

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide

Synthetic peptide Catalog # BP8120a

Specification

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide - Product Information

Primary Accession P49840

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide - Additional Information

Gene ID 2931

Other Names

Glycogen synthase kinase-3 alpha, GSK-3 alpha, Serine/threonine-protein kinase GSK3A, GSK3A

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP8120a was selected from the N-term region of human GSK3A . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide - Protein Information

Name GSK3A

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide - Background

Glycogen synthase kinase 3-alpha (GSK3A)is a multifunctional protein serine kinase implicated in the control of several regulatory proteins including glycogen synthase and transcription factors. It also plays a role in the WNT and PI3K signaling pathways. Under resting conditions GSK3A and its homologs are highly phosphorylated at tyr279 in the phosphorylation loop. Constitutive phosphorylation of this tyrosine is important for kinase activity. Dephosphorylation of tyr279 after mitogen activation is accompanied by kinase inactivation. PKA as well as PI3K-activated PKB inactivate GSK3A by phosphorylation at ser21. Lysophosphatidic acid primarily utilizes a PKC-dependent pathway to modulate GSK3 and certain growth factors (e.g., PDGFB), which control GSK3 mainly through PIK3-PKB, are able to regulate GSK3 through an alternative, redundant PKC pathway. In mice expressing familial AD-associated mutations in APP and PSEN1. lithium reduced the levels of beta-amyloid peptides GSK3A also phosphorylates the tau protein, the principal component of neurofibrillary tangles in AD, and suggested that inhibition of GSK3A may offer a new therapeutic approach to AD.

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide - References

Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002).



Function

Constitutively active protein kinase that acts as a negative regulator in the hormonal control of glucose homeostasis, Wnt signaling and regulation of transcription factors and microtubules, by phosphorylating and inactivating glycogen synthase (GYS1 or GYS2),

CTNNB1/beta-catenin, APC and AXIN1 (PubMed:<a href="http://www.uniprot.org/c itations/11749387"

target=" blank">11749387,

PubMed:<a href="http://www.uniprot.org/ci tations/17478001"

target=" blank">17478001,

PubMed:<a href="http://www.uniprot.org/ci tations/19366350"

target=" blank">19366350). Requires primed phosphorylation of the majority of its substrates (PubMed:<a href="http://ww w.uniprot.org/citations/11749387"

target=" blank">11749387,

PubMed:<a href="http://www.uniprot.org/ci tations/17478001"

target=" blank">17478001,

PubMed: tations/19366350"

target=" blank">19366350).

Contributes to insulin regulation of glycogen synthesis by phosphorylating and inhibiting GYS1 activity and hence glycogen synthesis (PubMed:<a href="http://www.uniprot.org/c itations/11749387"

target=" blank">11749387,

PubMed:<a href="http://www.uniprot.org/ci tations/17478001"

target=" blank">17478001,

PubMed:<a href="http://www.uniprot.org/ci tations/19366350"

target=" blank">19366350).

Regulates glycogen metabolism in liver, but not in muscle (By similarity). May also mediate the development of insulin resistance by regulating activation of transcription factors (PubMed:<a href="http ://www.uniprot.org/citations/10868943"

target="_blank">10868943,

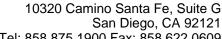
PubMed:<a href="http://www.uniprot.org/ci tations/17478001"

target=" blank">17478001). In Wnt signaling, regulates the level and transcriptional activity of nuclear CTNNB1/beta-catenin (PubMed:<a href="ht"

tp://www.uniprot.org/citations/17229088"

target=" blank">17229088).

Facilitates amyloid precursor protein (APP) processing and the generation of





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APP-derived amyloid plagues found in Alzheimer disease (PubMed:12761548). May be involved in the regulation of replication in pancreatic beta-cells (By similarity). Is necessary for the establishment of neuronal polarity and axon outgrowth (By similarity). Through phosphorylation of the anti-apoptotic protein MCL1, may control cell apoptosis in response to growth factors deprivation (By similarity). Acts as a regulator of autophagy by mediating phosphorylation of KAT5/TIP60 under starvation conditions, leading to activate KAT5/TIP60 acetyltransferase activity and promote acetylation of key autophagy regulators, such as ULK1 and RUBCNL/Pacer (PubMed:30704899). Negatively regulates extrinsic apoptotic signaling pathway via death domain receptors. Promotes the formation of an anti- apoptotic complex, made of DDX3X, BRIC2 and GSK3B, at death receptors, including TNFRSF10B. The anti-apoptotic function is most effective with weak apoptotic signals and can be overcome by stronger stimulation (By similarity).

GSK3 alpha (GSK3A) Antibody (N-term) Blocking peptide - Protocols

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

• Blocking Peptides