

**Phospho-PKCδ/θ (Ser643/676) Antibody**

**Orders:** 877-616-CELL (2355)  
orders@cellsignal.com

**Support:** 877-678-TECH (8324)

**Web:** info@cellsignal.com  
cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IHC-P	H M R Mk X	Endogenous	78	Rabbit	#Q05655, #Q04759	5580, 5588

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

Western Blotting  
Immunohistochemistry (Paraffin)

**Dilution**

1:1000  
1:50

**Storage**

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

**Specificity/Sensitivity**

Phospho-PKCdelta/theta (Ser643/676) Antibody detects endogenous levels of PKCdelta only when phosphorylated at serine 643, and PKCtheta only when phosphorylated at serine 676. This antibody does not cross-react with the phosphorylated PKC isoforms alpha, beta, gamma or epsilon.

**Source / Purification**

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser643 of rat PKCdelta. Antibodies are purified by protein A and peptide affinity chromatography.

**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).

**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.

**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 BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting **IHC-P:** Immunohistochemistry (Paraffin)

**Cross-Reactivity Key**

**H:** Human **M:** Mouse **R:** Rat **Mk:** Monkey **X:** Xenopus

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