Revision 1

#9616 Store at -20C

PKCδ (D10E2) Rabbit mAb

Cell Signaling TECHNOLOGY®

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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:
WB, IP	H M R Mk	Endogenous	78	Rabbit IgG	#Q05655	5580

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity / Sensitivity

PKCδ (D10E2) Rabbit mAb recognizes endogenous levels of total PKCδ protein. This antibody does not cross-react with other PKC isoforms.

Species predicted to react based on 100% sequence homology:

Xenopus, Bovine, Dog, Horse

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Arg216 of human PKCδ 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 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 (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).
PKCδ is classified among the calcium-independent, diacylglycerol-activated "novel" members of the PKC superfamily, which includes PKCδ, ε, η, and θ. Unlike other PKC family members, whose activation appears to contribute to tumorigenesis, PKCδ appears to function as a tumor suppresor as down-regulation of this enzyme is associated with tumor progression (8). Like other conventional and novel PKCs, PKCδ is potently activated by diacylglycerol and phorbol ester and its kinase activity is modulated by phosphorylation within the conserved activation loop (Thr505) as well as the autophosphorylation site (Ser645) and hydrophobic, carboxy-terminal residue (Ser664) (9-11). Interestingly, PKCδ funtionality is uniquely regulated by phosphorylation at tyrosine residues by receptor tyrosine kinases, members of the Src kinase family, and c-Abl (9,12-14). For more information regarding PKCδ phosphorylation sites, please see PhosphoSitePlus® (www.phosphosite.org).

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. Lu, Z. et al. (1997) *Mol Cell Biol* 17, 3418-28.
9. Benes, C. and Soltoff, S.P. (2001) *Am J Physiol Cell Physiol* 280, C1498-510.
10. Li, W. et al. (1997) *J Biol Chem* 272, 24550-5.
11. Le Good, J.A. et al. (1998) *Science* 281, 2042-5.
12. Morita, M. et al. (2008) *J Biochem* 143, 31-8.
13. Denning, M.F. et al. (1996) *J Biol Chem* 271, 5325-31.
14. Kronfeld, I. et al. (2000) *J Biol Chem* 275, 35491-8.

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® 20 at 4°C with gentle shaking, overnight.

Applications Key

WB: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: human **M:** mouse **R:** rat **Hm:** hamster **Mk:** monkey **Vir:** virus **Mi:** mink **C:** chicken **Dm:** D. melanogaster **X:** Xenopus **Z:** zebrafish **B:** bovine **Dg:** dog **Pg:** pig **Sc:** S. cerevisiae **Ce:** C. elegans **Hr:** horse **GP:** Guinea Pig **Rab:** rabbit **All:** all species expected

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Limited Uses