

## **AKT3 Antibody (clone 9H8)**

Mouse Monoclonal Antibody Catalog # ALS16969

## **Specification**

## AKT3 Antibody (clone 9H8) - Product Information

Application IHC, IF, WB
Primary Accession Q9Y243
Other Accession 10000

Reactivity Human, Mouse

Host Mouse
Clonality Monoclonal
Isotype IgG3

## AKT3 Antibody (clone 9H8) - Additional Information

### **Gene ID 10000**

Calculated MW

### **Other Names**

AKT3, AKT3/PKBgamma, PRKBG, PKB gamma, Protein kinase Akt-3, RAC-gamma, MPPH, PKB-GAMMA, PKBG, Protein kinase B gamma, RAC-PK-gamma, STK-2

## Target/Specificity

Human AKT3

## **Reconstitution & Storage**

PBS, pH 7.3, 1% BSA, 50% glycerol, 0.02% sodium azide. Store at -20°C. Minimize freezing and thawing.

55775

### **Precautions**

AKT3 Antibody (clone 9H8) is for research use only and not for use in diagnostic or therapeutic procedures.

# AKT3 Antibody (clone 9H8) - Protein Information

## Name AKT3

## **Synonyms PKBG**

#### **Function**

AKT3 is one of 3 closely related serine/threonine-protein kinases (AKT1, AKT2 and AKT3) called the AKT kinase, and which regulate many processes including metabolism, proliferation, cell survival, growth and angiogenesis. This is mediated through serine and/or threonine phosphorylation of a range of downstream substrates. Over 100 substrate candidates have been reported so far, but for most of them, no isoform specificity has been reported. AKT3 is the least studied AKT isoform. It plays an important role in brain development and is crucial for the viability of malignant glioma cells. AKT3 isoform may also be the key molecule in up-regulation and down-regulation of MMP13



via IL13. Required for the coordination of mitochondrial biogenesis with growth factor-induced increases in cellular energy demands. Down- regulation by RNA interference reduces the expression of the phosphorylated form of BAD, resulting in the induction of caspase- dependent apoptosis.

### **Cellular Location**

Nucleus. Cytoplasm. Membrane; Peripheral membrane protein Note=Membrane-associated after cell stimulation leading to its translocation

### **Tissue Location**

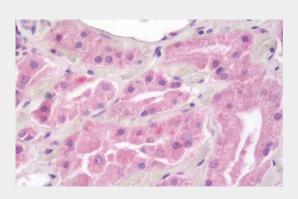
In adult tissues, it is highly expressed in brain, lung and kidney, but weakly in heart, testis and liver. In fetal tissues, it is highly expressed in heart, liver and brain and not at all in kidney

## AKT3 Antibody (clone 9H8) - Protocols

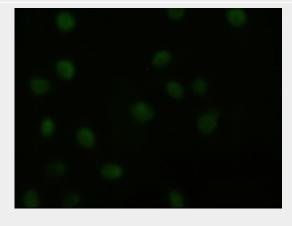
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

# AKT3 Antibody (clone 9H8) - Images

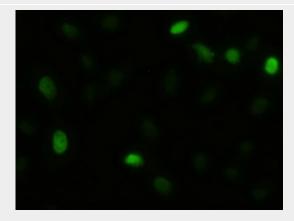


Anti-AKT3 antibody IHC staining of human kidney.

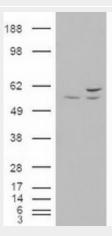




Immunofluorescent staining of HeLa cells using anti-AKT3 mouse monoclonal antibody.



Anti-AKT3 mouse monoclonal antibody immunofluorescent staining of HeLa cells transiently...



HEK293T cells were transfected with the pCMV6-ENTRY control (Left lane) or pCMV6-ENTRY AKT3...

## AKT3 Antibody (clone 9H8) - Background

AKT3 is one of 3 closely related serine/threonine- protein kinases (AKT1, AKT2 and AKT3) called the AKT kinase, and which regulate many processes including metabolism, proliferation, cell survival, growth and angiogenesis. This is mediated through serine and/or threonine phosphorylation of a range of downstream substrates. Over 100 substrate candidates have been reported so far, but for most of them, no isoform specificity has been reported. AKT3 is the least studied AKT isoform. It plays an important role in brain development and is crucial for the viability of malignant glioma cells. AKT3 isoform may also be the key molecule in up-regulation and down-regulation of MMP13 via IL13. Required for the coordination of mitochondrial biogenesis with growth factor-induced increases in cellular energy demands. Down-regulation by RNA interference reduces the expression of the phosphorylated form of BAD, resulting in the induction of caspase-dependent apoptosis.

## AKT3 Antibody (clone 9H8) - References

Brodbeck D., et al.J. Biol. Chem. 274:9133-9136(1999).

Nakatani K., et al.Biochem. Biophys. Res. Commun. 257:906-910(1999).

Masure S., et al.Eur. J. Biochem. 265:353-360(1999).

Li X., et al.Submitted (AUG-1998) to the EMBL/GenBank/DDBJ databases.

Wiemann S., et al.Genome Res. 11:422-435(2001).