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## Phospho-SAMHD1 (Thr592) (D7O2M) Rabbit mAb



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Applications: W, IP, FC-FP	<b>Reactivity:</b> H M R	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 72	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #Q9Y3Z3	Entrez-Gene Id: 25939	
Product Usage Information Storage	2	ApplicationDilutionWestern Blotting1:1000Immunoprecipitation1:200Flow Cytometry (Fixed/Permeabilized)1:800Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA, 50% glycerol and less than0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.				1:1000 1:200 1:800	
Specificity/Ser	sitivity	For a carrier free (BSA and azide free) version of this product see product #27254. Phospho-SAMHD1 (Thr592) (D7O2M) Rabbit mAb recognizes endogenous levels of SAMDH1 protein only when phosphorylated at Thr592.					
Source / Purifi	rce / Purification Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding residues surrounding Thr592 of human SAMHD1 protein.				orresponding to		
Background		SAM domain and HD domain-containing protein 1 (SAMHD1) is a negative regulator of the cell-intrinsic innate immune response (1). Research studies have identified mutations in <i>SAMHD1</i> as a cause of Aicardi-Goutieres syndrome, an autoimmune disease characterized by elevated production of interferon-α and symptoms resembling congenital viral infection (1). SAMHD1 was identified as the restriction factor that renders most myeloid cells refractory to human immunodeficiency virus (HIV) infection (2-4). Expression of the viral protein Vpx in refractory cells targets SAMHD1 for ubiquitin-mediated degradation and relieves HIV restriction. SAMHD1 prevents autoimmunity and HIV infection by hydrolyzing intracellular deoxynucleoside triphosphates (dNTPs), thereby limiting inappropriate immune activation by self nucleic acid and inhibiting reverse transcription of the HIV genome (4-6). Phosphorylation of Thr592 by cyclin A2/CDK1 was identified as a regulatory mechanism that controls SAMHD1 activity (7,8). SAMHD1 is phosphorylated in proliferating cells, which inhibits its ability to block HIV infection. In resting cells or in cells treated with PMA (TPA) or IFN-α, SAMHD1 phosphorylation is decreased and cells are refractory to HIV infection (7,8).					
Background R	eferences	<ol> <li>Rice, G.I. et al. (2009) Nat Genet 41, 829-32.</li> <li>Laguette, N. et al. (2011) Nature 474, 654-7.</li> <li>Hrecka, K. et al. (2011) Nature 474, 658-61.</li> <li>Powell, R.D. et al. (2011) J Biol Chem 286, 43596-600.</li> <li>Goldstone, D.C. et al. (2011) Nature 480, 379-82.</li> <li>Lahouassa, H. et al. (2012) Nat Immunol 13, 223-8.</li> <li>Cribier, A. et al. (2013) Cell Rep 3, 1036-43.</li> <li>White, T.E. et al. (2013) Cell Host Microbe 13, 441-51.</li> </ol>					
Species Reacti	vity	Species reactivity is determined by testing in at least one approved application (e.g., western blot).					
Western Blot E	Buffer		TANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X 1% Tween® 20 at 4°C with gentle shaking, overnight.				
Applications K	ey	W: Western Blotting IP: Immunoprecipitation FC-FP: Flow Cytometry (Fixed/Permeabilized)					
Cross-Reactivi	ty Key	H: Human M: Mouse R: Rat					
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