

GSK3α (phospho Ser21) Polyclonal Antibody

Catalog # AP67054

Product Information

Application	WB, IHC-P, IF, ICC, IP, E
Primary Accession	P49840
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	50981

Additional Information

Gene ID	2931
Other Names	GSK3A; Glycogen synthase kinase-3 alpha; GSK-3 alpha; Serine/threonine-protein kinase GSK3A
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunoprecipitation: 2-5 ug/mg lysate. ELISA: 1/20000. Not yet tested in other applications. IHC-P~~N/A IF~~1:50~200 ICC~~N/A IP~~N/A E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20°C

Protein Information

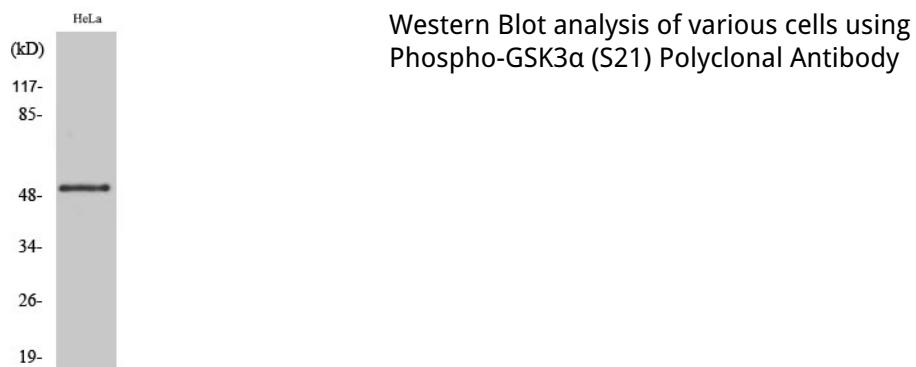
Name	GSK3A
Function	Constitutively active protein kinase that acts as a negative regulator in the hormonal control of glucose homeostasis, Wnt signaling and regulation of transcription factors and microtubules, by phosphorylating and inactivating glycogen synthase (GYS1 or GYS2), CTNNB1/beta-catenin, 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

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.

Images



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