

Product name:	PKA Iα reg Rabbit Polyclonal Antibody
Cat number:	ABN16182
Conjugate:	Unconjugated
Size:	100μL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human KAP0. AA range:271-320
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:200-1:1000,ELISA 1:10000-1:20000
Molecular Weight:	43kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

cAMP is a signaling molecule important for a variety of cellular functions. cAMP exerts its effects by activating the cAMP-dependent protein kinase, which transduces the signal through phosphorylation of different target proteins. The inactive kinase holoenzyme is a tetramer composed of two regulatory and two catalytic subunits. cAMP causes the dissociation of the inactive holoenzyme into a dimer of regulatory subunits bound to four cAMP and two free monomeric catalytic subunits. Four different regulatory subunits and three catalytic subunits have been identified in humans. This gene encodes one of the regulatory subunits. This protein was found to be a tissue-specific extinguisher that down-regulates the expression of seven liver genes in hepatoma x fibroblast hybrids. Mutations in this gene cause Carney complex (CNC). This gene can fuse to the RET protooncogene. Defects in PRKAR1A are the cause of Carney complex type 1 (CNC1) [MIM:160980]. CNC is a multiple neoplasia syndrome characterized by spotty skin pigmentation, cardiac and other myxomas, endocrine tumors, and psammomatous melanotic schwannomas. Defects in PRKAR1A are the cause of intracardiac myxoma [MIM:255960]. Inheritance is autosomal recessive. Defects in PRKAR1A are the cause of primary pigmented nodular adrenocortical disease type 1 (PPNAD1) [MIM:610489]. Primary pigmented nodular adrenocortical disease is a rare bilateral adrenal defect causing ACTH-independent Cushing syndrome. Macroscopic appearance of the adrenals is characteristic with small pigmented micronodules observed in the cortex. PPNAD1 is most often diagnosed in patients with Carney complex, but it can also be observed in patients without other manifestations or familial history. PTM: The pseudophosphorylation site binds to the substrate-binding region of the catalytic chain, resulting in the inhibition of its activity. Similarity: Belongs to the cAMP-dependent kinase regulatory chain family. Similarity: Contains 2 cyclic nucleotide-binding domains. Subunit: The inactive form of the enzyme is composed of two regulatory chains and two catalytic chains. Activation by cAMP produces two active catalytic monomers and a regulatory dimer that binds four cAMP molecules. PRKAR1A also interacts with RFC2; the complex may be involved in cell survival. Interacts with AKAP4. Tissue specificity: Four types of regulatory chains are found: I-α, I-β, II-α, and II-β. Their expression varies among tissues and is in some cases constitutive and in others inducible.