

p39 Antibody

Orders: 877-616-CELL (2355)
orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com
cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene ID:
W	H M R	Endogenous	39-45	Rabbit	#Q13319	8941

Product Usage Information**Application**

Western Blotting

Dilution

1:1000

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

p39 Antibody detects endogenous levels of total p39 protein.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide surrounding Thr90 of human p39. Antibodies are purified by peptide affinity chromatography.

Background

Cyclin-dependent kinases (CDKs) are serine/threonine kinases that are activated by cyclins and govern eukaryotic cell cycle progression. While CDK5 shares high sequence homology with its family members, it is thought mainly to function in postmitotic neurons, regulating the cytoarchitecture of these cells. Analogous to cyclins, p35 and p39 associate with and activate CDK5 despite the lack of sequence homology. CDK5 is ubiquitously expressed, but high levels of kinase activity are detected primarily in the nervous system due to the narrow expression pattern of p35 and p39 in post-mitotic neurons. A large number of CDK5 substrates have been identified although no discrete substrates have been attributed as a function of p35 vs. p39. Amongst many, substrates of CDK5 include p35 and p39. p35 is rapidly degraded (T1/2 <20 min) by the ubiquitin-proteasome pathway (1). However, p35 stability increases as CDK5 kinase activity decreases, and this is likely a result of decreased phosphorylation of p35 at Thr138 by CDK5 (2). NGF activates Erk and EGR1, and induces p35 expression in PC12 cells (3). Proteolytic cleavage of p35 by calpain produces p25 upon neurotoxic insult, resulting in prolonged activation of CDK5 by p25. Accumulation of p25 is found in neurodegenerative diseases such as Alzheimer's disease and Amyotrophic Lateral Sclerosis (ALS) (4-5). CDK5-null mice are perinatal lethal, whereas p35 or p39-null mice are viable. However, p35 and p39 double-null mutants display phenotypes identical to those of the CDK5-null mutant mice (6). Association of p39 but not p35 with CDK5 promotes Munc18-1 phosphorylation and Ca²⁺-dependent exocytosis (7). p39 binds to the actin cytoskeleton associated protein muskelin, and localizes to lamellipodia, filopodia and growth cones of neurons (8,9).

Background References

1. Dhavan, R. and Tsai, L.H. (2001) *Nat. Rev. Mol. Cell Biol.* 2, 749-759.
2. Patrick, G.N. et al. (1998) *J. Biol. Chem.* 273, 24057-24064.
3. Harada, T. et al. (2001) *Nat. Cell Biol.* 3, 453-459.
4. Lee, M.S. et al. (2000) *Nature* 405, 360-364.
5. Kusakawa, G. et al. (2000) *J. Biol. Chem.* 275, 17166-17172.
6. Ko, J. et al. (2001) *J. Neurosci.* 21, 6758-6771.
7. Lilja, L. et al. (2004) *J Biol Chem* 279, 29534-29541.
8. Ledee, D.R. et al. (2005) *J. Biol. Chem.* 280, 21376-21383.
9. Humbert, S. et al. (2000) *J. Cell Sci.* 113 (Pt 6), 975-983.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat

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