

Phospho-GSK3b-S9 Antibody Blocking Peptide
Synthetic peptide
Catalog # BP3112a**Specification**

Phospho-GSK3b-S9 Antibody Blocking Peptide - Product InformationPrimary Accession [P49841](#)**Phospho-GSK3b-S9 Antibody Blocking Peptide - Additional Information****Gene ID** 2932**Other Names**

Glycogen synthase kinase-3 beta, GSK-3 beta, Serine/threonine-protein kinase GSK3B, GSK3B

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP3112a](/product/products/AP3112a) was selected from the 5-13 region of human Phospho-GSK3b-S9. 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.

Phospho-GSK3b-S9 Antibody Blocking Peptide - Protein Information**Name** GSK3B ([HGNC:4617](#))**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), EIF2B, CTNNB1/beta-catenin, APC, AXIN1, DPYSL2/CRMP2, JUN, NFATC1/NFATC, MAPT/TAU and MACF1 (PubMed: [1846781](http://www.uniprot.org/citations/1846781), PubMed: [9072970](http://www.uniprot.org/citations/9072970), PubMed: [14690523](http://www.uniprot.org/citations/14690523), PubMed: [20937854](http://www.uniprot.org/citations/20937854), PubMed: [12554650](http://www.uniprot.org/citations/12554650), PubMed: [11430833](http://www.uniprot.org/citations/11430833), PubMed: [16484495](http://www.uniprot.org/citations/16484495)). Requires primed phosphorylation of the majority of its substrates (PubMed: [16484495](#)).

href="http://www.uniprot.org/citations/11430833" target="_blank">11430833, PubMed:16484495). In skeletal muscle, contributes to insulin regulation of glycogen synthesis by phosphorylating and inhibiting GYS1 activity and hence glycogen synthesis (PubMed:8397507). May also mediate the development of insulin resistance by regulating activation of transcription factors (PubMed:8397507). Regulates protein synthesis by controlling the activity of initiation factor 2B (EIF2BE/EIF2B5) in the same manner as glycogen synthase (PubMed:8397507). In Wnt signaling, GSK3B forms a multimeric complex with APC, AXIN1 and CTNNB1/beta-catenin and phosphorylates the N-terminus of CTNNB1 leading to its degradation mediated by ubiquitin/proteasomes (PubMed:12554650). Phosphorylates JUN at sites proximal to its DNA-binding domain, thereby reducing its affinity for DNA (PubMed:1846781). Phosphorylates NFATC1/NFATC on conserved serine residues promoting NFATC1/NFATC nuclear export, shutting off NFATC1/NFATC gene regulation, and thereby opposing the action of calcineurin (PubMed:9072970). Phosphorylates MAPT/TAU on 'Thr-548', decreasing significantly MAPT/TAU ability to bind and stabilize microtubules (PubMed:14690523). MAPT/TAU is the principal component of neurofibrillary tangles in Alzheimer disease (PubMed:14690523). Plays an important role in ERBB2-dependent stabilization of microtubules at the cell cortex (PubMed:20937854). Phosphorylates MACF1, inhibiting its binding to microtubules which is critical for its role in bulge stem cell migration and skin wound repair (By similarity). Probably regulates NF-kappa-B (NFKB1) at the transcriptional level and is required for the NF-kappa-B-mediated anti- apoptotic response to TNF-alpha (TNF/TNFA) (By similarity). Negatively regulates replication in pancreatic beta-cells, resulting in apoptosis, loss of beta-cells and diabetes (By similarity). Through phosphorylation of the anti-apoptotic protein MCL1, may control cell apoptosis in response to growth factors deprivation (By similarity). Phosphorylates MUC1 in breast cancer cells, decreasing the interaction of MUC1 with CTNNB1/beta-catenin (PubMed:9819408). Is necessary for the establishment of neuronal polarity and axon outgrowth (PubMed:20067585). Phosphorylates MARK2, leading to inhibition of its activity (By similarity). Phosphorylates SIK1 at 'Thr-182', leading to sustainment of its activity (PubMed:18348280). Phosphorylates ZC3HAV1 which enhances its antiviral activity (PubMed:22514281). Phosphorylates SNAI1, leading to its BTRC-triggered ubiquitination and proteasomal degradation (PubMed:15448698, PubMed:15647282). Phosphorylates SFPQ at 'Thr-687' upon T-cell activation (PubMed:20932480). Phosphorylates NR1D1 st 'Ser-55' and 'Ser-59' and stabilizes it by protecting it from proteasomal degradation. Regulates the circadian clock via phosphorylation of the major clock components including BMAL1, CLOCK and PER2 (PubMed:19946213, PubMed:28903391). Phosphorylates FBXL2 at 'Thr-404' and primes it for ubiquitination by the SCF(FBXO3) complex and proteasomal degradation (By similarity). Phosphorylates CLOCK AT 'Ser-427' and targets it for proteasomal degradation (PubMed:19946213). Phosphorylates BMAL1 at 'Ser-17' and 'Ser-21' and primes it for ubiquitination and proteasomal degradation (PubMed:28903391). Phosphorylates OGT at 'Ser-3' or 'Ser-4' which positively

regulates its activity. Phosphorylates MYCN in neuroblastoma cells which may promote its degradation (PubMed:24391509). Regulates the circadian rhythmicity of hippocampal long-term potentiation and BMAL1 and PER2 expression (By similarity). Acts as a regulator of autophagy by mediating phosphorylation of KAT5/TIP60 under starvation conditions, activating KAT5/TIP60 acetyltransferase activity and promoting 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 (PubMed:18846110). Phosphorylates E2F1, promoting the interaction between E2F1 and USP11, stabilizing E2F1 and promoting its activity (PubMed:17050006, PubMed:28992046). Phosphorylates mTORC2 complex component RICTOR at 'Thr-1695' which facilitates FBXW7-mediated ubiquitination and subsequent degradation of RICTOR (PubMed:25897075). Phosphorylates FXR1, promoting FXR1 ubiquitination by the SCF(FBXO4) complex and FXR1 degradation by the proteasome (By similarity). Phosphorylates interleukin-22 receptor subunit IL22RA1, preventing its proteasomal degradation (By similarity).

Cellular Location

Cytoplasm. Nucleus. Cell membrane. Note=The phosphorylated form shows localization to cytoplasm and cell membrane (PubMed:20937854). The MEMO1-RHOA-DIAPH1 signaling pathway controls localization of the phosphorylated form to the cell membrane (PubMed:20937854)

Tissue Location

Expressed in testis, thymus, prostate and ovary and weakly expressed in lung, brain and kidney. Colocalizes with EIF2AK2/PKR and TAU in the Alzheimer disease (AD) brain

Phospho-GSK3b-S9 Antibody Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

Phospho-GSK3b-S9 Antibody Blocking Peptide - Images

Phospho-GSK3b-S9 Antibody Blocking Peptide - Background

Glycogen synthase kinase-3 (GSK3) is a proline-directed serine-threonine kinase that was initially identified as a phosphorylating and inactivating glycogen synthase. Two isoforms, alpha and beta, show a high degree of amino acid homology. GSK3B is involved in energy metabolism, neuronal cell development, and body pattern formation.

Phospho-GSK3b-S9 Antibody Blocking Peptide - References

Wang, L., et al., J. Biol. Chem. 279(31):32444-32452 (2004). Yuan, Z., et al., J. Biol. Chem. 279(25):26105-26114 (2004). Liao, X., et al., Endocrinology 145(6):2941-2949 (2004). Salas, T.R., et al., J. Biol. Chem. 279(18):19191-19200 (2004). Takahashi-Yanaga, F., et al., Biochem. Biophys. Res. Commun. 316(2):411-415 (2004).