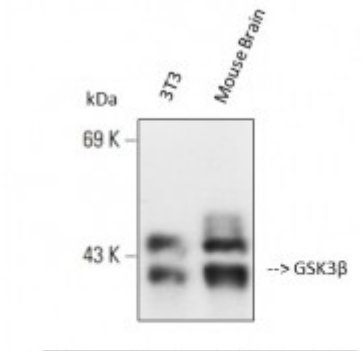
Anti-GSK beta antibody



Western Blot (WB) analysis of 3T3 and mouse brain cells using GSK3β Antibody (STJ93447).

Description	GSK3beta is a protein encoded by the GSK3B gene which is approximately 46,7 kDa. GSK3beta is localised to the cytoplasm, nucleus and cell membrane. It is involved in common cytokine receptor gamma-chain family signalling pathways, RET signalling, regulation of lipid metabolism and insulin signalling-generic cascades. It is a constitutively active protein kinase that acts as a negative regulator in the hormonal control of glucose homeostasis, Wnt signalling and regulation of transcription factors and microtubules. In skeletal muscle, it contributes to insulin regulation of glycogen synthesis. GSK3beta is expressed in the testis, thymus, prostate and ovary and weakly expressed in lung, brain and kidney. Mutations in the GSK3B gene may result in Alzheimer disease and Usher syndrome. STJ93447 was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. This polyclonal antibody detects endogenous levels of GSK3beta protein.

Model	STJ93447
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IHC, IP, WB
Immunogen	Synthesized peptide derived from human GSK3beta around the non-phosphorylation site of S9.
Immunogen Region	1-80 aa
Gene ID	2932
Gene Symbol	GSK3B
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IP 1:200-500ELISA 1:20000

Specificity	GSK3beta Polyclonal Antibody detects endogenous levels of GSK3beta protein.
Tissue Specificity	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.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Glycogen synthase kinase-3 beta GSK-3 beta Serine/threonine-protein kinase GSK3B
Molecular Weight	47 kDa
Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:4617OMIM:605004
Alternative Names	Glycogen synthase kinase-3 beta GSK-3 beta Serine/threonine-protein kinase GSK3B
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. Requires primed phosphorylation of the majority of its substrates. In skeletal muscle, contributes to insulin regulation of glycogen synthesis by phosphorylating and inhibiting GYS1 activity and hence glycogen synthesis. May also mediate the development of insulin resistance by regulating activation of transcription factors. Regulates protein synthesis by controlling the activity of initiation factor 2B (EIF2BE/EIF2B5) in the same manner as glycogen synthase. 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. Phosphorylates JUN at sites proximal to its DNA-binding domain, thereby reducing its affinity for DNA. 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. Phosphorylates MAPT/TAU on 'Thr-548', decreasing significantly MAPT/TAU ability to bind and stabilize microtubules. MAPT/TAU is the principal component of neurofibrillary tangles in Alzheimer disease. Plays an important role in ERBB2-dependent stabilization of microtubules at the cell cortex. Phosphorylates MACF1, inhibiting its binding to microtubules which is critical for its role in bulge stem cell migration and skin wound repair. 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). Negatively regulates replication in pancreatic beta-cells, resulting in apoptosis, loss of beta-cells and diabetes. Through phosphorylation of the anti-apoptotic protein MCL1, may control cell apoptosis in response to growth factors deprivation. Phosphorylates MUC1 in breast cancer cells, decreasing the interaction of MUC1 with CTNNB1/beta-catenin. Is necessary for the establishment of neuronal polarity and axon outgrowth. Phosphorylates MARK2, leading to inhibit its activity. Phosphorylates SIK1 at 'Thr-182', leading to sustain its activity. Phosphorylates ZC3HAV1 which enhances its antiviral activity. Phosphorylates SNAI1, leading to its BTRC-triggered ubiquitination and proteasomal degradation. Phosphorylates SFPQ at 'Thr-687' upon T-cell activation. 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 ARNTL/BMAL1, CLOCK and PER2. Phosphorylates CLOCK AT 'Ser-427' and targets it for proteasomal degradation. Phosphorylates ARNTL/BMAL1 at 'Ser-17' and 'Ser-21' and primes it for ubiquitination and proteasomal degradation. Phosphorylates OGT at 'Ser-3' or 'Ser-4' which positively regulates its activity. Phosphorylates MYCN in neuroblastoma cells which may promote its degradation .
Cellular Localization	Cytoplasm Nucleus. Cell membrane. The phosphorylated form shows localization to cytoplasm and cell membrane. The MEMO1-RHOA-DIAPH1 signaling pathway controls localization of the phosphorylated form to the cell membrane.
Post-translational Modifications	Phosphorylated by AKT1 and ILK1. Upon insulin-mediated signaling, the activated PKB/AKT1 protein kinase phosphorylates and desactivates GSK3B, resulting in the dephosphorylation and activation of GYS1. Activated by phosphorylation at Tyr-216 . Inactivated by phosphorylation at Ser-9 (Probable). Mono-ADP-ribosylation by PARP10 negatively regulates kinase activity.