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GSK3A Blocking Peptide (Center)

Synthetic peptide
Catalog # BP21095a

Specification

GSK3A Blocking Peptide (Center) - Product Information

Primary Accession	P49840
Other Accession	Q91757, P18266, Q9WV60, P49841, P18265, Q2NL51

GSK3A Blocking Peptide (Center) - 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 is selected from aa 348-362 of HUMAN GSK3A

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.

GSK3A Blocking Peptide (Center) - Protein Information

Name GSK3A

Function
Constitutively active protein kinase that acts as a negative regulator in the

GSK3A Blocking Peptide (Center) - Background

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. Requires primed phosphorylation of the majority of its substrates. Contributes to insulin regulation of glycogen synthesis by phosphorylating and inhibiting GYS1 activity and hence glycogen synthesis. Regulates glycogen metabolism in liver, but not in muscle. May also mediate the development of insulin resistance by regulating activation of transcription factors. In Wnt signaling, regulates the level and transcriptional activity of nuclear CTNNB1/beta-catenin. Facilitates amyloid precursor protein (APP) processing and the generation of APP-derived amyloid plaques found in Alzheimer disease. May be involved in the regulation of replication in pancreatic beta-cells. Is necessary for the establishment of neuronal polarity and axon outgrowth. Through phosphorylation of the anti-apoptotic protein MCL1, may control cell apoptosis in response to growth factors deprivation.

GSK3A Blocking Peptide (Center) - References

He X.,et al.Submitted (MAR-1995) to the EMBL/GenBank/DDBJ databases.
Hoshino T.,et al.Submitted (NOV-1997) to the EMBL/GenBank/DDBJ databases.
Grimwood J.,et al.Nature 428:529-535(2004).
Nikoulina S.E.,et al.Diabetes 49:263-271(2000).
Phiel C.J.,et al.Nature 423:435-439(2003).

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:11749387, PubMed:17478001, PubMed:19366350). Requires primed phosphorylation of the majority of its substrates (PubMed:11749387, PubMed:17478001, PubMed:19366350). Contributes to insulin regulation of glycogen synthesis by phosphorylating and inhibiting GYS1 activity and hence glycogen synthesis (PubMed:11749387, PubMed:17478001, PubMed: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:10868943, PubMed:17478001). In Wnt signaling, regulates the level and transcriptional activity of nuclear CTNNB1/beta-catenin (PubMed:17229088). Facilitates amyloid precursor protein (APP) processing and the generation of APP-derived amyloid plaques found in Alzheimer disease (PubMed:<a href="http://www.uniprot.org/citations/12761548"

target="_blank">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).

GSK3A Blocking Peptide (Center) - Protocols

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

- Blocking Peptides