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SLC25A4 Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP18826b

Specification

SLC25A4 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P12235

SLC25A4 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 291

Other Names
ADP/ATP translocase 1, ADP, ATP carrier protein 1, ADP, ATP carrier protein, heart/skeletal muscle isoform T1, Adenine nucleotide translocator 1, ANT 1, Solute carrier family 25 member 4, SLC25A4, ANT1

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.

SLC25A4 Antibody (C-term) Blocking Peptide - Protein Information

Name SLC25A4 {ECO:0000303|PubMed:25732997, ECO:0000312|HGNC:HGNC:10990}

Function
ADP:ATP antiporter that mediates import of ADP into the mitochondrial matrix for ATP synthesis, and export of ATP out to fuel the cell (PubMed:21586654, PubMed:27693233). Cycles between the cytoplasmic-open state (c-state) and the matrix-open state (m-state): operates by the alternating access mechanism with a single substrate- binding site intermittently exposed to either the cytosolic (c-state) or matrix (m-state) side of the inner mitochondrial membrane (By similarity). In addition to its ADP:ATP antiporter activity, also involved in mitochondrial uncoupling and mitochondrial permeability transition pore (mPTP) activity (PubMed:31883789). Plays a role in mitochondrial uncoupling by acting as a proton transporter: proton transport uncouples the proton flows via the electron transport chain and ATP synthase to reduce the efficiency of ATP production and cause mitochondrial thermogenesis (By similarity). Proton transporter activity is inhibited by ADP:ATP antiporter activity, suggesting that SLC25A4/ANT1 acts as a master regulator of mitochondrial energy output by maintaining a delicate balance between ATP production (ADP:ATP antiporter activity) and thermogenesis (proton transporter activity) (By similarity).

Proton transporter activity requires free fatty acids as cofactor, but does not transport it (By similarity). Also plays a key role in mPTP opening, a non-specific pore that enables free passage of the mitochondrial membranes to solutes of up to 1.5 kDa, and which contributes to cell death (PubMed:31883789). It is however unclear if SLC25A4/ANT1 constitutes a pore-forming component of mPTP or regulates it (By similarity). Acts as a regulator of mitophagy independently of ADP:ATP antiporter activity: promotes mitophagy via interaction with TIMM44, leading to inhibit the presequence translocase TIMM23, thereby promoting stabilization of PINK1 (By similarity).

Cellular Location
Mitochondrion inner membrane; Multi-pass membrane protein. Membrane; Multi-pass membrane protein. Note=The complex formed with ARL2BP, ARL2 and SLC25A4/ANT1 is expressed in mitochondria (By similarity). May localize to non-mitochondrial membranes (PubMed:27641616) {ECO:0000250|UniProtKB:P48962, ECO:0000269|PubMed:27641616}

Tissue Location
Expressed in erythrocytes (at protein level).

SLC25A4 Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

SLC25A4 Antibody (C-term) Blocking Peptide - Images

SLC25A4 Antibody (C-term) Blocking Peptide - Background

This gene is a member of the mitochondrial carriersubfamily of solute carrier protein genes. The product of this genefunctions as a gated pore that translocates ADP from themitochondrial matrix into the cytoplasm. The protein forms ahomodimer embedded in the inner mitochondria membrane. Mutations inthis gene have been shown to result in autosomal dominantprogressive external opthalmoplegia and familial hypertrophiccardiomyopathy.

SLC25A4 Antibody (C-term) Blocking Peptide - References

Valenti, D., et al. Biochem. J. 431(2):299-310(2010)Wang, W., et al. Nucleic Acids Res. (2010) In press :Forlani, G., et al. Hum. Mol. Genet. 19(16):3114-3123(2010)Lena, A., et al. FEBS J. 277(13):2853-2867(2010)Kruger, J., et al. Mol Neurodegener 5, 8 (2010) :