

Anti-CDK5 (pY15) Antibody

Rabbit polyclonal antibody to CDK5 (pY15) Catalog # AP60246

Product Information

Application WB, IF/IC, IHC
Primary Accession Q00535
Other Accession P49615

Reactivity Human, Mouse, Rat, Pig, Chicken, Bovine, SARS

Host Rabbit
Clonality Polyclonal
Calculated MW 33304

Additional Information

Gene ID 1020

Other Names CDKN5; Cyclin-dependent kinase 5; Cell division protein kinase 5;

Serine/threonine-protein kinase PSSALRE; Tau protein kinase II catalytic

subunit; TPKII catalytic subunit

Target/Specificity Recognizes endogenous levels of CDK5 (pY15) protein.

Dilution WB~~WB (1/500 - 1/1000), IHC (1/100 - 1/200), IF/IC (1/100 - 1/500)

IF/IC~~N/A IHC~~WB (1/500 - 1/1000), IHC (1/100 - 1/200), IF/IC (1/100 -

1/500)

Format Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30%

glycerol, and 0.09% (W/V) sodium azide.

Storage Store at -20 °C.Stable for 12 months from date of receipt

Protein Information

Name CDK5 (HGNC:1774)

Function Proline-directed serine/threonine-protein kinase essential for neuronal cell

cycle arrest and differentiation and may be involved in apoptotic cell death in neuronal diseases by triggering abortive cell cycle re-entry. Interacts with D1

and D3-type G1 cyclins. Phosphorylates SRC, NOS3, VIM/vimentin,

p35/CDK5R1, MEF2A, SIPA1L1, SH3GLB1, PXN, PAK1, MCAM/MUC18, SEPT5, SYN1, DNM1, AMPH, SYNJ1, CDK16, RAC1, RHOA, CDC42, TONEBP/NFAT5, MAPT/TAU, MAP1B, histone H1, p53/TP53, HDAC1, APEX1, PTK2/FAK1, huntingtin/HTT, ATM, MAP2, NEFH and NEFM. Regulates several neuronal development and physiological processes including neuronal survival, migration and differentiation, axonal and neurite growth, synaptogenesis, oligodendrocyte differentiation, synaptic plasticity and neurotransmission, by

phosphorylating key proteins. Negatively regulates the CACNA1B/CAV2.2 -mediated Ca(2+) release probability at hippocampal neuronal soma and synaptic terminals (By similarity). Activated by interaction with CDK5R1 (p35) and CDK5R2 (p39), especially in postmitotic neurons, and promotes CDK5R1 (p35) expression in an autostimulation loop. Phosphorylates many downstream substrates such as Rho and Ras family small GTPases (e.g. PAK1, RAC1, RHOA, CDC42) or microtubule-binding proteins (e.g. MAPT/TAU, MAP2, MAP1B), and modulates actin dynamics to regulate neurite growth and/or spine morphogenesis. Also phosphorylates exocytosis associated proteins such as MCAM/MUC18, SEPT5, SYN1, and CDK16/PCTAIRE1 as well as endocytosis associated proteins such as DNM1, AMPH and SYNJ1 at synaptic terminals. In the mature central nervous system (CNS), regulates neurotransmitter movements by phosphorylating substrates associated with neurotransmitter release and synapse plasticity; synaptic vesicle exocytosis, vesicles fusion with the presynaptic membrane, and endocytosis. Promotes cell survival by activating anti- apoptotic proteins BCL2 and STAT3, and negatively regulating of JNK3/MAPK10 activity. Phosphorylation of p53/TP53 in response to genotoxic and oxidative stresses enhances its stabilization by preventing ubiquitin ligase-mediated proteasomal degradation, and induces transactivation of p53/TP53 target genes, thus regulating apoptosis. Phosphorylation of p35/CDK5R1 enhances its stabilization by preventing calpain-mediated proteolysis producing p25/CDK5R1 and avoiding ubiquitin ligase-mediated proteasomal degradation. During aberrant cell-cycle activity and DNA damage, p25/CDK5 activity elicits cell-cycle activity and double-strand DNA breaks that precedes neuronal death by deregulating HDAC1. DNA damage triggered phosphorylation of huntingtin/HTT in nuclei of neurons protects neurons against polyglutamine expansion as well as DNA damage mediated toxicity. Phosphorylation of PXN reduces its interaction with PTK2/FAK1 in matrix-cell focal adhesions (MCFA) during oligodendrocytes (OLs) differentiation. Negative regulator of Wnt/beta-catenin signaling pathway. Activator of the GAIT (IFN-gamma-activated inhibitor of translation) pathway, which suppresses expression of a post-transcriptional regulon of proinflammatory genes in myeloid cells; phosphorylates the linker domain of glutamyl-prolyl tRNA synthetase (EPRS) in a IFN-gamma-dependent manner, the initial event in assembly of the GAIT complex. Phosphorylation of SH3GLB1 is required for autophagy induction in starved neurons. Phosphorylation of TONEBP/NFAT5 in response to osmotic stress mediates its rapid nuclear localization. MEF2 is inactivated by phosphorylation in nucleus in response to neurotoxin, thus leading to neuronal apoptosis. APEX1 APendodeoxyribonuclease is repressed by phosphorylation, resulting in accumulation of DNA damage and contributing to neuronal death. NOS3 phosphorylation down regulates NOS3-derived nitrite (NO) levels. SRC phosphorylation mediates its ubiquitin-dependent degradation and thus leads to cytoskeletal reorganization. May regulate endothelial cell migration and angiogenesis via the modulation of lamellipodia formation. Involved in dendritic spine morphogenesis by mediating the EFNA1-EPHA4 signaling. The complex p35/CDK5 participates in the regulation of the circadian clock by modulating the function of CLOCK protein: phosphorylates CLOCK at 'Thr-451' and 'Thr-461' and regulates the transcriptional activity of the CLOCK-BMAL1 heterodimer in association with altered stability and subcellular distribution.

Cellular Location

[Isoform 1]: Cytoplasm. Nucleus Cell membrane; Peripheral membrane protein. Perikaryon. Cell projection, lamellipodium {ECO:0000250 | UniProtKB:P49615}. Cell projection, growth cone {ECO:0000250 | UniProtKB:P49615}. Postsynaptic density {ECO:0000250 | UniProtKB:Q03114}. Synapse {ECO:0000250 | UniProtKB:Q03114} Note=In axonal growth cone with extension to the peripheral lamellipodia (By similarity). Under neurotoxic stress and neuronal injury conditions, CDK5R (p35) is cleaved by calpain to generate CDK5R1 (p25) in response to increased intracellular calcium. The

elevated level of p25, when in complex with CDK5, leads to its subcellular misallocation as well as its hyperactivation. Colocalizes with CTNND2 in the cell body of neuronal cells, and with CTNNB1 in the cell-cell contacts and plasma membrane of undifferentiated and differentiated neuroblastoma cells. Reversibly attached to the plasma membrane in an inactive form when complexed to dephosphorylated p35 or CDK5R2 (p39), p35 phosphorylation releases this attachment and activates CDK5

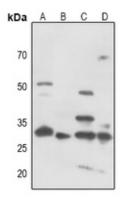
Tissue Location

[Isoform 1]: Ubiquitously expressed (PubMed:17009320, PubMed:19693690). Accumulates in cortical neurons (at protein level) (PubMed:17009320).

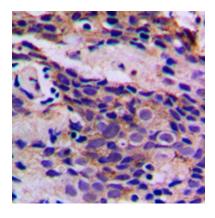
Background

KLH-conjugated synthetic peptide encompassing a sequence within the N-term region of human CDK5 (pY15). The exact sequence is proprietary.

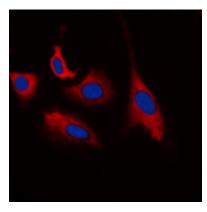
Images



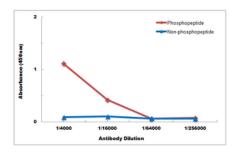
Western blot analysis of CDK5 (pY15) expression in HEK293T (A), mouse muscle (B), mouse lung (C), rat muscle (D) whole cell lysates.



Immunohistochemical analysis of CDK5 (pY15) staining in human breast cancer formalin fixed paraffin embedded tissue section. The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH 6.0). The section was then incubated with the antibody at room temperature and detected using an HRP conjugated compact polymer system. DAB was used as the chromogen. The section was then counterstained with haematoxylin and mounted with DPX.



Immunofluorescent analysis of CDK5 (pY15) staining in HEK293T cells. Formalin-fixed cells were permeabilized with 0.1% Triton X-100 in TBS for 5-10 minutes and blocked with 3% BSA-PBS for 30 minutes at room temperature. Cells were probed with the primary antibody in 3% BSA-PBS and incubated overnight at 4 °C in a hidified chamber. Cells were washed with PBST and incubated with a DyLight 594-conjugated secondary antibody (red) in PBS at room temperature in the dark. DAPI was used to stain the cell nuclei (blue).



Direct ELISA antibody dose-response curve using Anti-CDK5 (pY15) Antibody. Antigen (phosphopeptide and non-phosphopeptide) concentration is 5 ug/ml. Goat Anti-Rabbit IgG (H&L) - HRP was used as the secondary antibody, and signal was developed by TMB substrate.

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