

**ADRA1B Blocking Peptide (C-term)**  
**Synthetic peptide**  
**Catalog # BP19930b**

### Specification

#### ADRA1B Blocking Peptide (C-term) - Product Information

Primary Accession  
Other Accession

[P35368](#)  
[P15823](#), [P97717](#), [NP\\_000670.1](#)

#### ADRA1B Blocking Peptide (C-term) - Additional Information

##### Gene ID 147

##### Other Names

Alpha-1B adrenergic receptor, Alpha-1B adrenoreceptor, Alpha-1B adrenoceptor, ADRA1B

##### Target/Specificity

The synthetic peptide sequence is selected from aa 395-409 of HUMAN ADRA1B

##### 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.

#### ADRA1B Blocking Peptide (C-term) - Protein Information

##### Name ADRA1B ([HGNC:278](#))

##### Function

Alpha-1 adrenergic receptors are G protein-coupled receptors for catecholamines that signal through the G(q) family of G proteins, including G(q) and G(11). Upon activation, they stimulate the phosphatidylinositol-calcium second messenger pathway, leading to calcium release from intracellular stores and activation of protein kinase C (By similarity). ADRA1B binds the catecholamine ligands norepinephrine and epinephrine (PubMed:<a href="http://www.uniprot.org/citations/7815325" target="\_blank">7815325</a>, PubMed:<a href="http://www.uniprot.org/citations/8183249" target="\_blank">8183249</a>). Can also couple to G(14) and G(16) proteins (By similarity). Nuclear ADRA1B forms heterooligomers with ADRA1A to regulate phenylephrine(PE)- stimulated ERK signaling in cardiac myocytes (PubMed:<a href="http://www.uniprot.org/citations/18802028" target="\_blank">18802028</a>, PubMed:<a href="http://www.uniprot.org/citations/22120526" target="\_blank">22120526</a>). At the plasma membrane, ADRA1B interacts with CAVIN4/MURC to regulates ERK activation in cardiomyocytes, contributing to the regulation of cardiac hypertrophy (PubMed:<a href="http://www.uniprot.org/citations/24567387" target="\_blank">24567387</a>).

## Cellular Location

Nucleus membrane; Multi-pass membrane protein. Cell membrane; Multi-pass membrane protein. Cytoplasm. Membrane, caveola. Note=Location at the nuclear membrane facilitates heterooligomerization and regulates ERK-mediated signaling in cardiac myocytes. Colocalizes with GNAQ, PLCB1 as well as LAP2 at the nuclear membrane of cardiac myocytes (PubMed:18802028, PubMed:22120526). Colocalizes with CAVIN4 and CAV3 at the plasma membrane and partly within the cytoplasm in cardiomyocytes (PubMed:24567387).

## ADRA1B Blocking Peptide (C-term) - Protocols

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

- [Blocking Peptides](#)

## ADRA1B Blocking Peptide (C-term) - Images

## ADRA1B Blocking Peptide (C-term) - Background

Alpha-1-adrenergic receptors (alpha-1-ARs) are members of the G protein-coupled receptor superfamily. They activate mitogenic responses and regulate growth and proliferation of many cells. There are 3 alpha-1-AR subtypes: alpha-1A, -1B and -1D, all of which signal through the Gq/11 family of G-proteins and different subtypes show different patterns of activation. This gene encodes alpha-1B-adrenergic receptor, which induces neoplastic transformation when transfected into NIH 3T3 fibroblasts and other cell lines. Thus, this normal cellular gene is identified as a protooncogene. This gene comprises 2 exons and a single large intron of at least 20 kb that interrupts the coding region.

## ADRA1B Blocking Peptide (C-term) - References

- Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)  
Pinheiro, A.P., et al. Am. J. Med. Genet. B Neuropsychiatr. Genet. 153B (5), 1070-1080 (2010) :  
Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :  
Jensen, B.C., et al. Circ Heart Fail 2(6):654-663(2009)  
Talmud, P.J., et al. Am. J. Hum. Genet. 85(5):628-642(2009)