

## PI 3-Kinase alpha<sup>GST</sup> (bovine)

Phosphoinositide 3-Kinase  $\alpha$  GST, p110 $\alpha$  GST/p85 $\alpha$

bovine, recombinant, Sf9 cells

Cat. No.	Amount
PR-940	10 $\mu$ g

For *in vitro* use only  
 Quality guaranteed for 12 months  
 Store at -20°C

### Avoid freeze / thaw cycles

#### Form

Liquid. Supplied in 25 mM Tris-HCl pH 8.0, 50 mM NaCl, 0.5 mM MgCl<sub>2</sub> and 50% glycerol.

#### Molecular Weight

p110 $\alpha$ : 124.3 kDa (without Tag)  
 p85 $\alpha$ : 83.5 kDa.

#### Activity

3 nmol/mg/min using phosphatidylinositol as substrate.

#### Purity

>90% by SDS-PAGE

### Description

PI3K $\alpha$  plays a specific role in apoptosis in human colon cancer cells. Injection of neutralizing antibodies specific to PI3K $\alpha$  into adenocarcinoma cells induced apoptosis, a response that was reverted by treating cells with caspase inhibitor.

It was also shown that PI3K $\alpha$  mediated phosphorylation of the p85 $\alpha$  adapter reduces the lipid kinase activity of the heterodimer and this gives hints for PI3K-dependent signaling events not requiring production of 3'-phosphorylated phosphoinositides. PI3K $\alpha$  is a key regulator of the initiation of keratinocyte differentiation. A decrease in PI3K activity results in a loss of keratinocyte adhesion to the extracellular membrane and the initiation of early phase differentiation.

The PI3K $\alpha$ &#61472;catalytic and regulatory subunits are coexpressed in Sf9 insect cells. The catalytic subunit carries a GST-Tag and the heterodimer was purified by affinity chromatography.

**The GST-Tag facilitates the protein's application in typical GST pull-down assays.**

### General

Phosphoinositide 3-kinases (PI3Ks) phosphorylate phosphatidylinositols (PIs) at their 3' OH position generating lipid second messengers and thereby regulate numerous biological processes including cell growth, differentiation, survival, proliferation, migration and metabolism. On the basis of structural similarities and substrate specificity, the PI3K family can be subdivided into three classes termed I, II, and III.

All human class I members are heterodimers consisting of a catalytic subunit (MW approx. 110 kDa) and a non-catalytic subunit (MW 50, 55, 85, or 101 kDa) and are known to phosphorylate phosphatidylinositol (PI), phosphatidylinositol-4-monophosphate (PIP) and phosphatidylinositol-4,5-bisphosphate (PIP2) *in vitro*. The class I members can be further subdivided into class IA and IB PI3Ks. Class IA exists in three isoforms (p110 $\alpha$ , p110 $\beta$  and p110 $\delta$ &#61481; whereas the only class IB member is termed p110 $\gamma$ .

Class IA PI3Ks are activated by adaptor proteins such as Ras or BCAP, or tyrosine-kinase-associated receptors including antigen, co-stimulatory and cytokine receptors (e.g. CD19, CD28, Insulin receptor, EGFR, and PDGFR). p110 $\gamma$  is activated by G-protein-coupled receptors (GPCRs). Effectors of class I PI3Ks are pleckstrin-homology domain proteins such as Akt/PKB, BTK, TEC, ITK, BAM32, and small GTPases (e.g. Cdc42, Rac, or

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Ras).

The action of PI3Ks is regulated by the phosphatidylinositol-3,4,5-trisphosphate phosphatases SHIP and PTEN.

### Selected References:

Graupera *et al.* (2008) Angiogenesis selectively requires the p110 $\alpha$  isoform of PI3K to control endothelial cell migration. *Nature* **453**:662.

Foster *et al.* (2003) The phosphoinositide (PI) 3-kinase family. *J. Cell Science* **116**:3037.

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Pirola *et al.* (2001) Activation Loop Sequences Confer Substrate Specificity to Phosphoinositide 3-Kinase  $\alpha$  (PI3K $\alpha$ ). *J. Biol. Chem.* **276**:21544.

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