N-WASP (30D10) Rabbit mAb



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Applications: W, IP	Reactivity: H M R Hm Mk B	Sensitivity: Endogenous	MW (kDa): 65	Source/Isotype: Rabbit	UniProt ID: #O00401	Entrez-Gene Id: 8976
Product Usage Information	•	Application Western Blotting Immunoprecipitation			Dilution 1:1000 1:50	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		N-WASP (30D10) Rabbit mAb recognizes endogenous levels of total N-WASP protein. The antibody does not cross-react wth the hematopietic protein, WASP.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding the sequence of human N-WASP.				
Background		Wiskott-Aldrich syndrome proteins (WASPs) mediate actin dynamics by activating the Arp2/3 actin nucleation complex in response to activated Rho family GTPases. In mammals, five WASP family members have been described. Hematopoietic WASP and ubiquitously expressed N-WASP are autoinhibited in unstimulated cells. Upon stimulation they are activated by cdc42, which relieves the autoinhibition in conjunction with phosphatidyl inositol 4,5-bisphosphate. Three WAVE (Wasf, SCAR) family proteins are similar in sequence to WASP and N-WASP but lack the WASP/N-WASP autoinhibition domains and are indirectly activated by Rac (reviewed in 1). Both WASP and WAVE functions appear to be essential, as knockout of either N-WASP or Scar-2 in mice results in cardiac and neuronal defects and embryonic lethality (2,3). Loss of WASP results in immune system defects and fewer immune cells (4). WAVE-2 (WASF2) is widely distributed, while WAVE-1 and WAVE-3 are strongly expressed in brain (5). WAVE-3 may act as a tumor suppressor in neuroblastoma, a childhood disease of the sympathetic nervous system (6). Increased expression of WAVE-3 is seen in breast cancer, and studies in breast adenocarcinoma cells indicate that WAVE-3 regulates breast cancer progression, invasion and metastasis through the p38 mitogen-activated protein kinase (MAPK) pathway (7,8).				
Background References		1. Millard, T.H. et al. (2004) <i>Biochem J.</i> 380, 1-17. 2. Yan, C. et al. (2003) <i>EMBO J.</i> 22, 3602-3612. 3. Snapper, S.B. et al. (2001) <i>Nat. Cell Biol.</i> 3, 897-904. 4. Zhang, J. et al. (1999) <i>J. Exp. Med.</i> 190, 1329-4132. 5. Suetsugu, S. et al. (1999) <i>Biochem. Biophys. Res. Commun.</i> 260, 296-302. 6. Sossey-Alaoui, K. et al. (2002) <i>Oncogene</i> 21, 5967-5974. 7. Sossey-Alaoui, K. et al. (2005) <i>Exp. Cell Res.</i> 308, 135-145. 8. Sossey-Alaoui, K. et al. (2007) <i>Am J Pathol</i> 170, 2112-21.				
Species Reacti	vity	Species reactivity is de	etermined by testin	g in at least one approve	ed application (e.g.,	western blot).

Western Blot Buffer

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

Applications Key W: Western Blotting IP: Immunoprecipitation

Cross-Reactivity Key

H: Human M: Mouse R: Rat Hm: Hamster Mk: Monkey B: Bovine

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