

SET / TAF-I Antibody

Rabbit Polyclonal Antibody Catalog # ALS16587

Specification

SET / TAF-I Antibody - Product Information

Application IHC, ICC, WB
Primary Accession Q01105
Other Accession 6418

Reactivity Human, Mouse, Rat, Monkey, Chicken,

Bovine Rabbit Polyclonal

Clonality Polycle Isotype IgG
Calculated MW 33489

SET / TAF-I Antibody - Additional Information

Gene ID 6418

Host

Other Names

SET, 2PP2A, HLA-DR-associated protein II, I-2PP2A, TAF-IBETA, SET nuclear oncogene, TAF-I, Template-activating factor I, I2PP2A, IGAAD, IPP2A2, PHAPII, Protein SET

Target/Specificity

Human SET

Reconstitution & Storage

PBS, pH 7.2, 50% glycerol. Store at -20°C.

Precautions

SET / TAF-I Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

SET / TAF-I Antibody - Protein Information

Name SET

Function

Multitasking protein, involved in apoptosis, transcription, nucleosome assembly and histone chaperoning. Isoform 2 anti-apoptotic activity is mediated by inhibition of the GZMA-activated DNase, NME1. In the course of cytotoxic T-lymphocyte (CTL)-induced apoptosis, GZMA cleaves SET, disrupting its binding to NME1 and releasing NME1 inhibition. Isoform 1 and isoform 2 are potent inhibitors of protein phosphatase 2A. Isoform 1 and isoform 2 inhibit EP300/CREBBP and PCAF- mediated acetylation of histones (HAT) and nucleosomes, most probably by masking the accessibility of lysines of histones to the acetylases. The predominant target for inhibition is histone H4. HAT inhibition leads to silencing of HAT-dependent transcription and prevents active demethylation of DNA. Both isoforms stimulate DNA replication of the adenovirus genome



complexed with viral core proteins; however, isoform 2 specific activity is higher.

Cellular Location

Cytoplasm, cytosol. Endoplasmic reticulum. Nucleus, nucleoplasm. Note=In the cytoplasm, found both in the cytosol and associated with the endoplasmic reticulum. The SET complex is associated with the endoplasmic reticulum. Following CTL attack and cleavage by GZMA, moves rapidly to the nucleus, where it is found in the nucleoplasm, avoiding the nucleolus. Similar translocation to the nucleus is also observed for lymphocyte-activated killer cells after the addition of calcium

Tissue Location

Widely expressed. Low levels in quiescent cells during serum starvation, contact inhibition or differentiation. Highly expressed in Wilms' tumor

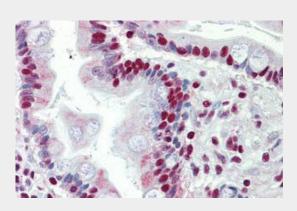
Volume 500 μl

SET / TAF-I Antibody - Protocols

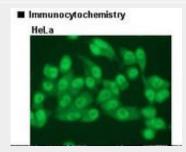
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

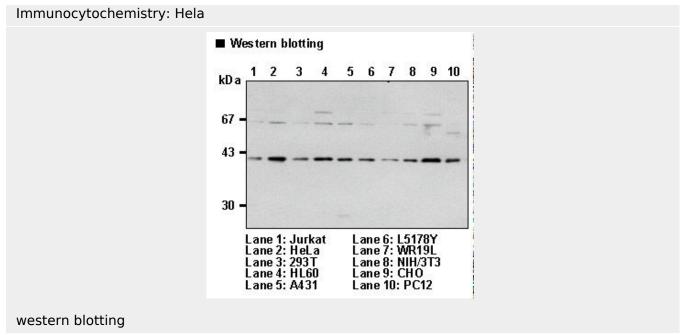
SET / TAF-I Antibody - Images



Anti-SET / TAF-I antibody IHC staining of human small intestine.







SET / TAF-I Antibody - Background

Multitasking protein, involved in apoptosis, transcription, nucleosome assembly and histone chaperoning. Isoform 2 anti-apoptotic activity is mediated by inhibition of the GZMA-activated DNase, NME1. In the course of cytotoxic T- lymphocyte (CTL)-induced apoptosis, GZMA cleaves SET, disrupting its binding to NME1 and releasing NME1 inhibition. Isoform 1 and isoform 2 are potent inhibitors of protein phosphatase 2A. Isoform 1 and isoform 2 inhibit EP300/CREBBP and PCAF-mediated acetylation of histones (HAT) and nucleosomes, most probably by masking the accessibility of lysines of histones to the acetylases. The predominant target for inhibition is histone H4. HAT inhibition leads to silencing of HAT-dependent transcription and prevents active demethylation of DNA. Both isoforms stimulate DNA replication of the adenovirus genome complexed with viral core proteins; however, isoform 2 specific activity is higher.

SET / TAF-I Antibody - References

von Lindern M.,et al.Mol. Cell. Biol. 12:3346-3355(1992). Vaesen M.,et al.Biol. Chem. Hoppe-Seyler 375:113-126(1994). Nagata K.,et al.Proc. Natl. Acad. Sci. U.S.A. 92:4279-4283(1995). Li M.,et al.J. Biol. Chem. 271:11059-11062(1996). Tsuijo I.,et al.FEBS Lett. 579:363-372(2005).