SunLong Biotech Co.,LTD
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Rabbit Anti-AKT1 antibody

SLM-52010R

Product Name	AKT1
Chinese Name	蛋白激酶 BRecombinant rabbit monoclonal anti
Alias	AKT 1; AKT; AKT1; AKT-1; AKT1_HUMAN; C AKT; cAKT; MGC9965; MGC99656; Onco PKB; PKB alpha; PKB-ALPHA; PRKBA; Protein Kinase B Alpha; Protein kinase B; Proto-onc RAC Alpha; RAC alpha serine/threonine protein kinase; RAC; RAC PK Alpha; Rac protein kina Serine/Threonine Protein Kinase; RAC-alpha serine/threonine-protein kinase; RAC-PK-alpha; v thymoma viral oncogene homolog 1; vAKT Murine Thymoma Viral Oncogene Homolog 1.
Research Area	Tumour Cell biology Neurobiology Signal transduction Apoptosis Kinases and Phosphatases
Immunogen Species	Rabbit
Clonality	Monoclonal
Clone NO.	11F2
React Species	Human,Mouse,Rat
Applications	WB=1:2000-10000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500,Flow-Cyt=1:50-100,ICC （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Theoretical molecular weight	56kDa
Cellular localization	The nucleus cytoplasmic The cell membrane
Form	Liquid
Concentration	1mg/ml
immunogen	Recombinant human AKT1 protein
Lsotype	IgG
Purification	affinity purified by Protein A
Buffer Solution	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
Storage	Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.

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Attention This product as supplied is intended for research use only, not for use in human, therapeutic or d applications.

PubMed PubMed

The serine-threonine protein kinase encoded by the AKT1 gene is catalytically inactive in serum-s and immortalized fibroblasts. AKT1 and the related AKT2 are activated by platelet-derived grov activation is rapid and specific, and it is abrogated by mutations in the pleckstrin homology dom was shown that the activation occurs through phosphatidylinositol 3-kinase. In the developing ne AKT is a critical mediator of growth factor-induced neuronal survival. Survival factors can suppr a transcription-independent manner by activating the serine/threonine kinase AKT1, which then and inactivates components of the apoptotic machinery. Mutations in this gene have been associa Proteus syndrome. Multiple alternatively spliced transcript variants have been found for this gen RefSeq, Jul 2011]

Product Detail

Function:

AKT1 is one of 3 closely related serine/threonine-protein kinases (AKT1, AKT2 and AKT3) cal kinase, and which regulate many processes including metabolism, proliferation, cell survival, gr angiogenesis. This is mediated through serine and/or threonine phosphorylation of a range of do substrates. Over 100 substrate candidates have been reported so far, but for most of them, no isof has been reported. AKT is responsible of the regulation of glucose uptake by mediating insulin-i translocation of the SLC2A4/GLUT4 glucose transporter to the cell surface. Phosphorylation of 'Ser-50' negatively modulates its phosphatase activity preventing dephosphorylation of the insuli the attenuation of insulin signaling. Phosphorylation of TBC1D4 triggers the binding of this effec 14-3-3 proteins, which is required for insulin-stimulated glucose transport. AKT regulates also t glucose in the form of glycogen by phosphorylating GSK3A at 'Ser-21' and GSK3B at 'Ser-9', re inhibition of its kinase activity. Phosphorylation of GSK3 isoforms by AKT is also thought to be by which cell proliferation is driven. AKT regulates also cell survival via the phosphorylation of (apoptosis signal-related kinase). Phosphorylation of 'Ser-83' decreases MAP3K5 kinase activity oxidative stress and thereby prevents apoptosis. AKT mediates insulin-stimulated protein synthe phosphorylating TSC2 at 'Ser-939' and 'Thr-1462', thereby activating mTORC1 signaling and lea phosphorylation of 4E-BP1 and in activation of RPS6KB1. AKT is involved in the phosphorylat of the FOXO factors (Forkhead family of transcription factors), leading to binding of 14-3-3 pro cytoplasmic localization. In particular, FOXO1 is phosphorylated at 'Thr-24', 'Ser-256' and 'Ser- and FOXO4 are phosphorylated on equivalent sites. AKT has an important role in the regulation NF-kappa-B-dependent gene transcription and positively regulates the activity of CREB1 (cyclic (cAMP)-response element binding protein). The phosphorylation of CREB1 induces the binding proteins that are necessary for the transcription of pro-survival genes such as BCL2 and MCL1. phosphorylates 'Ser-454' on ATP citrate lyase (ACLY), thereby potentially regulating ACLY act acid synthesis. Activates the 3B isoform of cyclic nucleotide phosphodiesterase (PDE3B) via pho 'Ser-273', resulting in reduced cyclic AMP levels and inhibition of lipolysis. Phosphorylates PIK 'Ser-318', which results in increased PI(3)P-5 activity. The Rho GTPase-activating protein DLC substrate and its phosphorylation is implicated in the regulation cell proliferation and cell growth role as key modulator of the AKT-mTOR signaling pathway controlling the tempo of the proces neurons integration during adult neurogenesis, including correct neuron positioning, dendritic de

synapse formation. Signals downstream of phosphatidylinositol 3-kinase (PI(3)K) to mediate the various growth factors such as platelet-derived growth factor (PDGF), epidermal growth factor (and insulin-like growth factor I (IGF-I). AKT mediates the antiapoptotic effects of IGF-I. Essent SPATA13-mediated regulation of cell migration and adhesion assembly and disassembly. May b the regulation of the placental development. Phosphorylates STK4/MST1 at 'Thr-120' and 'Thr-3 inhibition of its: kinase activity, nuclear translocation, autophosphorylation and ability to phospho Phosphorylates STK3/MST2 at 'Thr-117' and 'Thr-384' leading to inhibition of its: cleavage, kin autophosphorylation at Thr-180, binding to RASSF1 and nuclear translocation. Phosphorylates S enhances its kinase activity towards SRSF2 and ACIN1 and promotes its nuclear translocation. I RAF1 at 'Ser-259' and negatively regulates its activity. Phosphorylation of BAD stimulates its pr activity. AKT1-specific substrates have been recently identified, including palladin (PALLD), w phosphorylation modulates cytoskeletal organization and cell motility; prohibitin (PHB), playing role in cell metabolism and proliferation; and CDKN1A, for which phosphorylation at 'Thr-145' release from CDK2 and cytoplasmic relocalization. These recent findings indicate that the AKT more specific role in cell motility and proliferation. Phosphorylates CLK2 thereby controlling ce ionizing radiation.

Subunit:
Interacts (via the C-terminus) with CCDC88A (via its C-terminus). Interacts with GRB10; the in to GRB10 phosphorylation thus promoting YWHAE-binding. Interacts with AGAP2 (isoform 2/ interaction occurs in the presence of guanine nucleotides. Interacts with AKTIP. Interacts (via PI MTCP1, TCL1A AND TCL1B. Interacts with CDKN1B; the interaction phosphorylates CDKN 14-3-3 binding and cell-cycle progression. Interacts with MAP3K5 and TRAF6. Interacts with B STK3 and STK4. Interacts (via PH domain) with SIRT1. Interacts with SRPK2 in a phosphoryla manner. Interacts with RAF1. Interacts with TRIM13; the interaction ubiquitinates AKT1 leadin proteasomal degradation. Interacts with TNK2 and CLK2. Interacts (via the C-terminus) with TH C-terminus). Interacts with and phosphorylated by PDPK1.

Subcellular Location:
Cytoplasm. Nucleus. Cell membrane. Note=Nucleus after activation by integrin-linked protein k Nuclear translocation is enhanced by interaction with TCL1A. Phosphorylation on Tyr-176 by T its localization to the cell membrane where it is targeted for further phosphorylations on Thr-308 leading to its activation and the activated form translocates to the nucleus.

Tissue Specificity:
Expressed in prostate cancer and levels increase from the normal to the malignant state (at protei Expressed in all human cell types so far analyzed. The Tyr-176 phosphorylated form shows a sign in expression in breast cancers during the progressive stages i.e. normal to hyperplasia (ADH), du in situ (DCIS), invasive ductal carcinoma (IDC) and lymph node metastatic (LNMM) stages.

Post-translational modifications:
O-GlcNAcylation at Thr-305 and Thr-312 inhibits activating phosphorylation at Thr-308 via dis interaction between AKT1 and PDPK1. O-GlcNAcylation at Ser-473 also probably interferes wi phosphorylation at this site.

Phosphorylation on Thr-308, Ser-473 and Tyr-474 is required for full activity. Activated TNK2 pl
on Tyr-176 resulting in its binding to the anionic plasma membrane phospholipid PA. This phosp
localizes to the cell membrane, where it is targeted by PDPK1 and PDPK2 for further phosphory
Thr-308 and Ser-473 leading to its activation. Ser-473 phosphorylation by mTORC2 favors Thr-
phosphorylation by PDPK1. Ser-473 phosphorylation is enhanced by interaction with AGAP2 is
(PIKE-A). Ser-473 phosphorylation is enhanced in focal cortical dysplasias with Taylor-type bal
Ser-473 phosphorylation is enhanced by signaling through activated FLT3. Dephosphorylated at
Ser-473 by PP2A phosphatase. The phosphorylated form of PPP2R5B is required for bridging AI
phosphatase.
Ubiquitinated via 'Lys-48'-linked polyubiquitination by ZNRF1, leading to its degradation by the
Ubiquitinated; undergoes both 'Lys-48'- and 'Lys-63'-linked polyubiquitination. TRAF6-induced
AKT1 ubiquitination is critical for phosphorylation and activation. When ubiquitinated, it translo
plasma membrane, where it becomes phosphorylated. When fully phosphorylated and translocat
nucleus, undergoes 'Lys-48'-polyubiquitination catalyzed by TTC3, leading to its degradation by t
Also ubiquitinated by TRIM13 leading to its proteasomal degradation.
Acetylated on Lys-14 and Lys-20 by the histone acetyltransferases EP300 and KAT2B. Acetylat
reduced phosphorylation and inhibition of activity. Deacetylated at Lys-14 and Lys-20 by SIRT1
SIRT1-mediated deacetylation relieves the inhibition.

DISEASE:
Defects in AKT1 are a cause of susceptibility to breast cancer (BC) [MIM:114480]. A common
originating from breast epithelial tissue. Breast neoplasms can be distinguished by their histolog
Invasive ductal carcinoma is by far the most common type. Breast cancer is etiologically and ge
heterogeneous. Important genetic factors have been indicated by familial occurrence and bilatera
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].
Note=Genetic variations in AKT1 may play a role in susceptibility to ovarian cancer.
Defects in AKT1 are a cause of Proteus syndrome (PROTEUSS) [MIM:176920]. A highly varia
disorder of asymmetric and disproportionate overgrowth of body parts, connective tissue nevi, e
dysregulated adipose tissue, and vascular malformations. Many features of Proteus syndrome ov
overgrowth syndromes.

Similarity:
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.

SWISS:
P31749

Gene ID:
207

Database links:

Entrez Gene: 207 Human

Entrez Gene: 11651 Mouse

Entrez Gene: 24185 Rat

Omim: 164730 Human

SwissProt: O57513 Chicken

SwissProt: P31749 Human

SwissProt: P31750 Mouse

SwissProt: P47196 Rat

Unigene: 525622 Human

Unigene: 6645 Mouse

Unigene: 11422 Rat

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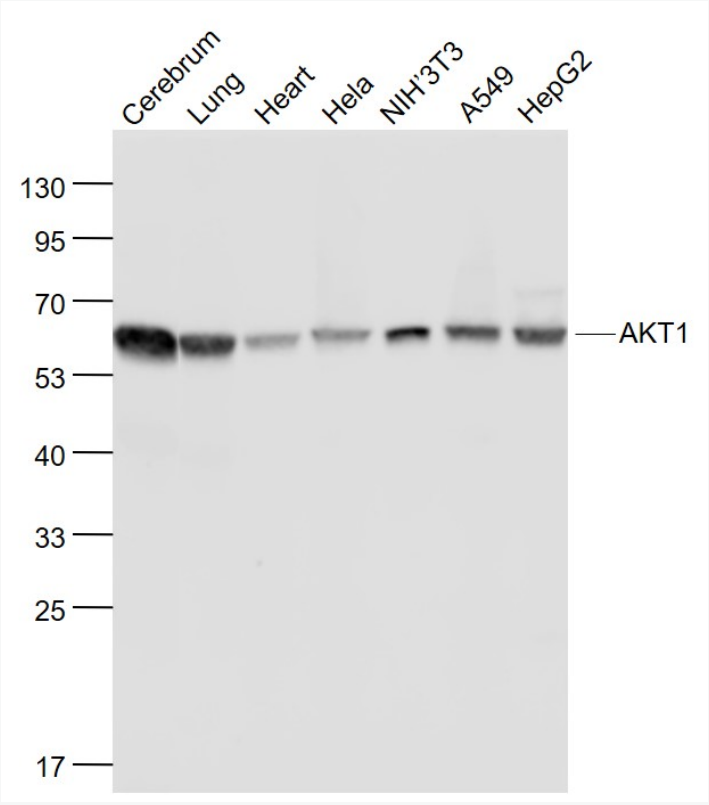
Product Picture



Sample:

Cerebrum (Mouse) Lysate at 40 ug

Lung (Mouse) Lysate at 40 ug

Heart (Mouse) Lysate at 40 ug

Hela(Human) Cell Lysate at 30 ug

NIH/3T3(Mouse) Cell Lysate at 30 ug

A549(Human) Cell Lysate at 30 ug

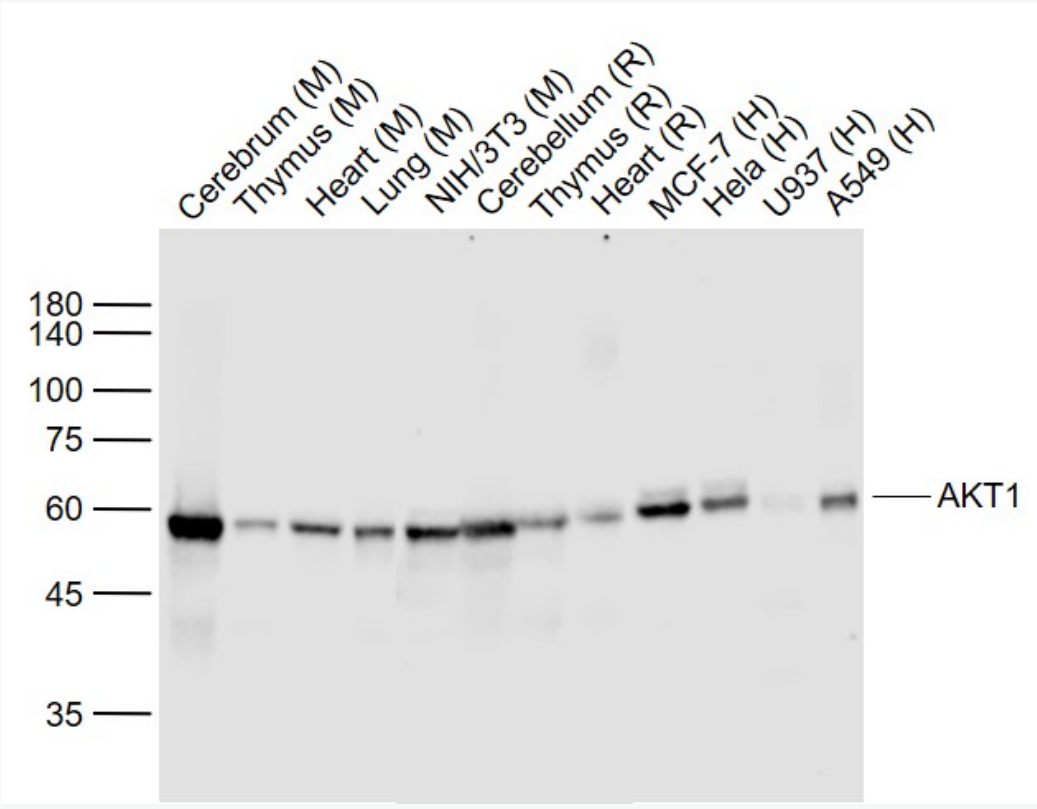
HepG2(Human) Cell Lysate at 30 ug

Primary: Anti- AKT1 (SLM-52010R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 56 kD

Observed band size: 56 kD



Sample:

Lane 1: Cerebrum (Mouse) Lysate at 40 ug

Lane 2: Thymus (Mouse) Lysate at 40 ug

Lane 3: Heart (Mouse) Lysate at 40 ug

Lane 4: Lung (Mouse) Lysate at 40 ug

Lane 5: NIH/3T3 (Mouse) Cell Lysate at 30 ug

Lane 6: Cerebellum (Rat) Lysate at 40 ug

Lane 7: Thymus (Rat) Lysate at 40 ug

Lane 8: Heart (Rat) Lysate at 40 ug

Lane 9: MCF-7 (Human) Cell Lysate at 30 ug

Lane 10: Hela (Human) Cell Lysate at 30 ug

Lane 11: U937 (Human) Cell Lysate at 30 ug

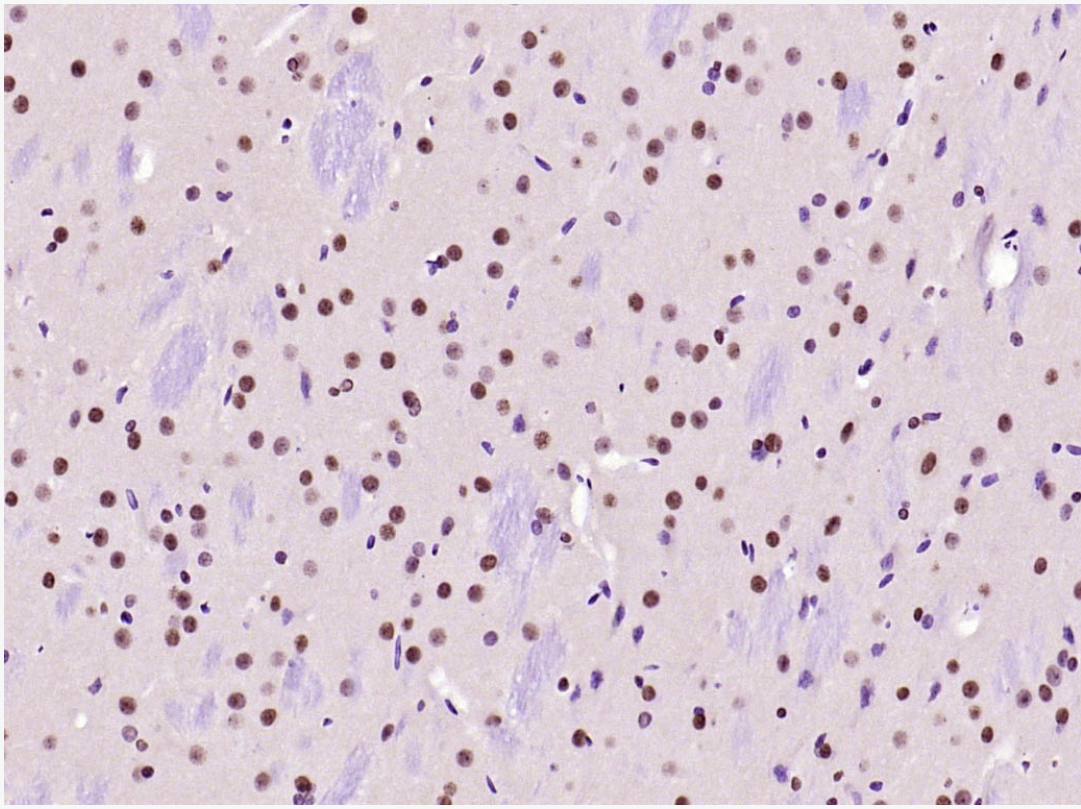
Lane 12: A549 (Human) Cell Lysate at 30 ug

Primary: Anti- AKT1 (SLM-52010R) at 1/1000 dilution

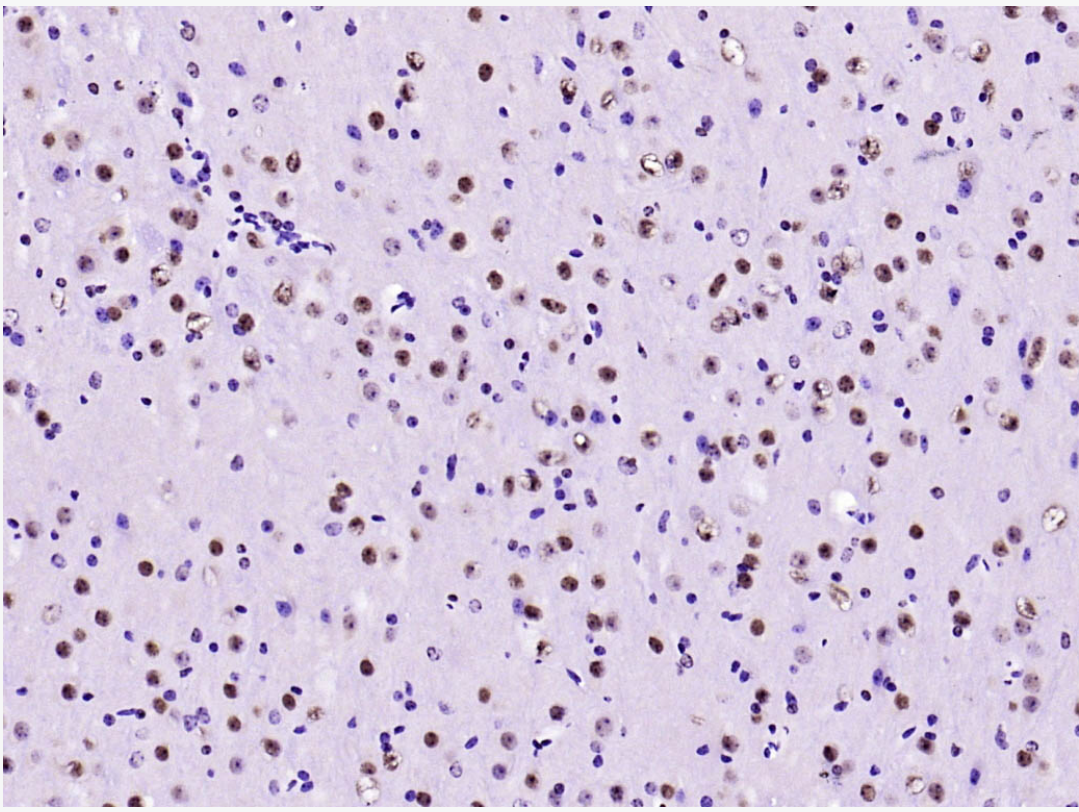
Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 59 kD

Observed band size: 59 kD



Paraformaldehyde-fixed, paraffin embedded (rat brain); Antigen retrieval by boiling in sodium
(pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Bl
(normal goat serum) at 37°C for 30min; Antibody incubation with (AKT1) Monoclonal Antib
Unconjugated (SLM-52010R) at 1:200 overnight at 4°C, followed by operating according to S
(sp-0023) instructionsand DAB staining.



Paraformaldehyde-fixed, paraffin embedded (mouse brain); Antigen retrieval by boiling in soc
buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minu
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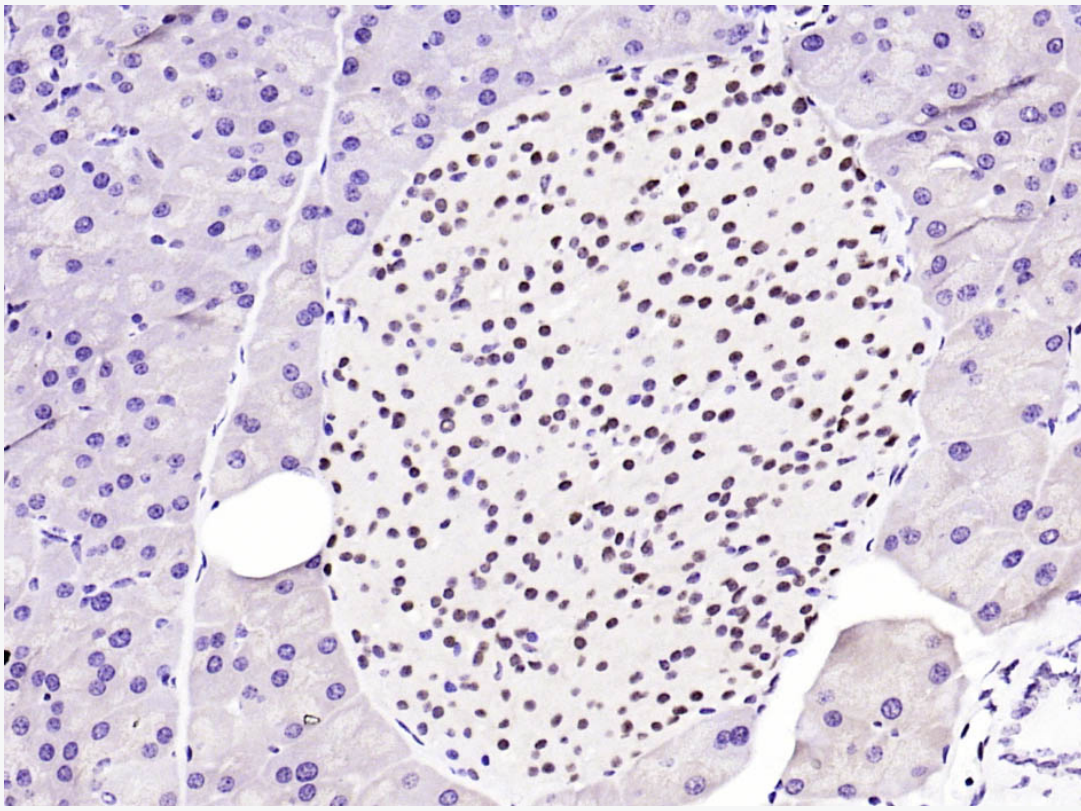

Paraformaldehyde-fixed, paraffin embedded (rat pancreas); Antigen retrieval by boiling in soc
buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minu
buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (AKT1) Monoclonal
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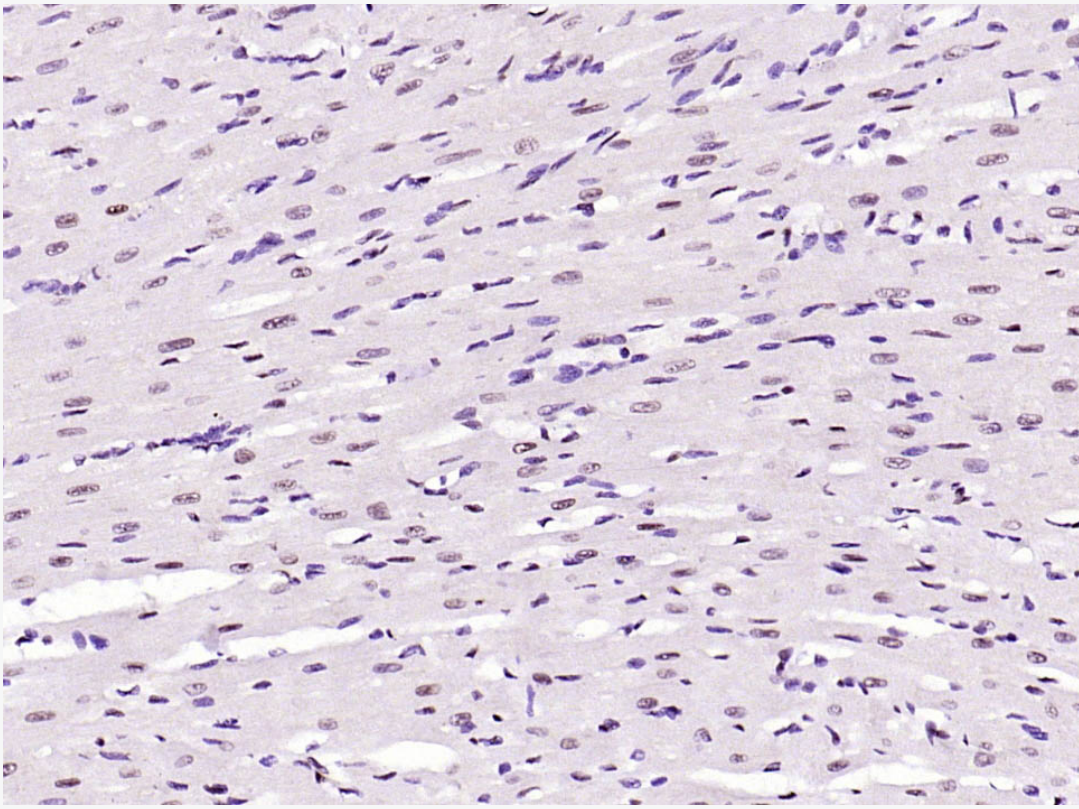

Paraformaldehyde-fixed, paraffin embedded (rat heart); Antigen retrieval by boiling in sodium
(pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Bl
(normal goat serum) at 37°C for 30min; Antibody incubation with (AKT1) Monoclonal Antib
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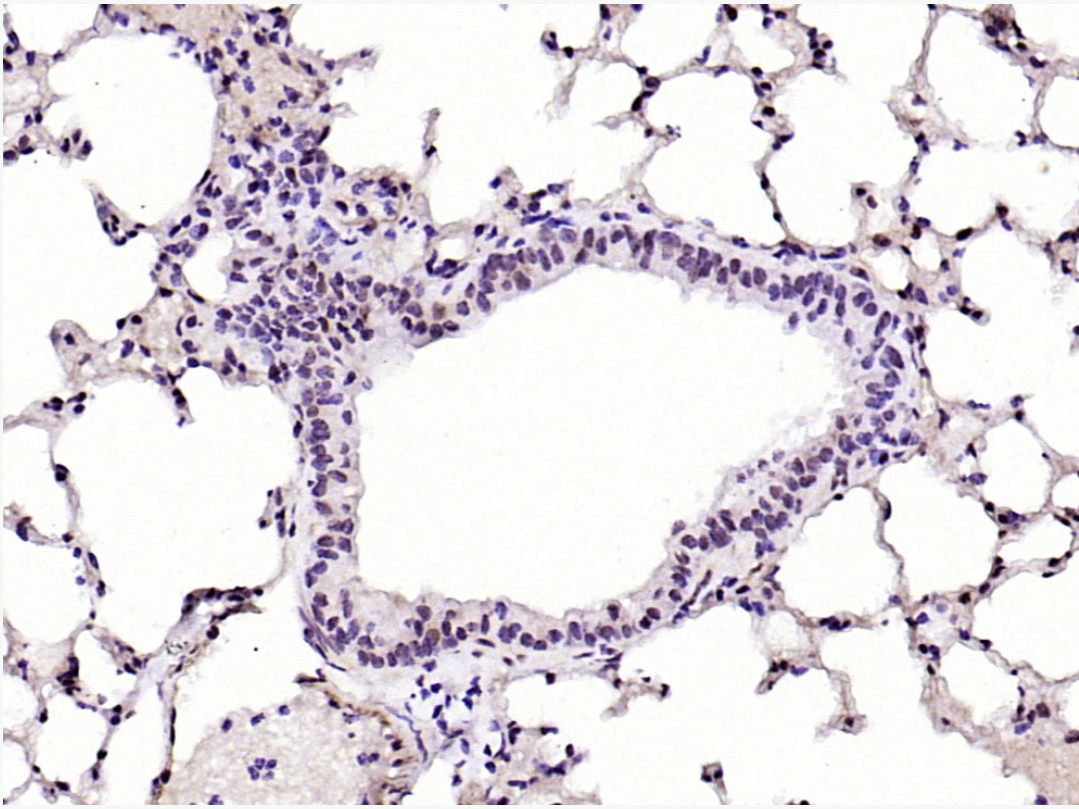

Paraformaldehyde-fixed, paraffin embedded (rat lung); Antigen retrieval by boiling in sodium
(pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Bl
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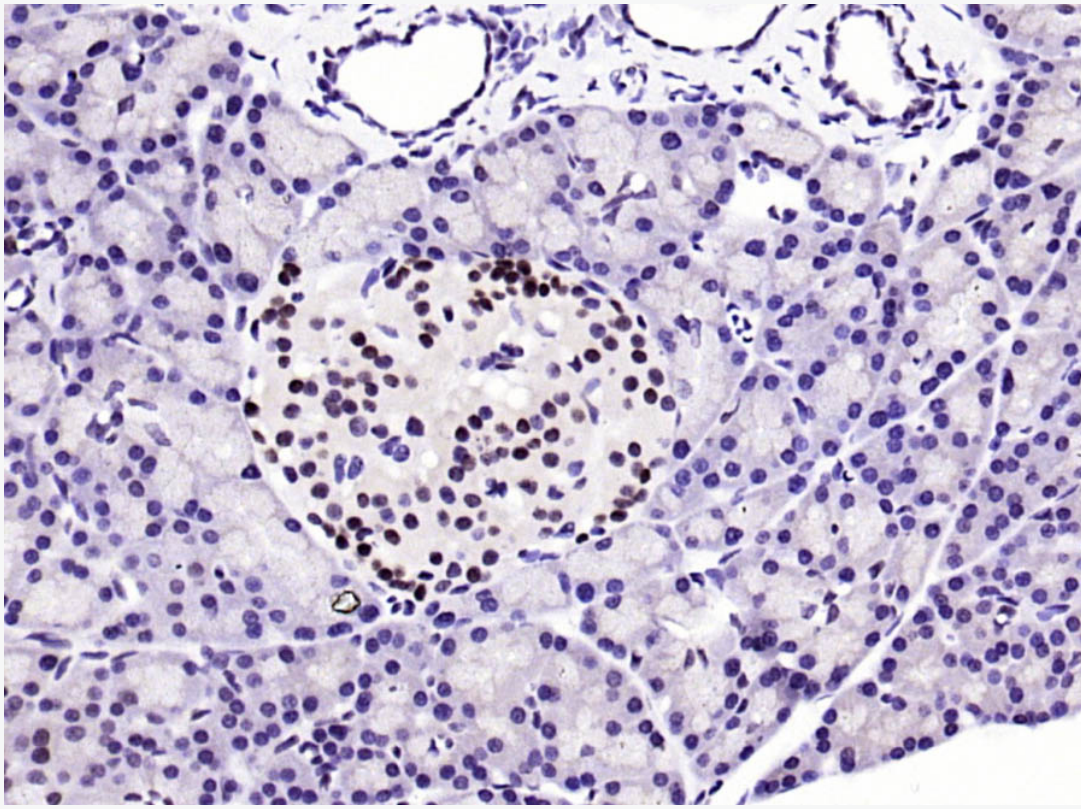

Paraformaldehyde-fixed, paraffin embedded (mouse pancreas); Antigen retrieval by boiling in s
buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minu
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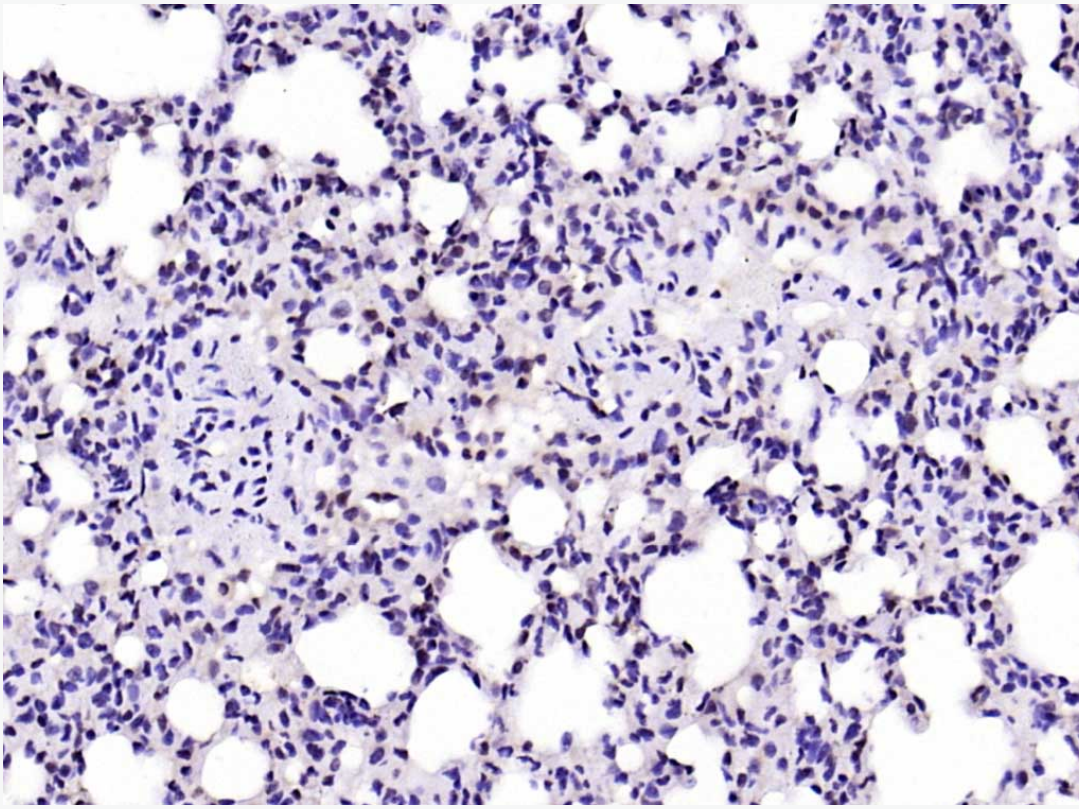

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