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NDRG Family Antibody Sampler Kit



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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-NDRG1 (Ser330) (D3A12) Rabbit mAb	11899	40 µl	46, 48 kDa	Rabbit
Phospho-NDRG1 (Thr346) (D98G11) XP [®] Rabbit mAb	5482	40 µl	46, 48 kDa	Rabbit IgG
NDRG1 (D8G9) XP [®] Rabbit mAb	9485	40 µl	46, 48 kDa	Rabbit IgG
NDRG2 Antibody	5667	40 µl	45 kDa	Rabbit
NDRG3 Antibody	5846	40 µl	45 kDa	Rabbit
NDRG4 (D4A6) Rabbit mAb	9039	40 µl	37-45 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description	The NDRG Family Antibody Sampler Kit provides an economical means of detecting members of the NDRG protein family. The kit includes enough antibody to perform four western blot experiments per primary antibody.
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.
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). Ubiquitously expressed N-Myc downstream-regulated gene 1 (NDRG1) is highly responsive to a variety of stress signals, including DNA damage, hypoxia, and elevated levels of nickel and calcium (6-9). Expression of NDRG1 is elevated in N-Myc defective mice and is negatively regulated by N- and c-Myc (1,10). Expression of NDRG1 and NDRG2 is upregulated by p53 and HIF-1 and contributes to apoptosis driven by those pathways (8,9,11-14).
	Research studies show that NDRG1 may play a role in cancer progression by promoting differentiation, inhibiting growth, and modulating metastasis and angiogenesis (7,8,10,15,16). Nonsense mutation in the corresponding NDRG1 gene can cause hereditary motor and sensory neuropathy-Lom (HMSNL), which is supported by studies demonstrating the role of NDRG1 in maintaining myelin sheaths and axonal survival (17,18). NDRG1 is upregulated during mast cell maturation and its deletion leads to attenuated allergic responses (19). Elevated NDRG2 expression has been observed with Alzheimer's disease (20). Both NDGR1 and NDGR2 are phosphorylated at multiple sites by Akt and/or SGK1, although the precise physiological role of this phosphorylation is unclear (21,22). NDRG1 is phosphorylated by SGK1 at Thr328, Ser330, Thr346, Thr356, and Thr366. Phosphorylation by SGK1 primes NDRG1 for phosphorylation by GSK-3.
	NDRG3 is most highly expressed in the testis, prostate, ovary, and brain and is upregulated by androgen in prostate cell lines, promoting cell growth in those cell lines (2,23-25). Unlike other widely expressed family members, several alternatively spliced NDRG4 (Bdm1) isoforms are expressed primarily in the heart and brain (2,3,26,27). Expression of NDRG4 is reduced in the brain of patients with Alzheimer's disease, but is elevated in glioblastoma where it contributes to cell cycle progression and survival (3,28). NDRG4 also may be inactivated by promoter methylation in colorectal cancer and function as a tumor suppressor gene (29).
Background References	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.

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