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PLK2 Polyclonal Antibody

Catalog # AP74037

Product Information

Application	IHC-P, IF, ICC, E
Primary Accession	Q9NYY3
Reactivity	Human, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	78237

Additional Information

Gene ID	10769
Other Names	Serine/threonine-protein kinase PLK2 (EC 2.7.11.21) (Polo-like kinase 2) (PLK-2) (hPlk2) (Serine/threonine-protein kinase SNK) (hSNK) (Serum-inducible kinase)
Dilution	IHC-P~~IHC-p 1:50-200, ELISA 1:10000-20000 IF~~1:50~200 ICC~~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℃

Protein Information

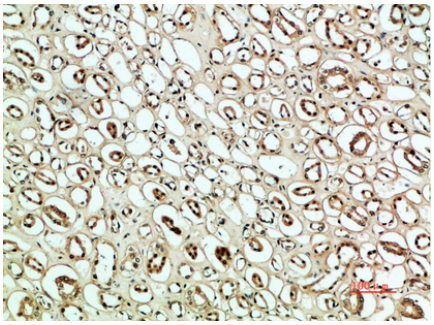
Name	PLK2
Synonyms	SNK
Function	Tumor suppressor serine/threonine-protein kinase involved in synaptic plasticity, centriole duplication and G1/S phase transition. Polo-like kinases act by binding and phosphorylating proteins that are already phosphorylated on a specific motif recognized by the POLO box domains. Phosphorylates CPAP, NPM1, RAPGEF2, RASGRF1, SNCA, SIPA1L1 and SYNGAP1. Plays a key role in synaptic plasticity and memory by regulating the Ras and Rap protein signaling: required for overactivity-dependent spine remodeling by phosphorylating the Ras activator RASGRF1 and the Rap inhibitor SIPA1L1 leading to their degradation by the proteasome. Conversely, phosphorylates the Rap activator RAPGEF2 and the Ras inhibitor SYNGAP1, promoting their activity. Also regulates synaptic plasticity independently of kinase activity, via its interaction with NSF that disrupts the interaction between NSF and the GRIA2 subunit of AMPARs, leading to a rapid rundown of AMPAR-mediated current that occludes long term depression. Required for procentriole formation and centriole duplication by phosphorylating CPAP and NPM1,

	respectively. Its induction by p53/TP53 suggests that it may participate in the mitotic checkpoint following stress.
Cellular Location	Cytoplasm, cytoskeleton, microtubule organizing center, centrosome, centriole. Cell projection, dendrite Note=Localizes to centrosomes during early G1 phase where it only associates to the mother centriole and then distributes equally to both mother and daughter centrioles at the onset of S phase
Tissue Location	Expressed at higher level in the fetal lung, kidney, spleen and heart.

Background

Tumor suppressor serine/threonine-protein kinase involved in synaptic plasticity, centriole duplication and G1/S phase transition. Polo-like kinases act by binding and phosphorylating proteins are that already phosphorylated on a specific motif recognized by the POLO box domains. Phosphorylates CENPJ, NPM1, RAPGEF2, RASGRF1, SNCA, SIPA1L1 and SYNGAP1. Plays a key role in synaptic plasticity and memory by regulating the Ras and Rap protein signaling: required for overactivity-dependent spine remodeling by phosphorylating the Ras activator RASGRF1 and the Rap inhibitor SIPA1L1 leading to their degradation by the proteasome. Conversely, phosphorylates the Rap activator RAPGEF2 and the Ras inhibitor SYNGAP1, promoting their activity. Also regulates synaptic plasticity independently of kinase activity, via its interaction with NSF that disrupts the interaction between NSF and the GRIA2 subunit of AMPARs, leading to a rapid rundown of AMPAR-mediated current that occludes long term depression. Required for procentriole formation and centriole duplication by phosphorylating CENPJ and NPM1, respectively. Its induction by p53/TP53 suggests that it may participate in the mitotic checkpoint following stress.

Images



Immunohistochemical analysis of paraffin-embedded human-kidney, antibody was diluted at 1:200

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