

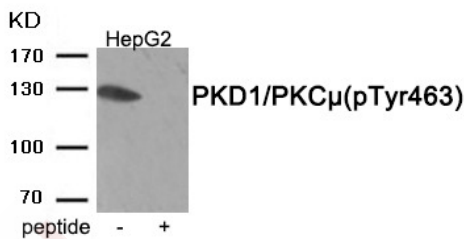
Anti-Phospho-PKD1/PKC μ (Tyr463) Polyclonal Antibody

Product Details

Ig Type:	IgG
Reactivity:	Human,Mouse,Rat
Conjugation:	Unconjugated
Molecular Weight:	Actual: 130 kDa.
Purification:	Antibodies were produced by immunizing rabbits with synthetic phosphopeptide and KLH conjugates. Antibodies were purified by affinity-chromatography using epitope-specific phosphopeptide. Non-phospho specific antibodies were removed by chromatography using non-phosphopeptide.

Applications

Verified Activity: 1. Western blot analysis of extracts from HepG2 tissue using PKD1/PKC μ (Phospho-Tyr463) antibody TMAC-03345. The lane on the right is treated with the antigen-specific peptide.



Application:	WB
Recommended	WB: 1:500-1000

Properties

Stability & Storage:	Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Peptide sequence around phosphorylation site of Tyrosine463 (R-Y-Y(p)-K-E) derived from Human PKD1/PKC μ
Antigen Species:	human
Synonyms:	p-PKD1/PKC μ (Y463);PKD1/PKC μ (p-Tyr463);PKD1/PKC μ (p-Y463);p-PKD1/PKC μ (Tyr463)

Research Background

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. 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.

Phosphorylates RIN1, inducing RIN1 binding to 14-3-3 proteins YWHAB, YWHAЕ 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. 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 morphogenic protein 2 (BMP2)-induced nuclear export of HDAC7, which results in the inhibition of HDAC7 transcriptional repression of RUNX2. 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. 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. 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. 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. 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.

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