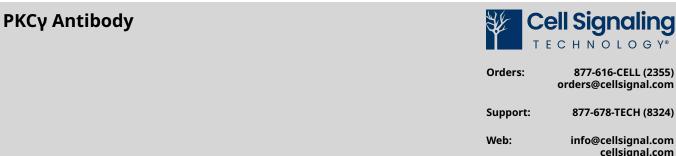
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877-678-TECH (8324)

info@cellsignal.com cellsignal.com

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Applications: W, IP	Reactivity: H M R	Sensitivity: Endogenous	MW (kDa): 80	Source/Isotype: Rabbit	UniProt ID: #P05129	Entrez-Gene Id: 5582
Product Usage Information		Application Western Blotting Immunoprecipitation			Dilution 1:1000 1:200	
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		PKCγ Antibody recognizes endogenous levels of total PKCγ protein.				
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy-terminus of human ΡΚCγ protein. 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, 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		 Nishizuka, Y. (1984) <i>Nature</i> 308, 693-8. Keranen, L.M. et al. (1995) <i>Curr Biol</i> 5, 1394-403. Mellor, H. and Parker, P.J. (1998) <i>Biochem J</i> 332 (Pt 2), 281-92. Ron, D. and Kazanietz, M.G. (1999) <i>FASEB J</i> 13, 1658-76. Moscat, J. and Diaz-Meco, M.T. (2000) <i>EMBO Rep</i> 1, 399-403. Baron, C.L. and Malhotra, V. (2002) <i>Science</i> 295, 325-8. Flynn, P. et al. (2000) <i>J Biol Chem</i> 275, 11064-70. 				
Species Reactiv	vitv	Species reactivity is det	ermined by testing	g in at least one approve	ed application (e.g.,	western blot).
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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				
Trademarks and Patents		Cell Signaling Technology is a trademark of Cell Signaling Technology, Inc.				

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