

**STOML2 Blocking Peptide (Center)**

Synthetic peptide

Catalog # BP20280c

**Specification**

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**STOML2 Blocking Peptide (Center) - Product Information**

Primary Accession

[O9UJZ1](#)

Other Accession

[O4FZT0](#), [O99JB2](#), [O32LL2](#), [NP\\_038470.1](#)**STOML2 Blocking Peptide (Center) - Additional Information****Gene ID** 30968**Other Names**

Stomatin-like protein 2, mitochondrial, SLP-2, EPB72-like protein 2, Paraprotein target 7, Paratarg-7, STOML2, SLP2

**Target/Specificity**

The synthetic peptide sequence is selected from aa 176-189 of HUMAN STOML2

**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.

**STOML2 Blocking Peptide (Center) - Protein Information****Name** STOML2**Synonyms** SLP2**Function**

Mitochondrial protein that probably regulates the biogenesis and the activity of mitochondria. Stimulates cardiolipin biosynthesis, binds cardiolipin-enriched membranes where it recruits and stabilizes some proteins including prohibitin and may therefore act in the organization of functional microdomains in mitochondrial membranes. Through regulation of the mitochondrial function may play a role into several biological processes including cell migration, cell proliferation, T-cell activation, calcium homeostasis and cellular response to stress. May play a role in calcium homeostasis through negative regulation of calcium efflux from mitochondria. Required for mitochondrial hyperfusion a pro-survival cellular response to stress which results in increased ATP production by mitochondria. May also regulate the organization of functional domains at the plasma membrane and play a role in T-cell activation through association with the T- cell receptor signaling complex and its regulation.

**Cellular Location**

Cell membrane; Peripheral membrane protein. Mitochondrion. Mitochondrion inner membrane; Lipid-anchor. Mitochondrion intermembrane space. Membrane raft. Cytoplasm, cytoskeleton  
Note=Behaves as an integral membrane protein of the mitochondrion despite the absence of a detectable transmembrane domain (PubMed:21746876). Also associates with the actin cytoskeleton and membrane rafts in activated T-cells (PubMed:18641330, PubMed:10713127) A minor pool is associated with the plasma membrane and is enriched at the immunological synapse in activated T-cells (PubMed:22623988)

**Tissue Location**

Ubiquitously expressed at low levels. Expressed in lymphoid tissues (at protein level).

**STOML2 Blocking Peptide (Center) - Protocols**

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

- [Blocking Peptides](#)

**STOML2 Blocking Peptide (Center) - Images****STOML2 Blocking Peptide (Center) - Background**

STOML2 is similar in sequence to stomatin. It is a 356 amino acid protein with a calculated molecular mass of 38.5 kDa. STOML2 has 3 potential initiator sites, all sharing the same open reading frame. The STOML2 protein contains the cognate stomatin family consensus sequence, but it lacks the characteristic N-terminal hydrophobic domain and palmitoylation consensus sequence. STOML2 shares greatest sequence homology with stomatin and SLP1 in a region predicted to contain beta sheet and alpha helix structures. Northern blot analysis detected a 1.5 kb STOML2 transcript in all tissues examined, with highest levels in heart, liver, and pancreas. Western blot analysis detected STOML2 at apparent molecular masses of 45.5 kDa or 44.6 kDa in all human and mammalian cell lines and tissues examined, including red blood cells. Some cells also showed a faint band at about 34.3 kDa, which may represent translation from an alternate initiation site.

**STOML2 Blocking Peptide (Center) - References**

Chang, D., et al. Biomarkers 15(2):104-110(2010)  
Da Cruz, S., et al. Cell Calcium 47(1):11-18(2010)  
Grass, S., et al. Lancet Oncol. 10(10):950-956(2009)  
Kirchhof, M.G., et al. J. Immunol. 181(3):1927-1936(2008)  
Green, J.B., et al. BMC Evol. Biol. 8, 44 (2008) :