Recombinant PKC mu Monoclonal Antibody

catalog number: **AN301985L**

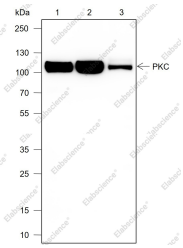
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Human
Immunogen	Peptide. This information is proprietary to PTMab
Host	Rabbit
Isotype	Rabbit IgG
Clone	C331606
Purification	Protein A affinity purification
Conjugation	Unconjugated
Buffer	PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protein protectant.

Applications	Recommended Dilution
WB	1:1000

Data



Western Blot with PKC mu Monoclonal Antibody at dilution of 1:1000. Lane 1: HeLa, Lane 2: PC-3, Lane 3: LNCaP

Observed-MW:110 kDa

Calculated-MW:102 kDa

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze/thaw cycles.
Shipping	Ice bag

Background

Activation of protein kinase C (PKC) is one of the earliest events in a cascade that controls a variety of cellular responses, including secretion, gene expression, proliferation, and muscle contraction. PKC isoforms belong to three groups based on calcium dependency and activators. Classical PKCs are calcium-dependent via their C2 domains and are activated by phosphatidylserine (PS), diacylglycerol (DAG), and phorbol esters (TPA, PMA) through their cysteine-rich C1 domains. Both novel and atypical PKCs are calcium-independent, but only novel PKCs are activated by PS, DAG, and phorbol esters. Members of these three PKC groups contain a pseudo-substrate or autoinhibitory domain that binds to substrate-binding sites in the catalytic domain to prevent activation in the absence of cofactors or activators. Control of PKC activity is regulated through three distinct phosphorylation events. Phosphorylation occurs in vivo at Thr500 in the activation loop, at Thr641 through autophosphorylation, and at the carboxy-terminal hydrophobic site Ser660. Atypical PKC isoforms lack hydrophobic region phosphorylation, which correlates with the presence of glutamic acid rather than the serine or threonine residues found in more typical PKC isoforms. The enzyme PDK1 or a close relative is responsible for PKC activation. A recent addition to the PKC superfamily is PKCμ (PKD), which is regulated by DAG and TPA through its C1 domain. PKD is distinguished by the presence of a PH domain and by its unique substrate recognition and Golgi localization. PKC-related kinases (PRK) lack the C1 domain and do not respond to DAG or phorbol esters. Phosphatidylinositol lipids activate PRKs, and small Rho-family GTPases bind to the homology region 1 (HR1) to regulate PRK kinase activity.