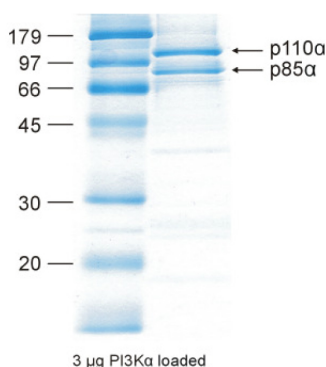


PI 3-Kinase alpha^{His} Phosphoinositide 3-Kinase α^{His} , p110 α^{His} /p85 α human, recombinant, Sf9 cells

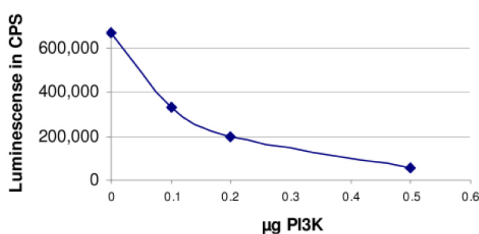
| Cat. No. | Amount |
|----------|------------------|
| PR-335 | 10 μg |

SDS-PAGE analysis



Activity test

ATP depletion assay, PI as substrate, 1 μM ATP



For *in vitro* use only
Quality guaranteed for 12 months
Store at -80°C

Avoid freeze / thaw cycles

Form

Liquid. Supplied in 300 mM NaCl, 50 mM Tris HCl, 0.05% Brij35, 10% glycerol pH 7.9.

Activity

9,900 units/mg
1 unit equals 1 picomole phosphate transferred to PIP2 per min.

Activity test

ATP depletion assay, PI as substrate, 1 μM ATP.

Molecular Weight

p110 α : 128.3 kDa
p85 α : 83.5 kDa

Purity

>90% by SDS-PAGE

Description

Purified recombinant human PI3 Kinase expressed in Sf9 cells as a complex of catalytic p110 α and regulatory p85 α subunits.

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.

Recombinant human PI3K α is expressed in Sf9 cells and carries a N-terminal polyhistidine affinity Tag at the p110 α subunit.

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

PI 3-Kinase alpha^{His} Phosphoinositide 3-Kinase α^{His} , p110 α^{His} /p85 α human, recombinant, Sf9 cells

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). 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:

- Graupera *et al.* (2008) Angiogenesis selectively requires the p110 α 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.
- Sayama *et al.* (2002) Phosphatidylinositol 3-kinase is a key regulator of early phase differentiation in keratinocytes. *J. Biol. Chem.* **277**:40390.
- Cantrell D.A. (2001) Phosphoinositide 3-kinase signalling pathways. *J. Cell Sci.* **114**:1439.
- Pirola *et al.* (2001) Activation Loop Sequences Confer Substrate Specificity to Phosphoinositide 3-Kinase α (PI3K α). *J. Biol. Chem.* **276**:21544.
- Yin *et al.* (1998) Involvement of p85 in p53-dependent apoptotic response to oxidative stress. *Nature* **391**:707.
- Shepherd *et al.* (1996) The role of phosphoinositide 3-kinase in insulin signalling. *Journal of Molecular Endocrinology* **17**:175.