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GBL Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP19234b

Specification

GBL Antibody (C-term) Blocking Peptide - Product Information

Primary Accession Q9BVC4

GBL Antibody (C-term) Blocking Peptide - 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

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.

GBL Antibody (C-term) Blocking Peptide - 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, PubMed:28489822). 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, MLST8 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). As part of the mTORC2 complex, transduces signals from growth factors to pathways involved in proliferation, cytoskeletal organization, lipogenesis and anabolic output (PubMed:15467718, PubMed:35926713). mTORC2 is also activated by growth factors, but seems to be nutrient-insensitive (PubMed:15467718, PubMed:35926713). In response to growth factors, mTORC2 phosphorylates and activates AGC protein kinase family members, including AKT (AKT1, AKT2 and AKT3), PKC (PRKCA, PRKCB and PRKCE) and SGK1 (PubMed:15467718, PubMed:35926713). mTORC2 functions 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 activation by mediating phosphorylation of different sites depending on the context, such as 'Thr-450', 'Ser-473', 'Ser-477' or 'Thr-479', facilitating the phosphorylation of the activation loop of AKT1 on 'Thr-308' by PDPK1/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). Within mTORC2, MLST8 acts as a bridge between MAPKAP1/SIN1 and MTOR (PubMed:31085701).

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 (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

GBL Antibody (C-term) Blocking Peptide - Images

GBL Antibody (C-term) Blocking Peptide - 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 (C-term) Blocking Peptide - 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)