

GABARAPL2 Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP1822d

Product Information

Application	IHC-P, FC, WB, E
Primary Accession	<u>P60520</u>
Reactivity	Human, Rat, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB22734
Calculated MW	13667

Additional Information

Gene ID	11345
Other Names	Gamma-aminobutyric acid receptor-associated protein-like 2, GABA(A) receptor-associated protein-like 2, Ganglioside expression factor 2, GEF-2, General protein transport factor p16, Golgi-associated ATPase enhancer of 16 kDa, GATE-16, MAP1 light chain 3-related protein, GABARAPL2, FLC3A, GEF2
Target/Specificity	This GABARAPL2 antibody is generated from rabbits immunized with human GABARAPL2 recombinant protein.
Dilution	IHC-P~~1:100~500 FC~~1:10~50 WB~~1:1000 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	GABARAPL2 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	GABARAPL2 (<u>HGNC:13291</u>)
Synonyms	FLC3A, GEF2
Function	Ubiquitin-like modifier involved in intra-Golgi traffic (By similarity).

	Modulates intra-Golgi transport through coupling between NSF activity and SNAREs activation (By similarity). It first stimulates the ATPase activity of NSF which in turn stimulates the association with GOSR1 (By similarity). Involved in autophagy (PubMed: <u>20418806</u> , PubMed: <u>23209295</u>). Plays a role in mitophagy which contributes to regulate mitochondrial quantity and quality by eliminating the mitochondria to a basal level to fulfill cellular energy requirements and preventing excess ROS production (PubMed: <u>20418806</u> , PubMed: <u>23209295</u>). Whereas LC3s are involved in elongation of the phagophore membrane, the GABARAP/GATE-16 subfamily is essential for a later stage in autophagosome maturation (PubMed: <u>20418806</u> , PubMed: <u>23209295</u>).
Cellular Location	Cytoplasmic vesicle, autophagosome. Endoplasmic reticulum membrane. Golgi apparatus {ECO:0000250 UniProtKB:P60519}
Tissue Location	Ubiquitous. Expressed at high levels in the brain, heart, prostate, ovary, spleen and skeletal muscle. Expressed at very low levels in lung, thymus and small intestine

Background

Membrane proteins located on vesicles (v-SNAREs) and on the target membrane (t-SNAREs) mediate specific recognition and, possibly, fusion between a transport vesicle and its target membrane. The activity of SNARE molecules is regulated by several soluble cytosolic proteins. We have cloned a bovine brain cDNA encoding a conserved 117 amino acid polypeptide, denoted Golgi-associated ATPase Enhancer of 16 kDa (GATE-16), that functions as a soluble transport factor. GATE-16 interacts with N-ethylmaleimidesensitive factor (NSF) and significantly stimulates its ATPase activity. It also interacts with the Golgi v-SNARE GOS-28 in an NSF-dependent manner. We propose that GATE-16 modulates intra-Golgi transport through coupling between NSF activity and SNAREs activation.

References

Sou,Y.S., J. Biol. Chem. 281 (6), 3017-3024 (2006) Mehrle,A., Nucleic Acids Res. 34 (DATABASE ISSUE), D415-D418 (2006) Wiemann,S., Genome Res. 14 (10B), 2136-2144 (2004) Sagiv,Y., EMBO J. 19 (7), 1494-1504 (2000)

Images



Anti-GABARAPL2 Antibody at 1:500 dilution + A20 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 14 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

Citations

[•] Lentiviral-Mediated shRNA Approaches: Applications in Cellular Differentiation and Autophagy.

- Haploinsufficiency networks identify targetable patterns of allelic deficiency in low mutation ovarian cancer.
 Induction of autophagy is a key component of all-trans-retinoic acid-induced differentiation in leukemia cells and a potential target for pharmacologic modulation.

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