## NDRG4 (D4A6) Rabbit mAb



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

<b>Applications:</b> W, IP, IHC-P, IF-F	Reactivity: H M R	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 37-45	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #Q9ULP0	Entrez-Gene Id 65009
Product Usage Information		Application Western Blotting			<b>Dilution</b> 1:1000	
		Immunoprecipitation Immunohistochemist Immunofluorescence	, ,		1:	100 800 200
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.				
		For a carrier free (BSA and azide free) version of this product see product #49334.				
Specificity/Sensitivity		NDRG4 (D4A6) Rabbit mAb recognizes endogenous levels of total NDRG4 protein.				
Species predicted to react based on 100% sequence homology		Monkey				
Source / Purific	ation	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Gly217 of human NDRG4 protein.				
Background		The NDRG (N-Myc downstream-regulated gene) family consisting of NDRG1, NDRG2, NDRG3, and NDRG4 are structurally related proteins with roles in cell proliferation, differentiation, apoptosis, stress responses, and cell migration/metastasis (1-3). NDRG1 was originally identified as a protein that was upregulated in N-Myc knockout mice (1). Proteins in the NDRG family, particularly NDRG1 and NDRG2, have been reported to be down-regulated in various cancer tissues and have been suggested to function as a tumor suppressors (4,5).  Unlike other members of the family that are widely expressed, NDRG4/Bdm1 expression is restricted primarily to the heart and brain (2,3,6,7). Multiple alternatively spliced isoforms of NDRG4 have been reported (3,7). Expression of NDRG4 is reduced in the brain of patients with Alzheimer's disease (3). In contrast, NDRG4 expression is elevated in glioblastoma and contributes to cell cycle progression and survival (8). NDRG4 also may be inactivated by promotor methylation in colorectal cancer and function as a tumor suppressor gene (9).				
Background Re	ferences	1. Shimono, A. et al. (1999) <i>Mech Dev</i> 83, 39-52. 2. Qu, X. et al. (2002) <i>Mol Cell Biochem</i> 229, 35-44. 3. Zhou, R.H. et al. (2001) <i>Genomics</i> 73, 86-97. 4. Yao, L. et al. (2008) <i>Acta Biochim Biophys Sin (Shanghai)</i> 40, 625-35. 5. Ellen, T.P. et al. (2008) <i>Carcinogenesis</i> 29, 2-8. 6. Yamauchi, Y. et al. (1999) <i>Brain Res Mol Brain Res</i> 68, 149-58. 7. Nakada, N. et al. (2002) <i>Brain Res Dev Brain Res</i> 135, 45-53. 8. Schilling, S.H. et al. (2009) <i>J Biol Chem</i> 284, 25160-9. 9. Melotte, V. et al. (2009) <i>J Natl Cancer Inst</i> 101, 916-27.				

**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 **IP**: Immunoprecipitation **IHC-P**: Immunohistochemistry (Paraffin) **IF-F**:

Immunofluorescence (Frozen)

**Cross-Reactivity Key** 

H: Human M: Mouse R: Rat

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