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Protein Kinase A regulatory subunit I alpha Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8095a

Product Information

Application	IHC-P, WB, E
Primary Accession	P10644
Reactivity	Human
Predicted	Rat, Canine, Rabbit
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB3247
Calculated MW	42982
Antigen Region	1-30

Additional Information

Gene ID	5573
Other Names	cAMP-dependent protein kinase type I-alpha regulatory subunit, Tissue-specific extinguisher 1, TSE1, cAMP-dependent protein kinase type I-alpha regulatory subunit, N-terminally processed, PRKAR1A, PKR1, PRKAR1, TSE1
Target/Specificity	This Protein Kinase A regulatory subunit I alpha antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1-30 amino acids from the N-terminal region of human Protein Kinase A regulatory subunit I alpha.
Dilution	IHC-P~~1:100~500 WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Protein Kinase A regulatory subunit I alpha Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PRKAR1A
Synonyms	PKR1, PRKAR1, TSE1
Function	Regulatory subunit of the cAMP-dependent protein kinases involved in cAMP signaling in cells.
Cellular Location	Cell membrane
Tissue Location	Four types of regulatory chains are found: I-alpha, I-beta, II-alpha, and II-beta. Their expression varies among tissues and is in some cases constitutive and in others inducible

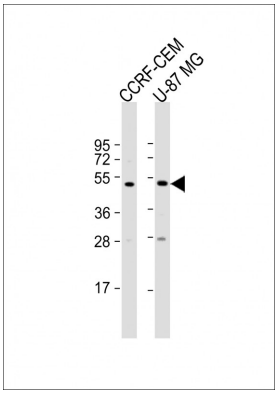
Background

cAMP is a signaling molecule important for a variety of cellular functions. cAMP exerts its effects by activating the cAMP-dependent protein kinase (AMPK), which transduces the signal through phosphorylation of different target proteins. The inactive holoenzyme of AMPK 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 of AMPK have been identified in humans. PKR1 is 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. Functional null mutations in the gene cause Carney complex (CNC), an autosomal dominant multiple neoplasia syndrome. The gene can fuse to the RET protooncogene by gene rearrangement and form the thyroid tumor-specific chimeric oncogene known as PTC2.

References

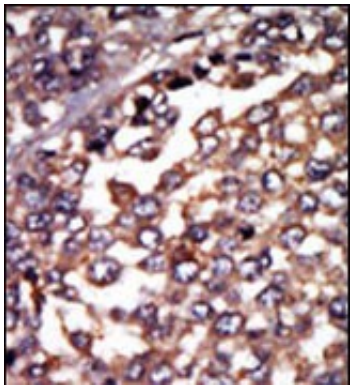
Gronholm, M., et al., J. Biol. Chem. 278(42):41167-41172 (2003).
Bertherat, J., et al., Cancer Res. 63(17):5308-5319 (2003).
Stergiopoulos, S.G., et al., FEBS Lett. 546(1):59-64 (2003).
Robinson-White, A., et al., Hum. Mol. Genet. 12(13):1475-1484 (2003).
Holm, A.M., et al., J. Immunol. 170(11):5772-5777 (2003).

Images



All lanes : Anti-PKR1 Antibody (M1) at 1:1000 dilution Lane 1: CCRF-CEM whole cell lysate Lane 2: U-87 MG whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 43 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma;



HC = hepatocarcinoma.

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