

Phospho-PKC (pan) (gamma Thr514) (D6Y3D) Rabbit mAb



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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	HMR	Endogenous	78, 80, 82, 85	Rabbit IgG	#P05771, #P17252,	5579, 5578, 5583,
		_		_	#P24723, #Q05655,	5580, 5588, 5581,
					#P05771-2,	5582
					#Q04759,	
					#Q02156, #P05129	

Product Usage Application Dilution Information 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.

Phospho-PKC (pan) (gamma Thr514) (D6Y3D) Rabbit mAb detects endogenous levels of PKC alpha, beta Specificity/Sensitivity

I, beta II, gamma, delta, epsilon, eta, and theta isoforms only when phosphorylated at a residue homologous to Thr514 of human PKC gamma. This antibody does not detect PKC phosphorylated at

other sites.

Source / Purification Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide

corresponding to residues surrounding Thr514 of human PKC gamma.

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 nonfat

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

Applications Key W: Western Blotting IP: Immunoprecipitation **Cross-Reactivity Key** H: Human M: Mouse R: Rat

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