WB,E
Primary Accession	O9BVC4
Other Accession	NP_071767.3
Reactivity	Mouse
Host	Mouse
Clonality	Monoclonal
Isotype	IgG1,k
Calculated MW	35876

GBL Antibody (ascites) - 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

This GBL monoclonal antibody is generated from mouse immunized with GBL recombinant protein.

Dilution

WB~~1:100~8000

Format

Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.

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

GBL Antibody (ascites) is for research use only and not for use in diagnostic or therapeutic procedures.

GBL Antibody (ascites) - 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

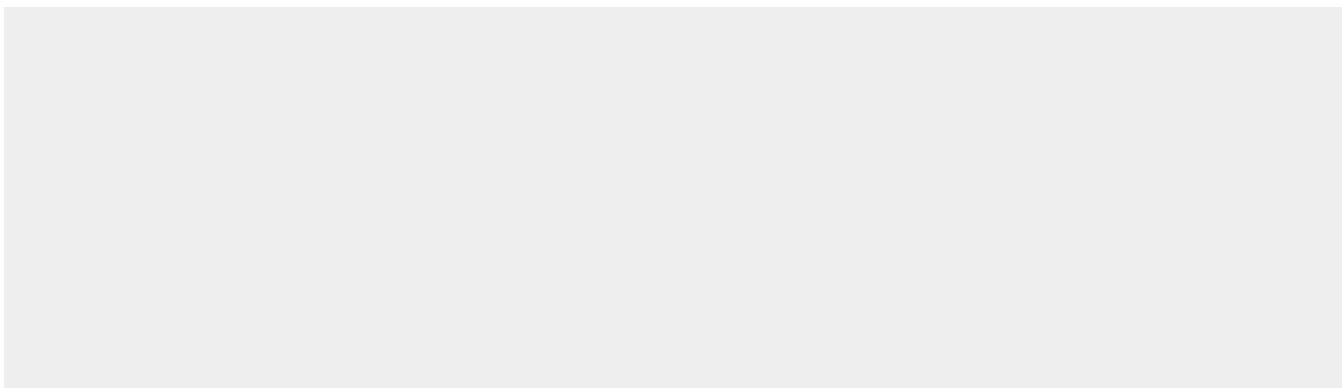
Broadly expressed, with highest levels in skeletal muscle, heart and kidney.

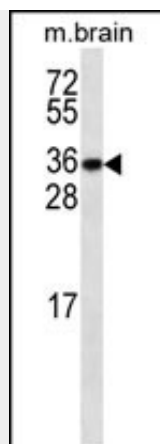
GBL Antibody (ascites) - Protocols

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](#)

GBL Antibody (ascites) - Images





GBL (Cat. #AM1904a) western blot analysis in mouse testis tissue lysates (35µg/lane). This demonstrates the GBL antibody detected the GBL protein (arrow).

GBL Antibody (ascites) - Background

Subunit of both mTORC1 and mTORC2, which regulate cell growth and survival in response to nutrient and hormonal signals. mTORC1 is activated in response to growth factors or amino-acids. Amino-acid-signaling to mTORC1 is mediated by Rag GTPases, which cause amino-acid-induced relocalization of mTOR within the endomembrane system. Growth factor-stimulated mTORC1 activation involves AKT1-mediated phosphorylation of TSC1-TSC2, which leads to the activation of the RHEB GTPase that potently activates the protein kinase activity of mTORC1. 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 FRAP1 and enhances its kinase activity. In nutrient-poor conditions, stabilizes the FRAP1-RPTOR interaction and favors RPTOR-mediated inhibition of FRAP1 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'.

GBL Antibody (ascites) - References

- Ali, S.M., et al. J. Biol. Chem. 280(20):19445-19448(2005)
- Inoki, K., et al. Microbiol. Mol. Biol. Rev. 69(1):79-100(2005)
- Sarbassov, D.D., et al. Science 307(5712):1098-1101(2005)
- Jacinto, E., et al. Nat. Cell Biol. 6(11):1122-1128(2004)
- Kim, D.H., et al. Mol. Cell 11(4):895-904(2003)