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Anti-Phospho-AKT1 (S129) Rabbit Monoclonal Antibody

Catalog Number: P00024-3

Overview

Product Name	Anti-Phospho-AKT1 (S129) Rabbit Monoclonal Antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-Phospho-AKT1 (S129) Rabbit Monoclonal Antibody catalog # P00024-3. Tested in WB application. This antibody reacts with Human, Mouse, Rat.
Application	WB
Clonality	Monoclonal HGD-1
Formulation	Rabbit IgG in stabilizing components, phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. *This antibody is supplied in a stabilized formulation. Compatibility with conjugation reactions depends on the chemistry of the conjugation method used. For conjugation methods that are not compatible with the stabilizing components present in this formulation, a carrier-free antibody format is required.
Storage Instructions	Store at -20°C for one year. For short term storage and frequent use, store at 4°C for up to one month. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P31749

Technical Details

Immunogen	A synthesized peptide derived from human AKT1
Isotype	Rabbit IgG
Form	Liquid
Concentration	0.5mg/ml
Purification	Affinity-chromatography
Suggested Dilutions	WB 1:500-2000

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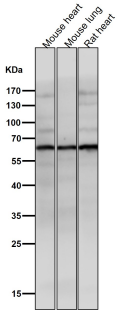
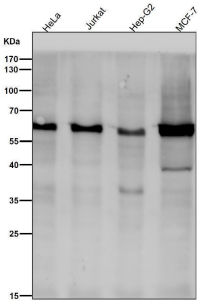
Anti-Phospho-AKT1 (S129) Rabbit Monoclonal Antibody (P00024-3) Images



All lanes use the Antibody at 1:1K dilution for 1 hour at room temperature.



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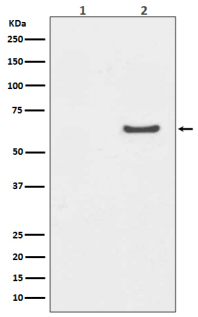
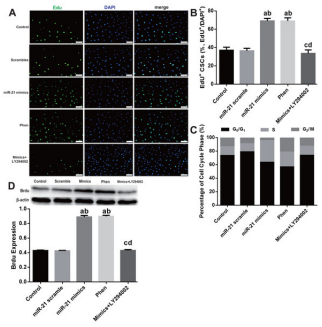


Western blot analysis of Phospho-AKT1 (S129) expression in (1) MCF-7 cell lysate treated with Alkaline Phosphatase; (2) MCF-7 cell lysate.



PTEN/PI3K/Akt pathway's contribution in miR-21 induced proliferation in c-kit + CSCs. Cultured c-kit + CSCs were treated with miR-21 mimics for 48 h before subjected to EdU immunofluorescence (A–B), flow cytometry (C) or Western blot (D). To test the contribution of PTEN/PI3K/Akt signaling, PTEN and PI3K were inhibited with Phen or LY294002 respectively. (A) c-kit + CSCs were double stained by EdU (green) and DAPI (blue), and observed under a fluorescence microscope (Olympus). Bar = 50 μm. DAPI = propidium iodide. (B) The statistics of EdU positive CSCs from immunofluorescence in (A). n = 6 in each group. (C) Flow cytometry was employed to detect cell cycle profiles in CSCs underwent different treatments miR-21 mimics or Phen increased the proportion of S phase CSCs compared with Control or scramble treated groups. n = 3. (D) PTEN/PI3K/Akt pathway's influences on BrdU expression, which was detected with immune blotting. Just like miR-21 mimics' effect on BrdU, when PTEN was inhibited by Phen, there was notably increase of BrdU compared with Normal or Scramble

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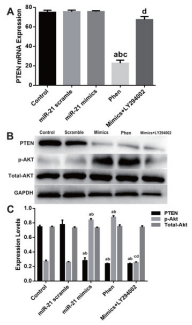
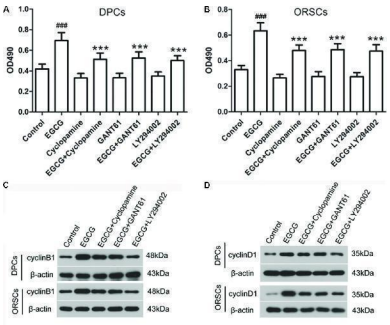
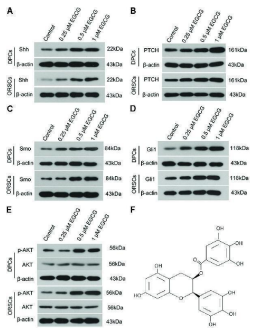
group. When PI3K was inhibited by LY294002, there was notably decrease of BrdU in mimics+LY294002 group compared with mimics group in CSCs. n = 3 in each group. a, P



Expression change of PTEN/PI3K/Akt pathway in the process of miR-21 mimics induced proliferation in c-kit + CSCs. Cultured CSCs were treated with miR-21 mimics for 48 h before the subsequent procedures. To test the contribution of PTEN/PI3K/Akt signaling to miR-21 mimics's pro-proliferation effects in c-kit + CSCs, PTEN and PI3K were inhibited with Phen or LY294002 respectively. (A) RT-PCR was carried out to detect miR-21 mimics's effects on PTEN expression at the mRNA level, which showed no change between Control, miR-21 scramble, miR-21 mimics and miR-21 mimics+ LY294002 group, while Phen resulted in a significant down-regulation of PTEN compared with the other groups. (B–C) Western blot was carried out to detect miR-21 mimics's effects on PTEN protein expression, which showed that miR-21 mimics significantly down-regulated PTEN protein in miR-21 mimics group compared with the scramble group. In addition, both Phen treatment and miR-21 mimics incubation increased p-Akt level, while PI3K inhibitor LY294002 decreased p-Akt level dramatically (P



Shh and AKT signaling pathway inhibitors abolish the effect of EGCG on the growth of DPCs and ORSCs. (A,B) After treatment with EGCG and/or cyclopamine, GANT61 or LY294002, the cell viability of DPCs and ORSCs was assessed by MTT assay. (C,D) Protein levels of cyclinB1 and cyclinD1 in DPCs and ORSCs were assessed by western blot with beta-actin as the internal reference. All experiments were repeated three times. The results are presented as mean ± SD. ### p < 0.001 compared with the control group; ∗∗∗ p < 0.001 compared with the EGCG group.Index in PubMed under a CC BY license. PMID: 29997505



Epigallocatechin-3-gallate activates the Shh and AKT signaling pathways in DPCS and ORSCs. Upon treatment with EGCG, the protein levels of Shh (A) , PTCH (B) , Smo (C) , and Gli1 (D) in DPCs and ORSCs were detected by western blot with beta-actin as the internal reference. (E) Western blot was performed to assess the levels of AKT and p-AKT in each group with beta-actin as the internal reference. (F) Chemical structure of EGCG. Each experiment was repeated three times.Index in PubMed under a CC BY license. PMID: 29997505

Epigallocatechin-3-gallate activates the Shh and AKT signaling pathways in hair follicles. After treatment with 0.5 and 1 uM EGCG, the mRNA levels of Shh (A) , PTCH (B) , Smo (C) , and Gli1 (D) in hair follicles were detected by qRT-PCR. The relative mRNA levels were calculated using 2 -ΔΔC t method. Western blot was also performed to detect the protein levels of Shh (E) , PTCH (F) , Smo (G) , and Gli1 (H) in hair follicles. beta-actin served as the internal reference. (I)

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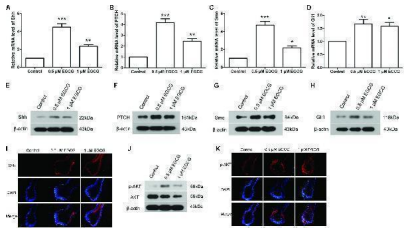
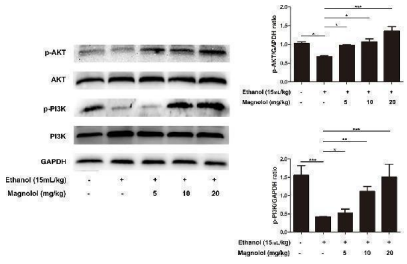


Level of Shh in hair follicles was assessed by immunofluorescence. Red fluorescence: Shh; blue fluorescence: DAPI. (J) The phosphorylation level of AKT was assessed by western blot. (K) Level of p-AKT in hair follicles was assessed by immunofluorescence. Red fluorescence: p-AKT; blue fluorescence: DAPI. Each experiment was repeated three times and the results are presented as mean ± SD. ∗ p < 0.05, ∗∗ p < 0.01, ∗∗∗ p < 0.001 compared with the control group.Index in PubMed under a CC BY license. PMID: 29997505



Effects of magnolol on mice alcohol-induced liver damage in the AKT/PI3K signaling pathway. Liver tissues were extracted for protein analysis by western blotting. AKT and PI3K, proteins expression were detected. The levels of AKT and PI3K were compared with GAPDH. The data were demonstrated as means ± SD. (*P < 0.05, **P < 0.01, ***P < 0.001).Index in PubMed under a CC BY license. PMID: 31920652

14 Publications Citing This Product

1. PubMed ID: 29044143, Li R, Cui K, Liu K, Li H, Zhang Y, Liu X, Chen R, Li M, Wang T, Wang S, Liu J, Rao K. Sci Rep. 2017 Oct 18;7(1):13464. doi: 10.1038/s41598-017-12907-1. Metabolic syndrome in rats is associated with erectile dysfunction by impairing PI3K/Akt/eNOS a...

2. PubMed ID: 27456341, Hyperthermia induced HIF-1a expression of lung cancer through AKT and ERK signaling pathways

3. PubMed ID: 25695729, Wan J, Che Y, Kang N, Wu W. Mol Med Rep. 2015 Jul;12(1):83-92. Doi: 10.3892/Mmr.2015.3368. Epub 2015 Feb 17. Socs3 Blocks Hif-1?? Expression To Inhibit Proliferation And Angiogenesis Of Human Small Cell Lung Cancer By Downregulating Activation Of ...

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