Pan-Akt Polyclonal Antibody

Catalog No. E-AB-30471

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

Description	
Reactivity	Human,Mouse,Rat
Immunogen	Synthesized peptide derived from human Akt around the non-phosphorylation site of Tyr315.
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Conjugation	Unconjugated
Buffer	PBS with 0.02% sodium azide, 0.5% protective protein and 50% glycerol, pH7.4
Applications	Recommended Dilution
WB	1:500-1:2000
IHC	1:100-1:300
IF	1:50-1:200
Data	



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Western Blot analysis of various cells using Pan-Akt Polyclonal Antibody at dilution of 1:1000. Observed Mw:50kDa Calculated Mw:56kDa





Immunohistochemistry of paraffin-embedded Human appendix tissue using Pan-Akt Polyclonal Antibody at dilution of 1:200.

Immunofluorescence analysis of Mouse spleen tissue using Pan-Akt Polyclonal Antibody at dilution of 1:200.

For Research Use Only

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Preparation & Storage

Storage

Store at -20°C. Avoid freeze / thaw cycles.

Background

Plays a role as a 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). General protein kinase capable of phosphorylating several known proteins. Phosphorylates TBC1D4. 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). Plays a role in glucose transport by mediating insulin-induced translocation of the GLUT4 glucose transporter to the cell surface. Mediates the antiapoptotic effects of IGF-I. 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. Promotes glycogen synthesis by mediating the insulin-induced form can suppress FoxO gene transcription and promote cell cycle progression. Essential for the SPATA13-mediated regulation of cell migration and adhesion assembly and disassembly.