

**AKT1/2/3 Blocking Peptide (Center)**  
**Synthetic peptide**  
**Catalog # BP20658c****Specification****AKT1/2/3 Blocking Peptide (Center) - Product Information**

Primary Accession [P31749](#)  
Other Accession [Q63484](#), [Q9WUA6](#),  
[Q9Y243](#), [P47197](#),  
[Q60823](#), [P31751](#),  
[Q98TY9](#), [P47196](#),  
[P31750](#), [Q01314](#)

**AKT1/2/3 Blocking Peptide (Center) - Additional Information****Gene ID 207****Other Names**

RAC-alpha serine/threonine-protein kinase,  
Protein kinase B, PKB, Protein kinase B  
alpha, PKB alpha, Proto-oncogene c-Akt,  
RAC-PK-alpha, AKT1, PKB, RAC

**Target/Specificity**

The synthetic peptide sequence is selected  
from aa 278-292 of HUMAN AKT1

**Format**

Peptides are lyophilized in a solid powder  
format. Peptides can be reconstituted in  
solution using the appropriate buffer as  
needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6  
months. For long term storage store at  
-20°C.

**Precautions**

This product is for research use only. Not  
for use in diagnostic or therapeutic  
procedures.

**AKT1/2/3 Blocking Peptide (Center) - Protein Information****Name AKT1****AKT1/2/3 Blocking Peptide (Center) - Background**

AKT1 is one of 3 closely related serine/threonine- protein kinases (AKT1, AKT2 and AKT3) called the AKT kinase, and which regulate many processes including metabolism, proliferation, cell survival, growth and angiogenesis. This is mediated through serine and/or threonine phosphorylation of a range of downstream substrates. Over 100 substrate candidates have been reported so far, but for most of them, no isoform specificity has been reported. AKT is responsible of the regulation of glucose uptake by mediating insulin-induced translocation of the SLC2A4/GLUT4 glucose transporter to the cell surface. Phosphorylation of PTPN1 at 'Ser-50' negatively modulates its phosphatase activity preventing dephosphorylation of the insulin receptor and the attenuation of insulin signaling. Phosphorylation of TBC1D4 triggers the binding of this effector to inhibitory 14-3-3 proteins, which is required for insulin-stimulated glucose transport. AKT regulates also the storage of glucose in the form of glycogen by phosphorylating GSK3A at 'Ser-21' and GSK3B at 'Ser-9', resulting in inhibition of its kinase activity. Phosphorylation of GSK3 isoforms by AKT is also thought to be one mechanism by which cell proliferation is driven. AKT regulates also cell survival via the phosphorylation of MAP3K5 (apoptosis signal-related kinase). Phosphorylation of 'Ser-83' decreases MAP3K5 kinase activity stimulated by oxidative stress and thereby prevents apoptosis. AKT 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. AKT is involved in the phosphorylation of members of the FOXO factors (Forkhead family of transcription factors), leading to binding of 14-3-3 proteins and cytoplasmic localization. In particular, FOXO1 is phosphorylated at 'Thr-24', 'Ser-256' and 'Ser-319'. FOXO3 and

## Synonyms PKB, RAC

### Function

AKT1 is one of 3 closely related serine/threonine-protein kinases (AKT1, AKT2 and AKT3) called the AKT kinase, and which regulate many processes including metabolism, proliferation, cell survival, growth and angiogenesis (PubMed:<a href="http://www.uniprot.org/citations/15526160" target="\_blank">15526160</a>, PubMed:<a href="http://www.uniprot.org/citations/11882383" target="\_blank">11882383</a>, PubMed:<a href="http://www.uniprot.org/citations/21620960" target="\_blank">21620960</a>, PubMed:<a href="http://www.uniprot.org/citations/21432781" target="\_blank">21432781</a>). This is mediated through serine and/or threonine phosphorylation of a range of downstream substrates (PubMed:<a href="http://www.uniprot.org/citations/15526160" target="\_blank">15526160</a>, PubMed:<a href="http://www.uniprot.org/citations/11882383" target="\_blank">11882383</a>, PubMed:<a href="http://www.uniprot.org/citations/21620960" target="\_blank">21620960</a>, PubMed:<a href="http://www.uniprot.org/citations/21432781" target="\_blank">21432781</a>). Over 100 substrate candidates have been reported so far, but for most of them, no isoform specificity has been reported (PubMed:<a href="http://www.uniprot.org/citations/15526160" target="\_blank">15526160</a>, PubMed:<a href="http://www.uniprot.org/citations/11882383" target="\_blank">11882383</a>, PubMed:<a href="http://www.uniprot.org/citations/21620960" target="\_blank">21620960</a>, PubMed:<a href="http://www.uniprot.org/citations/21432781" target="\_blank">21432781</a>). AKT is responsible of the regulation of glucose uptake by mediating insulin-induced translocation of the SLC2A4/GLUT4 glucose transporter to the cell surface (By similarity). Phosphorylation of PTPN1 at 'Ser-50' negatively modulates its phosphatase activity preventing dephosphorylation of the insulin receptor and the attenuation of insulin signaling (By

FOXO4 are phosphorylated on equivalent sites. AKT has an important role in the regulation of NF-kappa-B-dependent gene transcription and positively regulates the activity of CREB1 (cyclic AMP (cAMP)- response element binding protein). The phosphorylation of CREB1 induces the binding of accessory proteins that are necessary for the transcription of pro-survival genes such as BCL2 and MCL1. AKT phosphorylates 'Ser-454' on ATP citrate lyase (ACLY), thereby potentially regulating ACLY activity and fatty acid synthesis. Activates the 3B isoform of cyclic nucleotide phosphodiesterase (PDE3B) via phosphorylation of 'Ser-273', resulting in reduced cyclic AMP levels and inhibition of lipolysis. Phosphorylates PIKFYVE on 'Ser-318', which results in increased PI(3)P-5 activity. The Rho GTPase-activating protein DLC1 is another substrate and its phosphorylation is implicated in the regulation cell proliferation and cell growth. AKT plays a role as key modulator of the AKT-mTOR signaling pathway controlling the tempo of the process of newborn neurons integration during adult neurogenesis, including correct neuron positioning, dendritic development and synapse formation. Signals downstream of phosphatidylinositol 3-kinase (PI(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). AKT mediates the antiapoptotic effects of IGF-I. Essential for the SPATA13-mediated regulation of cell migration and adhesion assembly and disassembly. May be involved in the regulation of the placental development. Phosphorylates STK4/MST1 at 'Thr-120' and 'Thr-387' leading to inhibition of its: kinase activity, nuclear translocation, autophosphorylation and ability to phosphorylate FOXO3. Phosphorylates STK3/MST2 at 'Thr- 117' and 'Thr-384' leading to inhibition of its: cleavage, kinase activity, autophosphorylation at Thr-180, binding to RASSF1 and nuclear translocation. Phosphorylates SRPK2 and enhances its kinase activity towards SRSF2 and ACIN1 and promotes its nuclear translocation. Phosphorylates RAF1 at 'Ser-259' and negatively regulates its activity. Phosphorylation of BAD stimulates its pro-apoptotic activity. Phosphorylates KAT6A at 'Thr-369' and this phosphorylation inhibits the interaction of KAT6A with PML and negatively regulates its acetylation activity towards

similarity). Phosphorylation of TBC1D4 triggers the binding of this effector to inhibitory 14-3-3 proteins, which is required for insulin-stimulated glucose transport (PubMed:<a href="http://www.uniprot.org/citations/11994271" target="\_blank">11994271</a>). AKT regulates also the storage of glucose in the form of glycogen by phosphorylating GSK3A at 'Ser-21' and GSK3B at 'Ser-9', resulting in inhibition of its kinase activity (By similarity). Phosphorylation of GSK3 isoforms by AKT is also thought to be one mechanism by which cell proliferation is driven (By similarity). AKT regulates also cell survival via the phosphorylation of MAP3K5 (apoptosis signal-related kinase) (PubMed:<a href="http://www.uniprot.org/citations/11154276" target="\_blank">11154276</a>). Phosphorylation of 'Ser-83' decreases MAP3K5 kinase activity stimulated by oxidative stress and thereby prevents apoptosis (PubMed:<a href="http://www.uniprot.org/citations/11154276" target="\_blank">11154276</a>). AKT 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 (PubMed:<a href="http://www.uniprot.org/citations/12150915" target="\_blank">12150915</a>). AKT is involved in the phosphorylation of members of the FOXO factors (Forkhead family of transcription factors), leading to binding of 14-3-3 proteins and cytoplasmic localization (PubMed:<a href="http://www.uniprot.org/citations/10358075" target="\_blank">10358075</a>). In particular, FOXO1 is phosphorylated at 'Thr-24', 'Ser-256' and 'Ser-319' (PubMed:<a href="http://www.uniprot.org/citations/10358075" target="\_blank">10358075</a>). FOXO3 and FOXO4 are phosphorylated on equivalent sites (PubMed:<a href="http://www.uniprot.org/citations/10358075" target="\_blank">10358075</a>). AKT has an important role in the regulation of NF-kappa-B-dependent gene transcription and positively regulates the activity of CREB1 (cyclic AMP (cAMP)-response element binding protein) (PubMed:<a href="http://www.uniprot.org/citations/9829964" target="\_blank">9829964</a>). The

p53/TP53.

### **AKT1/2/3 Blocking Peptide (Center) - References**

Jones P.F.,et al.Proc. Natl. Acad. Sci. U.S.A. 88:4171-4175(1991).  
Matsubara A.,et al.Diabetologia 44:910-913(2001).  
Ota T.,et al.Nat. Genet. 36:40-45(2004).  
Goshima N.,et al.Nat. Methods 5:1011-1017(2008).  
Coffer P.J.,et al.Eur. J. Biochem. 201:475-481(1991).

phosphorylation of CREB1 induces the binding of accessory proteins that are necessary for the transcription of pro-survival genes such as BCL2 and MCL1 (PubMed:<a href="http://www.uniprot.org/citations/9829964" target="\_blank">9829964</a>). AKT phosphorylates 'Ser-454' on ATP citrate lyase (ACLY), thereby potentially regulating ACLY activity and fatty acid synthesis (By similarity). Activates the 3B isoform of cyclic nucleotide phosphodiesterase (PDE3B) via phosphorylation of 'Ser-273', resulting in reduced cyclic AMP levels and inhibition of lipolysis (By similarity). Phosphorylates PIKFYVE on 'Ser-318', which results in increased PI(3)P-5 activity (By similarity). The Rho GTPase-activating protein DLC1 is another substrate and its phosphorylation is implicated in the regulation cell proliferation and cell growth. AKT plays a role as key modulator of the AKT-mTOR signaling pathway controlling the tempo of the process of newborn neurons integration during adult neurogenesis, including correct neuron positioning, dendritic development and synapse formation (By similarity). Signals downstream of phosphatidylinositol 3-kinase (PI(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) (PubMed:<a href="http://www.uniprot.org/citations/12176338" target="\_blank">12176338</a>, PubMed:<a href="http://www.uniprot.org/citations/12964941" target="\_blank">12964941</a>). AKT mediates the antiapoptotic effects of IGF-I (By similarity). Essential for the SPATA13-mediated regulation of cell migration and adhesion assembly and disassembly (PubMed:<a href="http://www.uniprot.org/citations/19934221" target="\_blank">19934221</a>). May be involved in the regulation of the placental development (By similarity). Phosphorylates STK4/MST1 at 'Thr-120' and 'Thr-387' leading to inhibition of its: kinase activity, nuclear translocation, autophosphorylation and ability to phosphorylate FOXO3 (PubMed:<a href="http://www.uniprot.org/citations/17726016" target="\_blank">17726016</a>). Phosphorylates STK3/MST2 at 'Thr-117' and

'Thr-384' leading to inhibition of its:  
cleavage, kinase activity,  
autophosphorylation at Thr-180, binding to  
RASSF1 and nuclear translocation  
(PubMed:<a href="http://www.uniprot.org/citations/20086174"  
target="\_blank">20086174</a>,  
PubMed:<a href="http://www.uniprot.org/citations/20231902"  
target="\_blank">20231902</a>).  
Phosphorylates SRPK2 and enhances its  
kinase activity towards SRSF2 and ACIN1  
and promotes its nuclear translocation  
(PubMed:<a href="http://www.uniprot.org/citations/19592491"  
target="\_blank">19592491</a>).  
Phosphorylates RAF1 at 'Ser-259' and  
negatively regulates its activity (PubMed:<a href="http://www.uniprot.org/citations/10576742" target="\_blank">10576742</a>).  
Phosphorylation of BAD stimulates its  
pro-apoptotic activity (PubMed:<a href="http://www.uniprot.org/citations/10926925" target="\_blank">10926925</a>).  
Phosphorylates KAT6A at 'Thr-369' and this  
phosphorylation inhibits the interaction of  
KAT6A with PML and negatively regulates  
its acetylation activity towards p53/TP53  
(PubMed:<a href="http://www.uniprot.org/citations/23431171" target="\_blank">23431171</a>).  
Phosphorylates palladin (PALLD),  
modulating cytoskeletal organization and  
cell motility (PubMed:<a href="http://www.uniprot.org/citations/20471940" target="\_blank">20471940</a>).  
Phosphorylates prohibitin (PHB), playing an  
important role in cell metabolism and  
proliferation (PubMed:<a href="http://www.uniprot.org/citations/18507042" target="\_blank">18507042</a>).  
Phosphorylates CDKN1A, for which  
phosphorylation at 'Thr-145' induces its  
release from CDK2 and cytoplasmic  
relocalization (PubMed:<a href="http://www.uniprot.org/citations/16982699" target="\_blank">16982699</a>). These  
recent findings indicate that the AKT1  
isoform has a more specific role in cell  
motility and proliferation (PubMed:<a href="http://www.uniprot.org/citations/16139227" target="\_blank">16139227</a>).  
Phosphorylates CLK2 thereby controlling  
cell survival to ionizing radiation  
(PubMed:<a href="http://www.uniprot.org/citations/20682768" target="\_blank">20682768</a>).

Phosphorylates PCK1 at 'Ser-90', reducing the binding affinity of PCK1 to oxaloacetate and changing PCK1 into an atypical protein kinase activity using GTP as donor (PubMed:<a href="http://www.uniprot.org/citations/32322062" target="\_blank">32322062</a>). Also acts as an activator of TMEM175 potassium channel activity in response to growth factors: forms the lysoK(GF) complex together with TMEM175 and acts by promoting TMEM175 channel activation, independently of its protein kinase activity (PubMed:<a href="http://www.uniprot.org/citations/32228865" target="\_blank">32228865</a>).

### **Cellular Location**

Cytoplasm

{ECO:0000250|UniProtKB:P31750}.

Nucleus. Cell membrane

{ECO:0000250|UniProtKB:P31750}.

Note=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 translocates to the nucleus. Colocalizes with WDFY2 in intracellular vesicles (PubMed:16792529).

### **Tissue Location**

Expressed in prostate cancer and levels increase from the normal to the malignant state (at protein level). 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.

## **AKT1/2/3 Blocking Peptide (Center) - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)