abcam

Product datasheet

Modified AKT substrate peptide ab101578

製品の詳細

製品名 Modified AKT substrate peptide

精製度 > 95 % HPLC.

Animal free No

由来 Recombinant

配列 modified-CKRPRAASFAE

予測される分子量 1 kDa

配列の追加情報 The Modified AKT Substrate peptide sequence (modified-CKRPRAASFAE) is based on the N-

terminus of GSK3.

製品の詳細 AKT1 peptide

特性

Our Abpromise quarantee covers the use of ab101578 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

製品の状態 Liquid

備考 The Modified AKT Substrate peptide sequence (modified-CKRPRAASFAE) is based on the N-

terminus of GSK3. This peptide is modified at the N-terminus resulting in enhanced

phosphorylation by protein kinases compared to phosphorylation of the non-modified AKT

Substrate.

Modified AKT Substrate can be utilized as a substrate for the following active protein kinases:

ab60322

前処理および保存

保存方法および安定性 Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid repeated freeze / thaw cycles.

pH: 7.00

Constituent: Water

関連情報

機能 Plays a role as a key modulator of the AKT-mTOR signaling pathway controlling the tempo of the

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process of newborn neurons integration during adult neurogenesis, including correct neuron positioning, dendritic development and synapse formation (By similarity). General protein kinase capable of phosphorylating several known proteins. Phosphorylates TBC1D4. Signals downstream of phosphatidylinositol 3-kinase (Pl(3)K) to mediate the effects of various growth factors such as platelet-derived growth factor (PDGF), epidermal growth factor (EGF), insulin and insulin-like growth factor I (IGF-I). Plays a role in glucose transport by mediating insulin-induced translocation of the GLUT4 glucose transporter to the cell surface. Mediates the antiapoptotic effects of IGF-I. Mediates insulin-stimulated protein synthesis by phosphorylating TSC2 at 'Ser-939' and 'Thr-1462', thereby activating mTORC1 signaling and leading to both phosphorylation of 4E-BP1 and in activation of RPS6KB1. Promotes glycogen synthesis by mediating the insulin-induced activation of glycogen synthase. The activated form can suppress FoxO gene transcription and promote cell cycle progression. Essential for the SPATA13-mediated regulation of cell migration and adhesion assembly and disassembly.

組織特異性

関連疾患

配列類似性

ドメイン

翻訳後修飾

細胞内局在

Expressed in all human cell types so far analyzed. The Tyr-176 phosphorylated form shows a significant increase in expression in breast cancers during the progressive stages i.e. normal to hyperplasia (ADH), ductal carcinoma in situ (DCIS), invasive ductal carcinoma (IDC) and lymph node metastatic (LNMM) stages.

Defects in AKT1 are a cause of susceptibility to breast cancer (BC) [MIM:114480]. A common malignancy originating from breast epithelial tissue. Breast neoplasms can be distinguished by their histologic pattern. Invasive ductal carcinoma is by far the most common type. Breast cancer is etiologically and genetically heterogeneous. Important genetic factors have been indicated by familial occurrence and bilateral involvement. Mutations at more than one locus can be involved in different families or even in the same case.

Defects in AKT1 are associated with colorectal cancer (CRC) [MIM:114500].

Defects in AKT1 are associated with susceptibility to ovarian cancer [MIM:604370]; also called susceptibility to familial breast-ovarian cancer type 1 (BROVCA1).

Belongs to the protein kinase superfamily. AGC Ser/Thr protein kinase family. RAC subfamily. Contains 1 AGC-kinase C-terminal domain.

Contains 1 PH domain.

Contains 1 protein kinase domain.

Binding of the PH domain to the phosphatidylinositol 3-kinase alpha (PI(3)K) results in its targeting to the plasma membrane. The PH domain mediates interaction with TNK2 and Tyr-176 is also essential for this interaction.

The AGC-kinase C-terminal mediates interaction with THEM4.

Phosphorylation on Thr-308, Ser-473 and Tyr-474 is required for full activity. Activated TNK2 phosphorylates it on Tyr-176 resulting in its binding to the anionic plasma membrane phospholipid PA. This phosphorylated form localizes to the cell membrane, where it is targeted by PDPK1 and PDPK2 for further phosphorylations on Thr-308 and Ser-473 leading to its activation. Ser-473 phosphorylation by mTORC2 favors Thr-308 phosphorylation by PDPK1. Ser-473 phosphorylation is enhanced by interaction with AGAP2 isoform 2 (PIKE-A). Ser-473 phosphorylation is enhanced in focal cortical dysplasias with Taylor-type balloon cells. Ubiquitinated; undergoes both 'Lys-48'- and 'Lys-63'-linked polyubiquitination. TRAF6-induced 'Lys-63'-linked AKT1 ubiquitination is critical for phosphorylation and activation. When ubiquitinated, it translocates to the plasma membrane, where it becomes phosphorylated. When fully phosphorylated and translocated into the nucleus, undergoes 'Lys-48'-polyubiquitination catalyzed by TTC3, leading to its degradation by the proteasome.

Cytoplasm. Nucleus. Cell membrane. Nucleus after activation by integrin-linked protein kinase 1 (ILK1). Nuclear translocation is enhanced by interaction with TCL1A. Phosphorylation on Tyr-176 by TNK2 results in its localization to the cell membrane where it is targeted for further phosphorylations on Thr-308 and Ser-473 leading to its activation and the activated form

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