

### Bok BH3 Domain Antibody Blocking Peptide Synthetic peptide

Catalog # BP1310a

## Specification

# **Bok BH3 Domain Antibody Blocking Peptide - Product Information**

Primary Accession

<u>Q9UMX3</u>

## **Bok BH3 Domain Antibody Blocking Peptide - Additional Information**

Gene ID 666

**Other Names** Bcl-2-related ovarian killer protein, hBOK, Bcl-2-like protein 9, Bcl2-L-9, BOK, BCL2L9

Target/Specificity

The synthetic peptide sequence used to generate the antibody <a href=/product/products/AP1310a>AP1310a</a> was selected from the region of human Bok BH3 Domain. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

## **Bok BH3 Domain Antibody Blocking Peptide - Protein Information**

Name BOK (<u>HGNC:1087</u>)

Synonyms BCL2L9

Function

[Isoform 1]: Apoptosis regulator that functions through different apoptotic signaling pathways (PubMed:<a href="http://www.uniprot.org/citations/15102863" target="\_blank">15102863</a>, PubMed:<a href="http://www.uniprot.org/citations/20673843" target="\_blank">20673843</a>, PubMed:<a href="http://www.uniprot.org/citations/27076518" target="\_blank">27076518</a>). Plays a roles as pro-apoptotic protein that positively regulates intrinsic apoptotic process in a BAXand BAK1-dependent manner or in a BAX- and BAK1-independent manner (PubMed:<a href="http://www.uniprot.org/citations/15102863" target="\_blank">15102863</a>, PubMed:<a href="http://www.uniprot.org/citations/15102863" target="\_blank">27076518</a>). In response to endoplasmic reticulum stress promotes mitochondrial apoptosis through downstream BAX/BAK1 activation and positive regulation of PERK-mediated unfolded protein response (By similarity).



Activates apoptosis independently of heterodimerization with survival-promoting BCL2 and BCL2L1 through induction of mitochondrial outer membrane permeabilization, in a BAX- and BAK1-independent manner, in response to inhibition of ERAD- proteasome degradation system, resulting in cytochrome c release (PubMed:<a href="http://www.uniprot.org/citations/27076518" target="\_blank">27076518</a>). In response to DNA damage, mediates intrinsic apoptotic process in a TP53-dependent manner (PubMed:<a

href="http://www.uniprot.org/citations/15102863" target="\_blank">15102863</a>). Plays a role in granulosa cell apoptosis by CASP3 activation (PubMed:<a

href="http://www.uniprot.org/citations/20673843" target="\_blank">20673843</a>). Plays a roles as anti-apoptotic protein during neuronal apoptotic process, by negatively regulating poly ADP-ribose polymerase-dependent cell death through regulation of neuronal calcium homeostasis and mitochondrial bioenergetics in response to NMDA excitation (By similarity). In addition to its role in apoptosis, may regulate trophoblast cell proliferation during the early stages of placental development, by acting on G1/S transition through regulation of CCNE1 expression (PubMed:<a href="http://www.uniprot.org/citations/19942931" target="\_blank">19942931</a>). May also play a role as an inducer of autophagy by disrupting interaction between MCL1 and BECN1 (PubMed:<a href="http://www.uniprot.org/citations/24113155" target="\_blank">24113155</a>).

### **Cellular Location**

[Isoform 1]: Mitochondrion membrane {ECO:0000250|UniProtKB:O35425}; Single-pass membrane protein {ECO:0000250|UniProtKB:O35425}. Endoplasmic reticulum membrane; Single-pass membrane protein {ECO:0000250|UniProtKB:O35425}. Mitochondrion inner membrane. Cytoplasm. Nucleus. Mitochondrion. Endoplasmic reticulum. Mitochondrion outer membrane. Early endosome membrane {ECO:0000250|UniProtKB:O35425}. Recycling endosome membrane {ECO:0000250|UniProtKB:O35425}. Nucleus outer membrane {ECO:0000250|UniProtKB:O35425}. Golgi apparatus, cis-Golgi network membrane {ECO:0000250|UniProtKB:035425}. Golgi apparatus, trans-Golgi network membrane {ECO:0000250|UniProtKB:035425}. Membrane. Note=Nuclear and cytoplasmic compartments in the early stages of apoptosis and during apoptosis it associates with mitochondria (PubMed:19942931). In healthy cells, associates loosely with the membrane in a hit-and-run mode. The insertion and accumulation on membranes is enhanced through the activity of death signals, resulting in the integration of the membrane-bound protein into the membrane (PubMed:15868100). The transmembrane domain controls subcellular localization; constitutes a tail-anchor. Localizes in early and late endosome upon blocking of apoptosis. Must localize to the mitochondria to induce mitochondrial outer membrane permeabilization and apoptosis (By similarity) {ECO:0000250|UniProtKB:O35425, ECO:0000269|PubMed:15868100, ECO:0000269|PubMed:19942931}

#### **Tissue Location**

Expressed mainly in oocytes; weak expression in granulosa cells of the developing follicles. In adult human ovaries, expressed in granulosa cells at all follicular stages, but expression in primordial/primary follicles granulosa cell is stronger than in secondary and antral follicles.

## **Bok BH3 Domain Antibody Blocking Peptide - Protocols**

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

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

## Bok BH3 Domain Antibody Blocking Peptide - Images

## Bok BH3 Domain Antibody Blocking Peptide - Background

Bok belongs to the BCL-2 protein family. BCL-2 family members form hetero- or homodimers and act as anti- and pro-apoptotic regulators that are involved in a wide variety of cellular activities. This protein contains all four BCL-2 like domains (BH1, 2, 3 and 4) and is a pro-apoptotic BCL-2 protein identified in the ovary.



# Bok BH3 Domain Antibody Blocking Peptide - References

Hsu, S.Y., et al., Proc. Natl. Acad. Sci. U.S.A. 94(23):12401-12406 (1997).Zhang, H., et al., FEBS Lett. 480 (2-3), 311-313 (2000).