

**PKC $\beta$ II (D9S2U) Rabbit mAb**

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP, FC-FP	H M R	Endogenous	80	Rabbit IgG	#P05771-2	5579

**Product Usage Information****Application**

Western Blotting  
Immunoprecipitation  
Flow Cytometry (Fixed/Permeabilized)

**Dilution**

1:1000  
1:100  
1:400

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100  $\mu$ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at  $-20^{\circ}\text{C}$ . Do not aliquot the antibody.

**Specificity/Sensitivity**

PKC $\beta$ II (D9S2U) Rabbit mAb recognizes endogenous levels of total PKC $\beta$ II protein. This antibody does not cross-react with PKC $\beta$ I protein.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Val647 of human PKC $\beta$ II protein.

**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 (1,2). 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 (3-5). 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 (2). 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 PKC $\mu$  (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 (6). 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 (7). PKC $\beta$  has two isoforms, PKC $\beta$ I and PKC $\beta$ II, due to alternative splicing (8).

**Background References**

1. Nishizuka, Y. (1984) *Nature* 308, 693-8.
2. Keranen, L.M. et al. (1995) *Curr Biol* 5, 1394-403.
3. Mellor, H. and Parker, P.J. (1998) *Biochem J* 332 ( Pt 2), 281-92.
4. Ron, D. and Kazanietz, M.G. (1999) *FASEB J* 13, 1658-76.
5. Moscat, J. and Diaz-Meco, M.T. (2000) *EMBO Rep* 1, 399-403.
6. Baron, C.L. and Malhotra, V. (2002) *Science* 295, 325-8.
7. Flynn, P. et al. (2000) *J Biol Chem* 275, 11064-70.
8. Kawakami, T. et al. (2002) *J Biochem* 132, 677-82.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v nonfat dry milk, 1X TBS, 0.1% Tween $\text{\textcircled{R}}$  20 at  $4^{\circ}\text{C}$  with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting **IP:** Immunoprecipitation **FC-FP:** Flow Cytometry (Fixed/Permeabilized)

**Cross-Reactivity Key**

**H:** Human **M:** Mouse **R:** Rat

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