10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Anti-Akt (N-terminal region) Antibody

Catalog # AN1624

Product Information

Application	WB, ICC
Primary Accession	P31749
Host	Mouse
Clonality	Mouse Monoclonal
Isotype	IgG1
Clone Names	M101
Calculated MW	55686

Additional Information

Gene ID	207
Other Names	PKBalpha, PKB, AKT
Target/Specificity	Akt (PKB, Rac kinase) is a 60kDa ser/thr kinase critical for controlling diverse cellular functions, including glucose metabolism, gene transcription, cell proliferation, and apoptosis. Akt phosphorylates a number of substrates including MBP, glycogen synthetase, PKA RII subunit, and histone H1. Akt is activated in response to insulin and growth factors in a PI3-kinase dependent manner. Activation of PI3-Kinase generates phosphatidylinositol 3,4-bisphosphate, which induces membrane translocation of Akt coincident with its phosphorylation at Thr-308 and Ser-473. Upon activation, Akt associates with members of the PKC family of kinases, such as PKCδ and PKCζ. Ceramide-activated PKCζ leads to phosphorylation of Thr-34 within the pleckstrin homology domain of Akt. This phosphorylation inhibits PIP3 binding to Akt preventing activation of the kinase and may lead to cermide-induced cell death.
Dilution	WB~~1:1000 ICC~~N/A
Format	Protein A Purified
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-Akt (N-terminal region) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

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substrates including MBP, glycogen synthetase, PKA RII subunit, and histone H1. Akt is activated in response to insulin and growth factors in a PI3-kinase dependent manner. Activation of PI3-Kinase generates phosphatidylinositol 3,4-bisphosphate, which induces membrane translocation of Akt coincident with its phosphorylation at Thr-308 and Ser-473. Upon activation, Akt associates with members of the PKC family of kinases, such as PKCδ and PKCζ. Ceramide-activated PKCζ leads to phosphorylation of Thr-34 within the pleckstrin homology domain of Akt. This phosphorylation inhibits PIP3 binding to Akt preventing activation of the kinase and may lead to cermide-induced cell death.