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# Polyclonal Antibody to PKCγ [pT655] Phosphospecific Antibody

**Catalog No.:** BP7165

**Quantity:** 0.1 ml

**Concentration:** 0.5 mg/ml

**Host:** Rabbit

**Immunogen:** The antiserum was produced against a chemically synthesized phosphopeptide derived from a region of human PKCγ that contains threonine 655. The sequence is conserved in mouse and rat.

**Applications:** The antibody has been used for Western blotting. For Western blotting applications, we recommend using the antibody at 0.5-1.0 µg/mL. At 0.50 µg/mL, the dilution provides 100 mL working solution, which at 10 mL/blot allows 10 blots to be performed. Positive controls used: Hela cells treated with PMA, a phorbol ester. Other applications not tested. Optimal dilutions of this antibody are dependent on conditions and should be determined by the user.
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**Specificity:** PKCγ is an 80 kDa member of the conventional group (cPKCs: sensitive to calcium, diacylglycerol and phorbol esters) of the PKC family of serine/threonine family kinases that are involved in a wide range of physiological processes including mitogenesis, cell survival and transcriptional regulation. PKCγ plays a key role in neuronal signal transduction and is translocated from the nucleus to the cytoplasm upon activation by phorbol ester, where in epithelial cells it has been implicated in regulating intracellular communication. The activation loop threonine (threonine 514 in PKCγ) of conventional PKCs is phosphorylated by phosphoinositide-dependent kinase-1 (PDK1), which is necessary for their autophosphorylation, a critical step in the generation of a catalytically mature enzyme. Threonine 655 is an autophosphorylation site in the carboxy-terminus of PKCγ. Human PKCγ. Mouse and rat (100% homologous) PKCγ have not been tested, but are expected to react. PKCα (69%) and βII (62%) may cross-react in cells expressing high levels of these proteins.

**Storage:** Store at 4°C short term only. Aliquot and store at -20°C to -80°C for longer. Avoid repeated freezing and thawing. Shelf life: one year from despatch.

**General Readings:** Wagner, L.M., et al. (2002) Effect of protein kinase Cγ on gap junction disassembly in lens epithelial cells and retinal cells in culture. Mol. Vis. 8:59-66.
Dutil, E.M., et al. (1998) Regulation of conventional protein kinase C isozymes by phosphoinositide-dependent kinase 1 (PDK-1). Curr. Biol. 8(25):1366-1375.
Toker, A. (1998) Signaling through protein kinase C. Front. Biosci. 3:D1134-D1147.
Machide, M., et al. (1998) Selective activation of phospholipase C γ1 and distinct protein

kinase C subspecies in intracellular signaling by hepatocyte growth factor/scatter factor in primary cultured rat neocortical cells. J. Neurochem. 71(2):592-602.
Battaini, F., et al. (1995) Protein kinase C activity, translocation, and conventional isoforms in aging rat brain. Neurobiol. Aging 16(2):137-148.
Quest, A.F. and R.M. Bell (1994) The regulatory region of protein kinase Cγ. Studies of phorbol ester binding to individual and combined functional segments expressed as glutathione S-transferase fusion proteins indicate a complex mechanism of regulation by phospholipids, phorbol esters, and divalent cations. J. Biol. Chem. 269(31):20000-20012.
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