

**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 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/citations/11749387" target="\_blank">11749387</a>, PubMed:<a href="http://www.uniprot.org/citations/17478001" target="\_blank">17478001</a>, PubMed:<a href="http://www.uniprot.org/citations/19366350" target="\_blank">19366350</a>). Requires primed phosphorylation of the majority of its substrates (PubMed:<a href="http://www.uniprot.org/citations/11749387" target="\_blank">11749387</a>, PubMed:<a href="http://www.uniprot.org/citations/17478001" target="\_blank">17478001</a>, PubMed:<a href="http://www.uniprot.org/citations/19366350" target="\_blank">19366350</a>). Contributes to insulin regulation of glycogen synthesis by phosphorylating and inhibiting GYS1 activity and hence glycogen synthesis (PubMed:<a href="http://www.uniprot.org/citations/11749387" target="\_blank">11749387</a>, PubMed:<a href="http://www.uniprot.org/citations/17478001" target="\_blank">17478001</a>, PubMed:<a href="http://www.uniprot.org/citations/19366350" target="\_blank">19366350</a>).

[17478001](http://www.uniprot.org/citations/17478001), PubMed: [19366350](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/10868943), PubMed: [17478001](http://www.uniprot.org/citations/17478001)). In Wnt signaling, regulates the level and transcriptional activity of nuclear CTNNB1/beta-catenin (PubMed: [17229088](http://www.uniprot.org/citations/17229088)). Facilitates amyloid precursor protein (APP) processing and the generation of APP-derived amyloid plaques found in Alzheimer disease (PubMed: [12761548](http://www.uniprot.org/citations/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 which activates KAT5/TIP60 acetyltransferase activity and promotes acetylation of key autophagy regulators, such as ULK1 and RUBCNL/Pacer (PubMed: [30704899](http://www.uniprot.org/citations/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). Phosphorylates mTORC2 complex component RICTOR at 'Thr- 1695' which facilitates FBXW7-mediated ubiquitination and subsequent degradation of RICTOR (PubMed: [25897075](http://www.uniprot.org/citations/25897075)).

## **GSK3A Blocking Peptide (Center) - Protocols**

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

- [Blocking Peptides](#)

## **GSK3A Blocking Peptide (Center) - Images**

## **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).