

**CRYAA Blocking Peptide(Center)**

Synthetic peptide

Catalog # BP19751c

**Specification**

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

Primary Accession

[P02489](#)

Other Accession

[P24623](#), [P02493](#), [P02475](#), [P24622](#), [P02470](#),  
[NP\\_000385.1](#), [P02478](#), [Q5ENZ0](#)**CRYAA Blocking Peptide(Center) - Additional Information****Gene ID** 102724652;1409**Other Names**Alpha-crystallin A chain, Heat shock protein beta-4, HspB4, Alpha-crystallin A(1-172),  
Alpha-crystallin A(1-168), Alpha-crystallin A(1-162), CRYAA, CRYA1, HSPB4**Target/Specificity**

The synthetic peptide sequence is selected from aa 92-106 of HUMAN CRYAA

**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.

**CRYAA Blocking Peptide(Center) - Protein Information****Name** CRYAA**Synonyms** CRYA1, HSPB4**Function**Contributes to the transparency and refractive index of the lens (PubMed: [18302245](http://www.uniprot.org/citations/18302245)). In its oxidized form (absence of intramolecular disulfide bond), acts as a chaperone, preventing aggregation of various proteins under a wide range of stress conditions (PubMed: [22120592](http://www.uniprot.org/citations/22120592), PubMed: [31792453](http://www.uniprot.org/citations/31792453), PubMed: [18199971](http://www.uniprot.org/citations/18199971), PubMed: [19595763](http://www.uniprot.org/citations/19595763)). Required for the correct formation of lens intermediate filaments as part of a complex composed of BFSP1, BFSP2 and CRYAA (PubMed: [28935373](http://www.uniprot.org/citations/28935373))

target="\_blank">28935373</a>).

#### **Cellular Location**

Cytoplasm. Nucleus. Note=Translocates to the nucleus during heat shock and resides in sub-nuclear structures known as SC35 speckles or nuclear splicing speckles

#### **Tissue Location**

Expressed in the eye lens (at protein level).

### **CRYAA Blocking Peptide(Center) - Protocols**

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

- [Blocking Peptides](#)

### **CRYAA Blocking Peptide(Center) - Images**

### **CRYAA Blocking Peptide(Center) - Background**

Crystallins are separated into two classes: taxon-specific, or enzyme, and ubiquitous. The latter class constitutes the major proteins of vertebrate eye lens and maintains the transparency and refractive index of the lens. Since lens central fiber cells lose their nuclei during development, these crystallins are made and then retained throughout life, making them extremely stable proteins. Mammalian lens crystallins are divided into alpha, beta, and gamma families; beta and gamma crystallins are also considered as a superfamily. Alpha and beta families are further divided into acidic and basic groups. Seven protein regions exist in crystallins: four homologous motifs, a connecting peptide, and N- and C-terminal extensions. Alpha crystallins are composed of two gene products: alpha-A and alpha-B, for acidic and basic, respectively. Alpha crystallins can be induced by heat shock and are members of the small heat shock protein (sHSP also known as the HSP20) family. They act as molecular chaperones although they do not renature proteins and release them in the fashion of a true chaperone; instead they hold them in large soluble aggregates. Post-translational modifications decrease the ability to chaperone. These heterogeneous aggregates consist of 30-40 subunits; the alpha-A and alpha-B subunits have a 3:1 ratio, respectively. Two additional functions of alpha crystallins are an autokinase activity and participation in the intracellular architecture. Alpha-A and alpha-B gene products are differentially expressed; alpha-A is preferentially restricted to the lens and alpha-B is expressed widely in many tissues and organs. Defects in this gene cause autosomal dominant congenital cataract (ADCC). [provided by RefSeq].

### **CRYAA Blocking Peptide(Center) - References**

Deng, M., et al. Biochim. Biophys. Acta 1802 (7-8), 621-631 (2010) :  
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