Product Information

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Monoclonal Anti-Cdk5
Clone CDK-3G
produced in mouse, purified immunoglobulin

Catalog Number **C6118**

Product Description

Monoclonal Anti-Cdk5 (mouse IgM isotype) is derived from the hybridoma CDK-3G produced by the fusion of mouse myeloma cells (NS1) and splenocytes from BALB/c mice immunized with a synthetic peptide corresponding to amino acids 269-285 of human Cdk5 (Gene ID: 1020), conjugated to KLH. The isotype is determined using a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Catalog Number ISO2.

Monoclonal Anti-Cdk5 specifically recognizes human and mouse Cdk5, ~ 34 kDa. The antibody may be used in ELISA and immunoblotting.

Cdk5 is a serine/threonine kinase with close homology to other Cdks.[1] Cdk5 kinase activity is detected mainly in post-mitotic neurons in the central nervous system (CNS).[2] Association of Cdk5 with a neuron-specific regulatory subunit p35, is critical for this activity.[3-5] The p35/Cdk5 kinase has been implicated in a variety of functions in the CNS, including axon outgrowth, axon guidance and fasciculation (the formation of bundles or fascicles), and proper neuronal migration during cortical development. $Cdk5^{-/-}$ mice exhibit embryonic lethality associated with disruption of the cortical laminar structures in the cerebral cortex, olfactory bulb, hippocampus, and cerebellar cortex.[6] Mice lacking p35 display defects in cortical lamination and fasciculation of axonal fibers.[7-8] Neuronal birth date labeling by BrdU reveals an inverted pattern of cell layers in the cerebral cortex in $Cdk5^{-/-}$ and $p35^{-/-}$ mice. Mutant mice lacking either Cdk5 or p35 exhibit certain similarities with the Reelin/Dab1 (*reeler* and *scrambler/yotari*) mutant mice in the disorganization of cortical laminar structures in the brain. Cdk5/p35 may contribute synergistically with Reelin/Dab1 to the positioning of cortical neurons in the developing mouse brain.[9] Cdk5/p35 activity is also required in the mature CNS. Phosphorylation of DARPP32 by Cdk5 inhibits protein kinase A signaling and alters the response of striatal neurons to dopamine. Cdk5 also phosphorylates Munc18, which in turn affects synaptic vesicle exocytosis. p35/Cdk5 kinase associates with a β-catenin/N-cadherin adhesion complex in the cortex, and modulates N-cadherin-mediated aggregation in embryonic cortical neurons.[10]

Deregulation of Cdk5 activity contributes to the pathogenesis of neurodegenerative diseases such as Alzheimer's disease (AD). Deregulation of Cdk5 is caused by the accumulation of a truncated fragment of p35 (p25) produced in the brain of patients with AD. p25 causes Cdk5 to be constitutively activated and mislocalized *in vivo*. The p25/Cdk5 kinase hyperphosphorylates tau protein and reduces the ability of tau to bind to microtubules. Moreover, p25/Cdk5 causes morphological degeneration and profound apoptotic cell death of primary neurons, suggesting that the conversion of p35 to p25 is involved in the pathogenesis of AD.[11-12]

Reagent

Supplied as a solution in 0.01M PBS, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody concentration: ~ 2 mg/mL.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For extended storage, freeze at –20 °C in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discard if not used within 12 hours.

Product Profile

Immunoblotting: a working concentration of 2-4 µg/mL is recommended using total cell extracts of mouse brain cytosolic fraction S1.
Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

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