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Goat anti-PRKD1 (aa383-395) Antibody

Peptide-affinity purified goat antibody
Catalog # AF4423a

Product Information

Application	IP, Pep-ELISA
Primary Accession	Q15139
Other Accession	NP_002733.2
Reactivity	Human
Host	Goat
Clonality	Polyclonal
Clone Names	PRKD1
Calculated MW	101704

Additional Information

Gene ID	5587
Other Names	PRKD1; PKC-MU; PKCM; PKD; PRKCM;
Dilution	IP~~N/A Pep-ELISA~~N/A
Format	Supplied at 0.5 mg/ml in Tris saline, 0.02% sodium azide, pH7.3 with 0.5% bovine serum albumin. Aliquot and store at -20°C. Minimize freezing and thawing.
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Goat anti-PRKD1 (aa383-395) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PRKD1
Synonyms	PKD, PKD1, PRKCM
Function	Serine/threonine-protein kinase that converts transient diacylglycerol (DAG) signals into prolonged physiological effects downstream of PKC, and is involved in the regulation of MAPK8/JNK1 and Ras signaling, Golgi membrane integrity and trafficking, cell survival through NF-kappa-B activation, cell migration, cell differentiation by mediating HDAC7 nuclear export, cell proliferation via MAPK1/3 (ERK1/2) signaling, and plays a role in cardiac hypertrophy, VEGFA-induced angiogenesis, genotoxic-induced apoptosis and flagellin-stimulated inflammatory response (PubMed:10764790,

PubMed:12505989, PubMed:12637538, PubMed:17442957, PubMed:18509061, PubMed:19135240, PubMed:19211839). Phosphorylates the epidermal growth factor receptor (EGFR) on dual threonine residues, which leads to the suppression of epidermal growth factor (EGF)-induced MAPK8/JNK1 activation and subsequent JUN phosphorylation (PubMed:10523301). Phosphorylates RIN1, inducing RIN1 binding to 14-3-3 proteins YWHAB, YWHAE and YWHAZ and increased competition with RAF1 for binding to GTP-bound form of Ras proteins (NRAS, HRAS and KRAS). Acts downstream of the heterotrimeric G protein beta/gamma-subunit complex to maintain the structural integrity of the Golgi membranes, and is required for protein transport along the secretory pathway. In the trans-Golgi network (TGN), regulates the fission of transport vesicles that are on their way to the plasma membrane. May act by activating the lipid kinase phosphatidylinositol 4-kinase beta (PI4KB) at the TGN for the local synthesis of phosphorylated inositol lipids, which induces a sequential production of DAG, phosphatidic acid (PA) and lyso-PA (LPA) that are necessary for membrane fission and generation of specific transport carriers to the cell surface. Under oxidative stress, is phosphorylated at Tyr-463 via SRC-ABL1 and contributes to cell survival by activating IKK complex and subsequent nuclear translocation and activation of NFKB1 (PubMed:12505989). Involved in cell migration by regulating integrin alpha-5/beta-3 recycling and promoting its recruitment in newly forming focal adhesion. In osteoblast differentiation, mediates the bone morphogenetic protein 2 (BMP2)-induced nuclear export of HDAC7, which results in the inhibition of HDAC7 transcriptional repression of RUNX2 (PubMed:18509061). In neurons, plays an important role in neuronal polarity by regulating the biogenesis of TGN-derived dendritic vesicles, and is involved in the maintenance of dendritic arborization and Golgi structure in hippocampal cells. May potentiate mitogenesis induced by the neuropeptide bombesin or vasopressin by mediating an increase in the duration of MAPK1/3 (ERK1/2) signaling, which leads to accumulation of immediate-early gene products including FOS that stimulate cell cycle progression. Plays an important role in the proliferative response induced by low calcium in keratinocytes, through sustained activation of MAPK1/3 (ERK1/2) pathway. Downstream of novel PKC signaling, plays a role in cardiac hypertrophy by phosphorylating HDAC5, which in turn triggers XPO1/CRM1-dependent nuclear export of HDAC5, MEF2A transcriptional activation and induction of downstream target genes that promote myocyte hypertrophy and pathological cardiac remodeling (PubMed:18332134). Mediates cardiac troponin I (TNNI3) phosphorylation at the PKA sites, which results in reduced myofilament calcium sensitivity, and accelerated crossbridge cycling kinetics. The PRKD1-HDAC5 pathway is also involved in angiogenesis by mediating VEGFA-induced specific subset of gene expression, cell migration, and tube formation (PubMed:19211839). In response to VEGFA, is necessary and required for HDAC7 phosphorylation which induces HDAC7 nuclear export and endothelial cell proliferation and migration. During apoptosis induced by cytarabine and other genotoxic agents, PRKD1 is cleaved by caspase-3 at Asp-378, resulting in activation of its kinase function and increased sensitivity of cells to the cytotoxic effects of genotoxic agents (PubMed:10764790). In epithelial cells, is required for transducing flagellin-stimulated inflammatory responses by binding and phosphorylating TLR5, which contributes to MAPK14/p38 activation and production of inflammatory cytokines (PubMed:17442957). Acts as an activator of NLRP3 inflammasome assembly by mediating phosphorylation of NLRP3 (By similarity). May play a role in inflammatory response by mediating activation of NF-kappa-B. May be involved in pain transmission by directly modulating TRPV1 receptor (PubMed:15471852). Plays a role in activated KRAS-mediated stabilization of ZNF304 in colorectal cancer (CRC) cells (PubMed:24623306). Regulates nuclear translocation of transcription factor TFEB in macrophages upon live S.enterica infection (By similarity). Acts as a regulator of social behavior by mediating phosphorylation of OXTR, enhancing G(q)-dependent G protein-coupled

receptor signaling (PubMed:35104164).

Cellular Location	Cytoplasm. Cell membrane. Golgi apparatus, trans-Golgi network {ECO:0000250|UniProtKB:Q62101}. Note=Translocation to the cell membrane is required for kinase activation

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.