

PKAca, Active recombinant protein

PKA, cAMP-dependent protein kinase catalytic subunit alpha Catalog # PBV11318r

Specification

PKAca, Active recombinant protein - Product info

Primary Accession	<u>P17612</u>
Concentration	0.1
Calculated MW	69.0 kDa KDa

PKAca, Active recombinant protein - Additional Info

Gene ID5566Gene SymbolPRKACAOther NamesPKA, cAMP-dependent protein kinase catalytic subunit alpha

Source Assay&Purity Assay2&Purity2 Recombinant Format Liquid Baculovirus (Sf9 insect cells) SDS-PAGE; ≥90% HPLC; Yes

Storage

-80°C; Recombinant proteins in storage buffer (50 mM Tris-HCl, pH 7.5, 150 mM NaCl, 0.25 mM DTT, 0.1 mM EGTA, 0.1 mM EDTA, 0.1 mM PMSF, 25% glycerol).

PKAca, Active recombinant protein - Protocols

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

- <u>Western Blot</u>
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- <u>Immunofluorescence</u>
- Immunoprecipitation
- Flow Cytomety
- <u>Cell Culture</u>

PKAca, Active recombinant protein - Images

PKAca, Active recombinant protein - Background

Most of the effects of cAMP are mediated through the phosphorylation of target proteins on serine or threonine residues by the cAMP-dependent protein kinase (AMPK). The inactive holoenzyme of AMPK is a tetramer composed of two regulatory and two catalytic subunits. The mammalian catalytic subunit has been shown to consist of three PKA gene products: $C-\alpha$, $C-\beta$, and $C-\gamma$. Two PKA



isoforms exist, designated types I and II, which differ in their dimeric regulatory subunits, designated RI and RII, respectively. Furthermore, there are at least four different regulatory subunits: RI- α , RI- β , RII- α , and RII- β . cAMP causes the dissociation of the inactive holoenzyme into a dimer of regulatory subunits bound to four cAMP and two free monomeric catalytic subunits. The catalytic subunit C- α of PKA (PKAca) is a member of the Ser/Thr protein kinase family and is a catalytic subunit C- β of AMPK. Tasken et al. assigned the PKAca gene to 19p13.1 (1). Yasuda et al found that protein kinase A is required for long-term potentiation in neonatal tissue and suggested that developmental changes in synapse morphology may underlie the changes in the kinase activity (2). Skalhegg et al generated a null mutation in the major catalytic subunit of PKAca, and observed early postnatal lethality in the majority of C- α knockout mice. Surprisingly, a small percentage of C- α knockout mice, although runted, survived to adulthood. In these animals, compensatory increases in C- β levels occurred in brain whereas many tissues, including skeletal muscle, heart, and sperm, contained less than 10% of the normal PKA activity (3).