

## AKT/PKB substrate peptide RPRAATF

Cat. No.	Amount
PE-200	1 mg

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

### Avoid freeze / thaw cycles

### Form

Supplied as lyophilized powder. Reconstitution in 3 ml dd H<sub>2</sub>O results in a 400  $\mu$ M solution recommended for AKT1/PKB $\alpha$  activity assays.

### Sequence

RPRAATF

### Molecular Weight

818 g/mol.

### Purity

85-90% by HPLC.

### Description

The synthetic peptide RPRAATF can be used as a substrate for AKT/PKB in *in vitro* kinase assays. It is phosphorylated by AKT1/PKB $\alpha$  with a  $K_m$  of 25  $\mu$ M.

Protein kinase B or Akt (PKB/Akt) is a serine/threonine kinase, which in mammals comprises three highly homologous members known as PKB $\alpha$  (AKT1, Cat.-No. PR-324), PKB $\beta$  (AKT2, Cat.-No. PR-333), and PKB $\gamma$  (AKT3). AKT/PKB is activated in cells exposed to diverse stimuli such as hormones, growth factors, and extracellular matrix components. The activation mechanism remains to be fully characterised but occurs downstream of phosphoinositide 3-kinase (PI 3K). PI-3K generates phosphatidylinositol-3,4,5-trisphosphate (PIP(3)), lipid second messenger essential for the translocation of PKB/Akt to the plasma membrane where it is phosphorylated and activated by phosphoinositide-dependent kinase-1 (PDK-1) and possibly other kinases. PKB/Akt phosphorylates and regulates the function of many cellular proteins involved in processes that include metabolism, apoptosis, and proliferation. Recent evidence indicates that PKB/Akt is frequently constitutively active in many types of human cancer.

Akt1 contains a region homologous to a pleckstrin domain found in multiple signaling molecules and is stimulated by a number of receptor tyrosine kinases, including receptors for IGF, NGF, PDGF, VEGF, angiotensin, and insulin, by the action of phosphatidylinositol 3-kinase (PI 3-kinase).

### Selected References:

- Alessi *et al.* (1996) Molecular basis for the substrate specificity of protein kinase B: comparison with MAPKAP kinase-1 and p70 S6 kinase. *FEBS Lett.* **399**:333.
- Hixon *et al.* (2000) Akt1/PKB upregulation leads to vascular smooth muscle cell hypertrophy and polyploidization. *J. Clin. Invest.* **106**:1011.
- Nicholson *et al.* (2002) The protein kinase B/Akt signalling pathway in human malignancy. *Cell Signal.* **14**:381.
- Hoffmeister *et al.* (2008) Cyclic Nucleotide-dependent Protein Kinases Inhibit Binding of 14-3-3 to the GTPase-activating Protein Rap1GAP2 in Platelets. *J. Biol. Chem.* **283**(4):2297-2306.