

ATP2A2 Antibody (Center) Blocking peptide Synthetic peptide Catalog # BP12148c

## Specification

# ATP2A2 Antibody (Center) Blocking peptide - Product Information

Primary Accession

<u>P16615</u>

## ATP2A2 Antibody (Center) Blocking peptide - Additional Information

Gene ID 488

**Other Names** 

Sarcoplasmic/endoplasmic reticulum calcium ATPase 2, SERCA2, SR Ca(2+)-ATPase 2, Calcium pump 2, Calcium-transporting ATPase sarcoplasmic reticulum type, slow twitch skeletal muscle isoform, Endoplasmic reticulum class 1/2 Ca(2+) ATPase, ATP2A2, ATP2B

#### 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.

# ATP2A2 Antibody (Center) Blocking peptide - Protein Information

Name ATP2A2 (HGNC:812)

## Synonyms ATP2B

#### Function

This magnesium-dependent enzyme catalyzes the hydrolysis of ATP coupled with the translocation of calcium from the cytosol to the sarcoplasmic reticulum lumen (PubMed:<a href="http://www.uniprot.org/citations/16402920" target="\_blank">16402920</a>, PubMed:<a href="http://www.uniprot.org/citations/12542527" target="\_blank">12542527</a>). Involved in autophagy in response to starvation. Upon interaction with VMP1 and activation, controls ER-isolation membrane contacts for autophagosome formation (PubMed:<a href="http://www.uniprot.org/citations/28890335" target="\_blank">28890335</a>). Also modulates ER contacts with lipid droplets, mitochondria and endosomes (PubMed:<a href="http://www.uniprot.org/citations/28890335" target="\_blank">28890335</a>). In coordination with FLVCR2 mediates heme-stimulated switching from mitochondrial ATP synthesis to thermogenesis (By similarity).

#### **Cellular Location**

Endoplasmic reticulum membrane {ECO:0000250|UniProtKB:055143}; Multi-pass membrane



protein. Sarcoplasmic reticulum membrane; Multi-pass membrane protein. Note=Colocalizes with FLVCR2 at the mitochondrial-ER contact junction. {ECO:0000250|UniProtKB:055143}

### **Tissue Location**

Isoform 1 is widely expressed in smooth muscle and nonmuscle tissues such as in adult skin epidermis, with highest expression in liver, pancreas and lung, and intermediate expression in brain, kidney and placenta. Also expressed at lower levels in heart and skeletal muscle. Isoforms 2 and 3 are highly expressed in the heart and slow twitch skeletal muscle. Expression of isoform 3 is predominantly restricted to cardiomyocytes and in close proximity to the sarcolemma Both isoforms are mildly expressed in lung, kidney, liver, pancreas and placenta. Expression of isoform 3 is amplified during monocytic differentiation and also observed in the fetal heart

## ATP2A2 Antibody (Center) Blocking peptide - Protocols

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

### <u>Blocking Peptides</u>

## ATP2A2 Antibody (Center) Blocking peptide - Images

## ATP2A2 Antibody (Center) Blocking peptide - Background

This gene encodes one of the SERCA Ca(2+)-ATPases, whichare intracellular pumps located in the sarcoplasmic or endoplasmicreticula of muscle cells. This enzyme catalyzes the hydrolysis of ATP coupled with the translocation of calcium from the cytosol into the sarcoplasmic reticulum lumen, and is involved in regulation of the contraction/relaxation cycle. Mutations in this gene causeDarier-White disease, also known as keratosis follicularis, anautosomal dominant skin disorder characterized by loss of adhesionbetween epidermal cells and abnormal keratinization. Alternativesplicing results in multiple transcript variants encoding differentisoforms.

## ATP2A2 Antibody (Center) Blocking peptide - References

Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)Tuusa, J.T., et al. FEBS J. 277(13):2815-2829(2010)Godic, A., et al. Eur J Dermatol 20(3):271-275(2010)Godic, A., et al. J. Am. Acad. Dermatol. 62(5):819-823(2010)Kiec-Wilk, B., et al. Prz. Lek. 67(3):151-156(2010)