

AKT Polyclonal Antibody

catalog number: E-AB-63467

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description

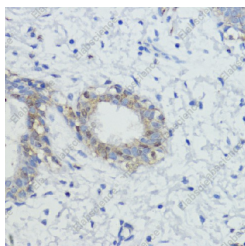
Reactivity	Human;Mouse;Rat
Immunogen	A synthetic peptide of human AKT (NP_005154.2).
Host	Rabbit
Isotype	Rabbit IgG
Purification	Affinity purification
Buffer	Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

Applications

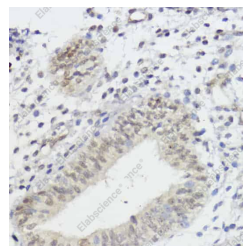
Recommended Dilution

IHC	1:50-1:100
IF	1:50-1:100

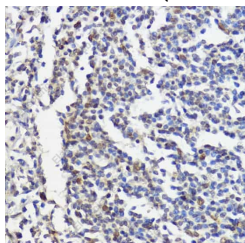
Data



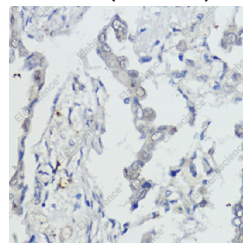
Immunohistochemistry of paraffin-embedded Human mammary cancer using AKT Polyclonal Antibody at dilution of 1:100 (40x lens).



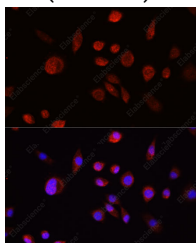
Immunohistochemistry of paraffin-embedded Human uterine cancer using AKT Polyclonal Antibody at dilution of 1:100 (40x lens).



Immunohistochemistry of paraffin-embedded Human tonsil using AKT Polyclonal Antibody at dilution of 1:100 (40x lens).



Immunohistochemistry of paraffin-embedded Human lung cancer using AKT Polyclonal Antibody at dilution of 1:100 (40x lens).



Immunofluorescence analysis of L929 cells using AKT Polyclonal Antibody at dilution of 1:100. Blue: DAPI for nuclear staining.

Preparation & Storage

Storage Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.

For Research Use Only

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Web: www.elabscience.com

Tel: 1-832-243-6086
Email: techsupport@elabscience.com

Fax: 1-832-243-6017

Rev. V1.7

Shipping

The product is shipped with ice pack, upon receipt, store it immediately at the temperature recommended.

Background

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. Mutations in this gene have been associated with the Proteus syndrome. Multiple alternatively spliced transcript variants have been found for this gene.

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