

Product Name: PKC beta 1 (9E18) Rabbit Monoclonal Antibody

Catalog #: AMRe16192

For research use only.

Summary

Description Recombinant rabbit monoclonal antibody

Host Rabbit

Application WB,IHC,ICC/IF,FC

Reactivity Human,Mouse,Rat

Conjugation Unconjugated

Modification Unmodified

Isotype IgG

Clonality Monoclonal
Form Liquid

Concentration 0.5mg/ml. The concentration of this product may be batch-dependent. **Storage** Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.

Shipping Ice bags

Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% New type preservative

Buffer N and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw

cycle.

Purification Affinity purification

Application

Dilution Ratio WB 1:500-1:2000,IHC 1:50-1:200,ICC/IF 1:100-1:200,FC 1:100-1:200

Molecular Weight 77kDa

Antigen Information

Alternative Names

Gene Name PRKCB

protein kinase C beta type; PRKCB; Prkcb; PKC-B; PKC-beta; PRKCB1; PKC Beta-I;

EC:2.7.11.13

 Gene ID
 5579.0

 SwissProt ID
 P05771

Immunogen A synthetic peptide of human PKC beta 1

Background



Plays a key role in B-cell activation and function by regulating BCR-induced NF-kappa-B activation and B-cell suvival. Calciumactivated, phospholipid- and diacylglycerol (DAG)- dependent serine/threonine-protein kinase involved in various cellular processes such as regulation of the B-cell receptor (BCR) signalosome, oxidative stress-induced apoptosis, androgen receptordependent transcription regulation, insulin signaling and endothelial cells proliferation. Plays a key role in B-cell activation by regulating BCR- induced NF-kappa-B activation. Mediates the activation of the canonical NF-kappa-B pathway (NFKB1) by direct phosphorylation of CARD11/CARMA1 at 'Ser-559', 'Ser-644' and 'Ser-652'. Phosphorylation induces CARD11/CARMA1 association with lipid rafts and recruitment of the BCL10-MALT1 complex as well as MAP3K7/TAK1, which then activates IKK complex, resulting in nuclear translocation and activation of NFKB1. Plays a direct role in the negative feedback regulation of the BCR signaling, by down-modulating BTK function via direct phosphorylation of BTK at 'Ser-180', which results in the alteration of BTK plasma membrane localization and in turn inhibition of BTK activity (PubMed:11598012). Involved in apoptosis following oxidative damage: in case of oxidative conditions, specifically phosphorylates 'Ser-36' of isoform p66Shc of SHC1, leading to mitochondrial accumulation of p66Shc, where p66Shc acts as a reactive oxygen species producer. Acts as a coactivator of androgen receptor (AR)-dependent transcription, by being recruited to AR target genes and specifically mediating phosphorylation of 'Thr-6' of histone H3 (H3T6ph), a specific tag for epigenetic transcriptional activation that prevents demethylation of histone H3 'Lys-4' (H3K4me) by LSD1/KDM1A (PubMed:20228790). In insulin signaling, may function downstream of IRS1 in muscle cells and mediate insulin-dependent DNA synthesis through the RAF1-MAPK/ERK signaling cascade. Participates in the regulation of glucose transport in adipocytes by negatively modulating the insulinstimulated translocation of the glucose transporter SLC2A4/GLUT4. Phosphorylates SLC2A1/GLUT1, promoting glucose uptake by SLC2A1/GLUT1 (PubMed:25982116). Under high glucose in pancreatic beta-cells, is probably involved in the inhibition of the insulin gene transcription, via regulation of MYC expression. In endothelial cells, activation of PRKCB induces increased phosphorylation of RB1, increased VEGFA-induced cell proliferation, and inhibits PI3K/AKT-dependent nitric oxide synthase (NOS3/eNOS) regulation by insulin, which causes endothelial dysfunction. Also involved in triglyceride homeostasis (By similarity). Phosphorylates ATF2 which promotes cooperation between ATF2 and JUN, activating transcription (PubMed:19176525).

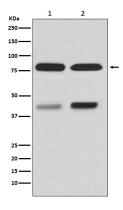
Research Area

Signal Transduction

Image Data

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Western blot analysis of PKC beta 1 expression in (1) HeLa cell lysate; (2) NIH/3T3 cell lysate.