

PI(3)K β

Phosphoinositide 3-kinase p110 β /p85 α

human, recombinant, Sf9 insect cells

Cat. No.	Amount
PR-344	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 10 mM HEPES, pH 8.0, 100 mM NaCl, 0.5 mM MgCl₂, 10% glycerol.

Activity

3,000 units/mg (1 unit is defined as 1 picomole phosphate transferred to PIP₂ per minute)

Molecular weight

p110 β : 124.3 kDa

p85 α : 83.6 kDa

Purity

>90% by SDS PAGE

Description

The PI3K β isoform can be activated by insulin via the insulin receptor to initiate a cascade of events that control cell growth and metabolism. The activation of PI3K β is mediated by the p85 regulatory subunit binding to tyrosine phosphorylated insulin receptor substrate (IRS) proteins (e.g. IRS-1 and IRS-2).

It was also shown that PI3K β is involved in apoptosis in human colon carcinoma cells. Injection of neutralizing antibodies specific to p110 β in WiDr, HCT116 and CO 115 adenocarcinoma cells inhibited *de novo* DNA synthesis.

PI3K β is the major PI3K isoform required for apoptotic cell and Fc- γ receptor mediated phagocytosis shown for primary mouse macrophages and the Jurkat human leukemia T cell line.

It was shown by several research groups that the catalytic subunit of PI3K β can be activated by G $\beta\gamma$ subunits of G-protein coupled receptors.

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 noncatalytic 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). 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

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phosphatidylinositol- 3,4,5-trisphosphate phosphatases
SHIP and PTEN.

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