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PKAca, Active recombinant protein

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

Specification

PKAca, Active recombinant protein - Product info

Primary Accession	P17612
Concentration	0.1
Calculated MW	69.0 kDa KDa

PKAca, Active recombinant protein - Additional Info

Gene ID	5566
Gene Symbol	PRKACA

Other Names
PKA, cAMP-dependent protein kinase catalytic subunit alpha

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

Format
Liquid

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.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

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-α, C-β, and C-γ. 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).