CRYBA1 Polyclonal Antibody

catalog number: E-AB-91092

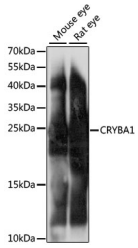
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Mouse;Rat
Immunogen	Recombinant fusion protein of human CRYBA1
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Buffer	Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

Applications	Recommended Dilution
WB	1:200-1:2000

Data



Western blot analysis of extracts of various cell lines using CRYBA1 Polyclonal Antibody at 1:1000 dilution.

Observed-MV:25 kDa

Calculated-MV:23 kDa/25 kDa

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended.

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. Beta-crystallins, the most heterogeneous, differ by the presence of the C-terminal extension (present in the basic group, none in the acidic group). Beta-crystallins form aggregates of different sizes and are able to self-associate to form dimers or to form heterodimers with other beta-crystallins. This gene, a beta acidic group member, encodes two proteins (crystallin, beta A3 and crystallin, beta A1) from a single mRNA, the latter protein is 17 aa shorter than crystallin, beta A3 and is generated by use of an alternate translation initiation site. Deletion of exons 3 and 4 causes the autosomal dominant disease 'zonular cataract with sutural opacities'.

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