

**Phospho-SAMHD1 (Thr592) Antibody**

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

<b>Applications:</b> W, IP	<b>Reactivity:</b> H M	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 69, 72	<b>Source/Isotype:</b> Rabbit	<b>UniProt ID:</b> #Q9Y3Z3	<b>Entrez-Gene Id:</b> 25939
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**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 and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

**Specificity/Sensitivity**

Phospho-SAMHD1 (Thr592) Antibody recognizes endogenous levels of SAMHD1 protein only when phosphorylated at Thr592.

**Source / Purification**

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Thr592 of human SAMHD1 protein. Antibodies are purified by protein A and peptide affinity chromatography.

**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 *SAMHD1* 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 References**

1. Rice, G.I. et al. (2009) *Nat Genet* 41, 829-32.
2. Laguette, N. et al. (2011) *Nature* 474, 654-7.
3. Hrecka, K. et al. (2011) *Nature* 474, 658-61.
4. Powell, R.D. et al. (2011) *J Biol Chem* 286, 43596-600.
5. Goldstone, D.C. et al. (2011) *Nature* 480, 379-82.
6. Lahouassa, H. et al. (2012) *Nat Immunol* 13, 223-8.
7. Cribier, A. et al. (2013) *Cell Rep* 3, 1036-43.
8. White, T.E. et al. (2013) *Cell Host Microbe* 13, 441-51.

**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

**Cross-Reactivity Key**

**H:** Human **M:** Mouse

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