

PI 3-Kinase alpha (bovine)

Phosphoinositide 3-Kinase α , p110 α /p85 α
bovine, recombinant, Sf9 cells

Cat. No.	Amount
PR-341	10 μ 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 HEPES, pH 8.0, 25 mM NaCl, 2.5 mM MgCl₂, 50% glycerol.

Activity

1,000 units/mg (1 unit is defined as 1 picomole phosphate transferred to PIP₂ per minute)

Molecular weight

p110 α : 124.3 kDa
p85 α : 83.5 kDa

Purity

>90% by SDS PAGE

Description

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

It was also shown that PI3K α mediated phosphorylation of the p85 α 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 α is a key regulator of the initiation of keratinocyte differentiation. A decrease in PI3K α activity results in a loss of keratinocyte adhesion to the extracellular membrane and the initiation of early phase differentiation.

The PI3K α catalytic and regulatory subunits are co-expressed in Sf9 insect cells.

Note

Bovine PI 3-Kinase alpha differs from the human enzyme in only 2 positions, K532R and S535C. Both lie in the PIK domain of the enzyme (aa 525-696) and are not expected to interfere with binding of p85 (aa 31-108) or Ras (aa 173-292) or with catalytic function (aa 699-1064).

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-mono-phosphate (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 α , p110 β and p110 δ); whereas the only class IB member is termed p110 γ .

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).

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p110 γ 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 Ras).

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

Selected References:

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