

Recombinant Phospho-Akt (Thr308) Monoclonal Antibody

catalog number: AN301330L

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

Description

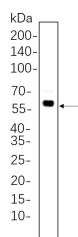
Reactivity	Human;Mouse;Rat;Drosophila
Immunogen	A synthetic peptide corresponding to residues around (Thr308) of Human Phospho-Akt (pan)
Host	Rabbit
Isotype	IgG, κ
Clone	B1093
Purification	Protein A
Buffer	PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protein protectant.

Applications

Recommended Dilution

WB	1:1000-1:5000
IF	1:200-1:1000
ELISA	1:5000-1:20000
IP	1:50-1:200

Data



Western Blot with Recombinant Phospho-Akt (Thr308)
Monoclonal Antibody at dilution of 1:1000 dilution. Lane

A: NIH-3T3 whole cell lysate.

Observed-MW:60 kDa

Calculated-MW:60 kDa

Preparation & Storage

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

Background

For Research Use Only

Toll-free: 1-888-852-8623
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Rev. V1.4

AKT1 gene encodes one of the three members of the human AKT serine-threonine protein kinase family which are often referred to as protein kinase B alpha, beta, and gamma. These highly similar AKT proteins all have an N-terminal pleckstrin homology domain, a serine/threonine-specific kinase domain and a C-terminal regulatory domain. These proteins are phosphorylated by phosphoinositide 3-kinase (PI3K). AKT/PI3K forms a key component of many signalling pathways that involve the binding of membrane-bound ligands such as receptor tyrosine kinases, G-protein coupled receptors, and integrin-linked kinase. These AKT proteins therefore regulate a wide variety of cellular functions including cell proliferation, survival, metabolism, and angiogenesis in both normal and malignant cells. AKT proteins are recruited to the cell membrane by phosphatidylinositol 3,4,5-trisphosphate (PIP3) after phosphorylation of phosphatidylinositol 4,5-bisphosphate (PIP2) by PI3K. Subsequent phosphorylation of both threonine residue 308 and serine residue 473 is required for full activation of the AKT1 protein encoded by this gene. Phosphorylation of additional residues also occurs, for example, in response to insulin growth factor-1 and epidermal growth factor. Protein phosphatases act as negative regulators of AKT proteins by dephosphorylating AKT or PIP3. The PI3K/AKT signalling pathway is crucial for tumor cell survival. Survival factors can suppress apoptosis in a transcription-independent manner by activating AKT1 which then phosphorylates and inactivates components of the apoptotic machinery. AKT proteins also participate in the mammalian target of rapamycin (mTOR) signalling pathway which controls the assembly of the eukaryotic translation initiation factor 4F (eIF4E) complex and this pathway, in addition to responding to extracellular signals from growth factors and cytokines, is dysregulated in many cancers. Mutations in this gene are associated with multiple types of cancer and excessive tissue growth including Proteus syndrome and Cowden syndrome 6, and breast, colorectal, and ovarian cancers. Multiple alternatively spliced transcript variants have been found for this gene. AKT2 gene is a putative oncogene encoding a protein belonging to a subfamily of serine/threonine kinases containing SH2-like (Src homology 2-like) domains, which is involved in signaling pathways. The gene serves as an oncogene in the tumorigenesis of cancer cells. For example, its overexpression contributes to the malignant phenotype of a subset of human ductal pancreatic cancers. The encoded protein is a general protein kinase capable of phosphorylating several known proteins, and has also been implicated in insulin signaling. The protein encoded by AKT3 is a member of the AKT, also called PKB, serine/threonine protein kinase family. AKT kinases are known to be regulators of cell signaling in response to insulin and growth factors. They are involved in a wide variety of biological processes including cell proliferation, differentiation, apoptosis, tumorigenesis, as well as glycogen synthesis and glucose uptake. This kinase has been shown to be stimulated by platelet-derived growth factor (PDGF), insulin, and insulin-like growth factor 1 (IGF1). Alternatively splice transcript variants encoding distinct isoforms have been described.