

AKT1/2/3 Ab

Cat.#: AF0791
Size: 100ul,200ul

Concn.: 1mg/ml
Source: Rabbit

Mol.Wt.: 50,56kDa
Clonality: Polyclonal

Application: WB 1:500-1:2000, IF/ICC 1:100-1:500

Reactivity: Human, Mouse, Rat

Purification: The antiserum was purified by peptide affinity chromatography using SulfoLink™ Coupling Resin (Thermo Fisher Scientific).

Specificity: AKT1/2/3 Ab detects endogenous levels of AKT1/2/3.

Immunogen: A synthesized peptide derived from human AKT1/2/3.

Uniprot: P31749/Q9Y243

Description: The serine-threonine protein kinase encoded by the AKT1 gene is catalytically inactive in serum-starved primary and immortalized fibroblasts. AKT1 and the related AKT2 are activated by platelet-derived growth factor. The activation is rapid and specific, and it is abrogated by mutations in the pleckstrin homology domain of AKT1. It was shown that the activation occurs through phosphatidylinositol 3-kinase. In the developing nervous system AKT is a critical mediator of growth factor-induced neuronal survival. Survival factors can suppress apoptosis in a transcription-independent manner by activating the serine/threonine kinase AKT1, which then phosphorylates and inactivates components of the apoptotic machinery. Multiple alternatively spliced transcript variants have been found for this gene.

Subcellular Location: 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 translocates to the nucleus.

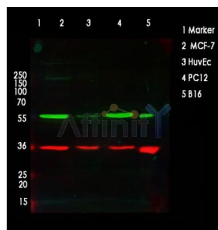
Tissue Specificity: Expressed in prostate cancer and levels increase from the normal to the malignant state (at protein level). Expressed in all human cell types so far analyzed. The Tyr-176 phosphorylated form shows a significant increase in expression in breast cancers during the progressive stages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

Similarity:

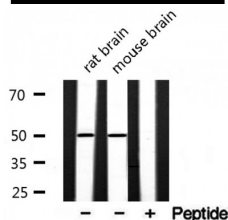
Binding of the PH domain to phosphatidylinositol 3,4,5-trisphosphate (PI(3,4,5)P₃) following phosphatidylinositol 3-kinase alpha (PIK3CA) activity results in its targeting to the plasma membrane. The PH domain mediates interaction with TNK2 and Tyr-176 is also essential for this interaction. The AGC-kinase C-terminal mediates interaction with THEM4. Belongs to the protein kinase superfamily. AGC Ser/Thr protein kinase family. RAC subfamily.

Storage Condition and Buffer:

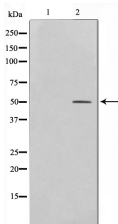
Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at -20 °C. Stable for 12 months from date of receipt.



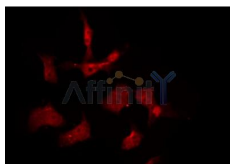
Western blot analysis of AKT1/2/3 using various lysates. Lanes 1 - 2: Merged signal (red and green). Green - AF0791 observed at 55 kDa. Red - loading control, T0004, observed at 36 kDa. Blots were developed with Goat Anti-Rabbit IgG(H+L) FITC-conjugated (S0008) and Goat Anti-Mouse IgG(H+L) Alexa Fluor 594-conjugated (S0005) secondary antibodies.



Western blot analysis of extracts of various samples, using AKT1/2/3 Ab.



Western blot analysis on K562 cell lysate using AKT1/2/3 Ab. The lane on the left is treated with the antigen-specific peptide.



AF0791 staining K562 by IF/ICC. The sample were fixed with PFA and permeabilized in 0.1% Triton X-100, then blocked in 10% serum for 45 minutes at 25°C. The primary Ab was diluted at 1/200 and incubated with the sample for 1 hour at 37°C. An Alexa Fluor 594 conjugated goat anti-rabbit IgG (H+L) Ab, diluted at 1/600, was used as the secondary Ab.

IMPORTANT: For western blot, incubate membrane with diluted primary Ab in 5% w/v milk, 1X TBS, 0.1% Tween@20 at 4°C with gentle shaking,

overnight.

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