

PI3K δ ^{GST}

Phosphoinositide 3-kinase p110 δ /p85 α
human, recombinant, Sf9 insect cells

Cat. No.	Amount
PR-345	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 Tris-HCl pH 8.0, 50 mM NaCl, 0.5 mM MgCl₂ and 50% glycerol.

Activity

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

Molecular Weight

p110 δ : 115 kDa without tag and 135 kDa with tag
p85 α : 83.5 kDa

Purity

>95% by SDS-PAGE

Description

The class IA PI3K δ is expressed in endothelial and in white blood cells. Recently it was shown that the inactivation of PI3K δ in bone marrow mast cells (BBMCs) leads to defective stem cell factor-mediated proliferation, adhesion and migration of these cells, and to impaired allergen-IgE-induced degranulation and cytokine release. In neutrophils a role for PI3K δ in TNF α -induced signalling was demonstrated by a reduction in Aktphosphorylation and PDK1 activity upon treatment with the δ -specific inhibitor IC87114.

The PI3K δ 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 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 phosphatidylinositol-3,4,5-trisphosphate phosphatases SHIP and PTEN.

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Selected References:

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- Sawyer *et al.* (2003) Regulation of breast cancer cell chemotaxis by the phosphoinositide 3-kinase p110delta. *Cancer Res.* **63**:1667.
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- Ali *et al.* (2004) Essential role for the p110 δ phosphoinositide 3-kinase in the allergic response. *Nature* **431**:1007.
- Puri *et al.* (2004) Mechanisms and implications of phosphoinositide 3-kinase δ in promoting neutrophil trafficking into inflamed tissue. *Blood* **103**:3448.