

SAMHD1 (phospho Thr592) Antibody
Catalog # ASC12060**Specification****SAMHD1 (phospho Thr592) Antibody - Product Information**

Application	WB, IHC, IF, E
Primary Accession	O9Y3Z3
Other Accession	38016914 , NP_056289 , 25939
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	72201
Application Notes	SAMHD1 (phospho Thr592) antibody can be used for detection of SAMHD1 (phospho Thr592) by western blot at at 0.5 - 1 µg/ml. Antibody can also be used for immunohistochemistry starting at 2.5 µg/mL. For immunofluorescence start at 20 µg/mL.

SAMHD1 (phospho Thr592) Antibody - Additional Information

Gene ID	25939
Other Names	SAM domain and HD domain 1, DCIP, CHBL2, HDDC1, MOP-5, SBBI88

Precautions

SAMHD1 (phospho Thr592) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

SAMHD1 (phospho Thr592) Antibody - Protein Information

Name SAMHD1 ([HGNC:15925](#))

Function

Protein that acts both as a host restriction factor involved in defense response to virus and as a regulator of DNA end resection at stalled replication forks (PubMed:[19525956](http://www.uniprot.org/citations/19525956), PubMed:[21613998](http://www.uniprot.org/citations/21613998), PubMed:[21720370](http://www.uniprot.org/citations/21720370), PubMed:[22056990](http://www.uniprot.org/citations/22056990), PubMed:[23601106](http://www.uniprot.org/citations/23601106), PubMed:[23602554](http://www.uniprot.org/citations/23602554), PubMed:[24336198](http://www.uniprot.org/citations/24336198), PubMed:[26294762](http://www.uniprot.org/citations/26294762), PubMed:[26431200](http://www.uniprot.org/citations/26431200), PubMed:[28229507](http://www.uniprot.org/citations/28229507), PubMed:[19525956](http://www.uniprot.org/citations/19525956), PubMed:[21613998](http://www.uniprot.org/citations/21613998), PubMed:[21720370](http://www.uniprot.org/citations/21720370), 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<http://www.uniprot.org/citations/28834754> target="_blank">28834754, PubMed:29670289). Has deoxynucleoside triphosphate (dNTPase) activity, which is required to restrict infection by viruses, such as HIV-1: dNTPase activity reduces cellular dNTP levels to levels too low for retroviral reverse transcription to occur, blocking early- stage virus replication in dendritic and other myeloid cells (PubMed:19525956, PubMed:21613998, PubMed:21720370, PubMed:22056990, PubMed:23364794, PubMed:23601106, PubMed:23602554, PubMed:24336198, PubMed:25038827, PubMed:26101257, PubMed:26294762, PubMed:26431200, PubMed:28229507). Likewise, suppresses LINE-1 retrotransposon activity (PubMed:24035396, PubMed:24217394, PubMed:29610582). Not able to restrict infection by HIV-2 virus; because restriction activity is counteracted by HIV-2 viral protein Vpx (PubMed:21613998, PubMed:21720370). In addition to virus restriction, dNTPase activity acts as a regulator of DNA precursor pools by regulating dNTP pools (PubMed:23858451). Phosphorylation at Thr-592 acts as a switch to control dNTPase-dependent and -independent functions: it inhibits dNTPase activity and ability to restrict infection by viruses, while it promotes DNA end resection at stalled replication forks (PubMed:23601106, PubMed:23602554, PubMed:29610582, PubMed:29670289). Functions during S phase at stalled DNA replication forks to promote the resection of gapped or reversed forks: acts by stimulating the exonuclease activity of MRE11, activating the ATR-CHK1 pathway and allowing the forks to restart replication (PubMed:29670289). Its ability to promote degradation of nascent DNA at stalled replication forks is required to prevent induction of type I interferons, thereby preventing chronic inflammation (PubMed:27477283, PubMed:29670289). Ability to promote DNA end resection at stalled replication forks is independent of dNTPase activity (PubMed:29670289). Enhances immunoglobulin hypermutation in B-lymphocytes by promoting transversion mutation (By similarity).

Cellular Location

Nucleus. Chromosome Note=Localizes to sites of DNA double-strand breaks in response to DNA damage.

Tissue Location

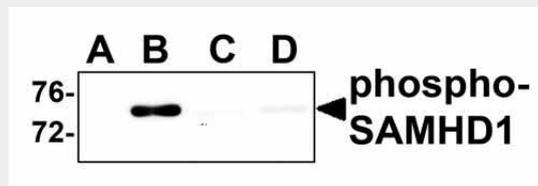
Expressed in heart, skeletal muscle, spleen, liver, small intestine, placenta, lung and peripheral blood leukocytes (PubMed:11064105). No expression is seen in brain and thymus (PubMed:11064105).

SAMHD1 (phospho Thr592) Antibody - Protocols

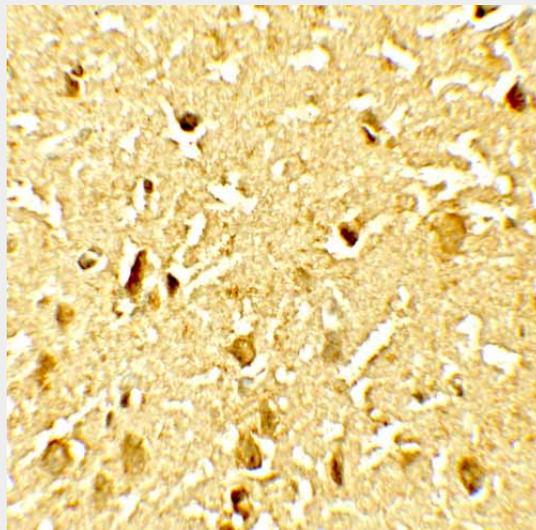
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

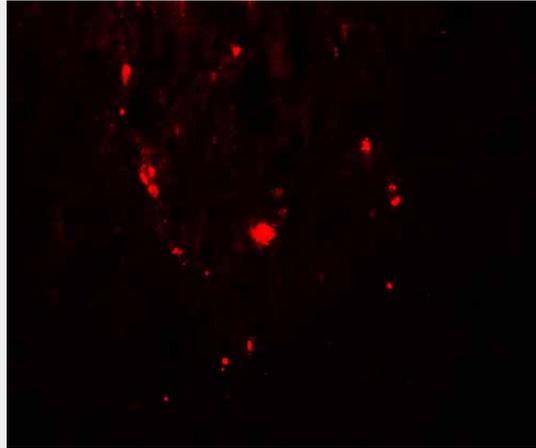
SAMHD1 (phospho Thr592) Antibody - Images



Western blot analysis of SAMHD1 (phospho Thr592) in 293 cells transfected with (A) empty expression vector, (B) wild-type SAMHD1, (C) SAMHD1 (T592A) and (D) SAMHD1 (T592E) with SAMHD1 (phospho Thr592) antibody at 1 µg/ml.



Immunohistochemistry of SAMHD1 (phospho Thr592) in human brain tissue with SAMHD1 (phospho Thr592) antibody at 2.5 µg/ml.



Immunofluorescence of phosphoSAMHD1 in human brain tissue with phosphoSAMHD1 antibody at 20 µg/mL.

SAMHD1 (phospho Thr592) Antibody - Background

The SAM domain and HD domain 1 (SAMHD1) protein is upregulated in response to viral infection and is thought to play a role in innate immunity (1). SAMHD1 blocks the infection of HIV-1 and SIVdeltaVpx before reverse transcription in macrophages and dendritic cells (2), and this restriction is regulated by phosphorylation of SAMHD1 (3). Mutations in this gene have been associated with Aicardi-Goutieres syndrome (1).

SAMHD1 (phospho Thr592) Antibody - References

Rice GI, Bond J, Asipu A, et al. Mutations involved in Aicardi-Goutieres syndrome implicate SAMHD1 as regulator of the innate immune system. *Nat. Genet.* 2009; 41:829-32.; Hrecka K, Hao C, Gierszewska M, et al. Vpx relieves the inhibition of HIV-1 infection of macrophages mediated by the SAMHD1 protein. *Nature* 2011; 474:654-7.; Welbourn S, Dutta SM, Semmes OJ, et al. Restriction of virus infection but not catalytic dNTPase activity is regulated by phosphorylation of SAMHD1. *J. Virol.* 2013; 87:11516-24.;