

SEPN1 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP12452B**Specification**

SEPN1 Antibody (C-term) - Product Information

Application	WB, IHC-P,E
Primary Accession	O9NZV5
Other Accession	NP_996809.1 , NP_065184.2
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	65813
Antigen Region	417-445

SEPN1 Antibody (C-term) - Additional Information**Gene ID** 57190**Other Names**

Selenoprotein N, SelN, SEPN1, SELN

Target/Specificity

This SEPN1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 417-445 amino acids from the C-terminal region of human SEPN1.

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

SEPN1 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

SEPN1 Antibody (C-term) - Protein Information**Name** SELENON {ECO:0000303|PubMed:27645994, ECO:0000312|HGNC:HGNC:15999}

Function [Isoform 2]: Plays an important role in cell protection against oxidative stress and in the regulation of redox-related calcium homeostasis. Regulates the calcium level of the ER by protecting the calcium pump ATP2A2 against the oxidoreductase ERO1A-mediated oxidative damage. Within the ER, ERO1A activity increases the concentration of H₂O₂, which attacks the luminal thiols in ATP2A2 and thus leads to cysteinyl sulfenic acid formation (-SOH) and SEPN1 reduces the SOH back to free thiol (-SH), thus restoring ATP2A2 activity (PubMed:[25452428](#)). Acts as a modulator of ryanodine receptor (RyR) activity: protects RyR from oxidation due to increased oxidative stress, or directly controls the RyR redox state, regulating the RyR-mediated calcium mobilization required for normal muscle development and differentiation (PubMed:[18713863](#), PubMed:[19557870](#)).

Cellular Location

[Isoform 2]: Endoplasmic reticulum membrane

Tissue Location

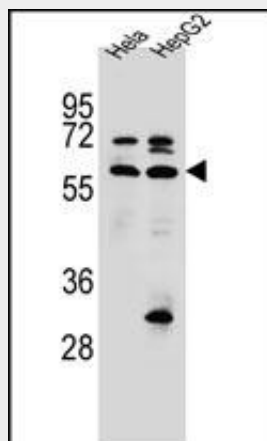
Isoform 1 and isoform 2 are expressed in skeletal muscle, brain, lung and placenta. Isoform 2 is also expressed in heart, diaphragm and stomach.

SEPN1 Antibody (C-term) - Protocols

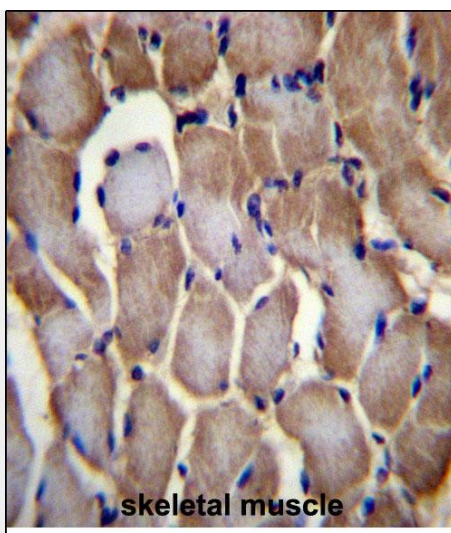
Provided below are standard protocols that you may find useful for product applications.

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

SEPN1 Antibody (C-term) - Images



SEPN1 Antibody (C-term) (Cat. #AP12452b) western blot analysis in HeLa, HepG2 cell line lysates (35ug/lane). This demonstrates the SEPN1 antibody detected the SEPN1 protein (arrow).



SEPN1 Antibody (C-term) (Cat. #AP12452b) immunohistochemistry analysis in formalin fixed and paraffin embedded human skeletal muscle followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of SEPN1 Antibody (C-term) for immunohistochemistry. Clinical relevance has not been evaluated.

SEPN1 Antibody (C-term) - Background

This gene encodes a selenoprotein, which contains a selenocysteine (Sec) residue at its active site. The selenocysteine is encoded by the UGA codon that normally signals translation termination. The 3' UTR of selenoprotein genes have a common stem-loop structure, the sec insertion sequence (SECIS), that is necessary for the recognition of UGA as a Sec codon rather than as a stop signal. Mutations in this gene cause the classical phenotype of multimimicore disease and congenital muscular dystrophy with spinal rigidity and restrictive respiratory syndrome. Two alternatively spliced transcript variants encoding distinct isoforms have been found for this gene.

SEPN1 Antibody (C-term) - References

Arbogast, S., et al. Ann. Neurol. 65(6):677-686(2009)
Maiti, B., et al. Hum. Mutat. 30(3):411-416(2009)
Jurynek, M.J., et al. Proc. Natl. Acad. Sci. U.S.A. 105(34):12485-12490(2008)
Lin, L., et al. PLoS Genet. 4 (10), E1000225 (2008) :
Wu, C., et al. Proteomics 7(11):1775-1785(2007)