

ITM2B Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP13163b**Specification**

ITM2B Antibody (C-term) - Product Information

Application	WB, IHC-P,E
Primary Accession	O9Y287
Other Accession	Q60HC1 , NP_068839.1
Reactivity	Mouse
Predicted	Monkey
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	30338
Antigen Region	218-247

ITM2B Antibody (C-term) - Additional Information**Gene ID** 9445**Other Names**

Integral membrane protein 2B, Immature BRI2, imBRI2, Protein E25B, Transmembrane protein BRI, Bri, BRI2, membrane form, Mature BRI2, mBRI2, BRI2 intracellular domain, BRI2 ICD, BRI2C, soluble form, Bri23 peptide, Bri2-23, ABri23, C-terminal peptide, P23 peptide, ITM2B, BRI, BRI2

Target/Specificity

This ITM2B antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 218-247 amino acids from the C-terminal region of human ITM2B.

Dilution

WB~~1:1000

IHC-P~~1:10~50

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

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

ITM2B Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

ITM2B Antibody (C-term) - Protein Information

Name ITM2B

Synonyms BRI, BRI2

Function Plays a regulatory role in the processing of the amyloid-beta A4 precursor protein (APP) and acts as an inhibitor of the amyloid-beta peptide aggregation and fibrils deposition. Plays a role in the induction of neurite outgrowth. Functions as a protease inhibitor by blocking access of secretases to APP cleavage sites. Bri23 peptide prevents aggregation of APP amyloid-beta protein 42 into toxic oligomers.

Cellular Location

[Integral membrane protein 2B]: Golgi apparatus membrane; Single-pass type II membrane protein Note=Immature BRI2 (imBRI2) is cleaved by furin in the Golgi into mBRI2 and a Bri23 peptide. mBRI2 is transported to the plasma membrane and Bri23 peptide is secreted [Bri23 peptide]: Secreted. Note=Detected in the cerebral spinal fluid (CSF).

Tissue Location

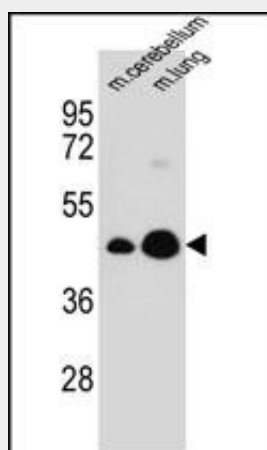
Ubiquitous. Expressed in brain.

ITM2B Antibody (C-term) - Protocols

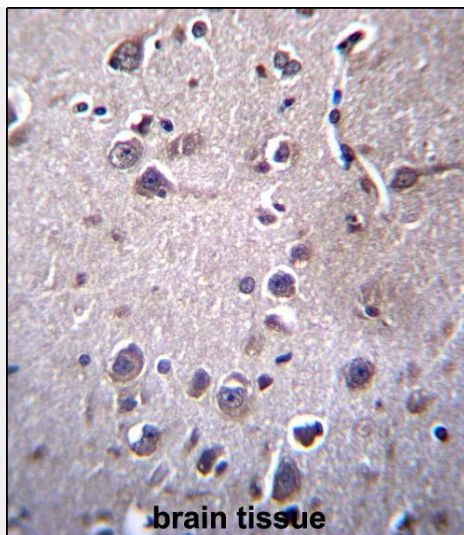
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

ITM2B Antibody (C-term) - Images



ITM2B Antibody (C-term) (Cat. #AP13163b) western blot analysis in mouse cerebellum, lung tissue lysates (35ug/lane). This demonstrates the ITM2B antibody detected the ITM2B protein (arrow).



ITM2B Antibody (C-term) (Cat. #AP13163b) immunohistochemistry analysis in formalin fixed and paraffin embedded human brain tissue followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of ITM2B Antibody (C-term) for immunohistochemistry. Clinical relevance has not been evaluated.

ITM2B Antibody (C-term) - Background

Amyloid precursor proteins are processed by beta-secretase and gamma-secretase to produce beta-amyloid peptides which form the characteristic plaques of Alzheimer disease. This gene encodes a transmembrane protein which is processed at the C-terminus by furin or furin-like proteases to produce a small secreted peptide which inhibits the deposition of beta-amyloid. Mutations which result in extension of the C-terminal end of the encoded protein, thereby increasing the size of the secreted peptide, are associated with two neurodegenerative diseases, familial British dementia and familial Danish dementia.

ITM2B Antibody (C-term) - References

Peng, S., et al. Biochem. Biophys. Res. Commun. 393(3):356-361(2010)
Matsuda, S., et al. J. Biol. Chem. 284(23):15815-15825(2009)
Matsuda, S., et al. Mol Neurodegener 4, 41 (2009) :
Tsachaki, M., et al. Biotechnol J 3(12):1548-1554(2008)
Kim, J., et al. J. Neurosci. 28(23):6030-6036(2008)