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GSK3A Antibody (S21)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8120c

Product Information

Application	WB, E
Primary Accession	P49840
Other Accession	P18265, Q2NL51
Reactivity	Human
Predicted	Mouse, Rat, Bovine, Canine
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB21545
Calculated MW	50981

Additional Information

Gene ID	2931
Other Names	Glycogen synthase kinase-3 alpha, GSK-3 alpha, Serine/threonine-protein kinase GSK3A, GSK3A
Target/Specificity	This GSK3A antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide selected from amino acid residues surrounding S21 of human GSK3A.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	GSK3A Antibody (S21) is for research use only and not for use in diagnostic or therapeutic procedures.

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: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: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). 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).

Background

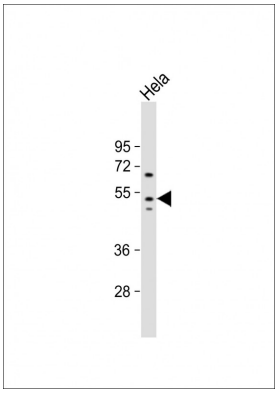
Glycogen synthase kinase 3-alpha (GSK3A)is a multifunctional protein serine kinase implicated in the control of several regulatory proteins including glycogen synthase and transcription factors. It also plays a role in the WNT and PI3K signaling pathways. Under resting conditions GSK3A and its homologs 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.

References

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

Images

Anti-GSK3A Antibody (S21) at 1:1000 dilution + Hela whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 51 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



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