



Rabbit Anti-phospho-Ep300 (Ser1834) antibody

SL5339R

Product Name:	phospho-Ep300 (Ser1834)
Chinese Name:	磷酸化转录接头蛋白EP300抗体
Alias:	CREBBP/EP300 inhibitory protein 1; Cyclic AMP responsive enhancer binding protein; E1A associated protein p300; E1A binding protein p300; EC 2.3.1.48; EP300; EP300: E1A binding protein p300; RB and P300 binding protein EID 1; Histone acetyltransferase p300; p300 HAT; Retinoblastoma protein associated protein; EP300_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Chicken,Dog,Pig,Cow,Horse,Guinea Pig,Danio
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800IF=1:100-500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	266kDa
Cellular localization:	The nucleuscytoplasmic
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated Synthesised phosphopeptide derived from human Ep300 around the phosphorylation site of Ser1834:MA(p-S)MQ
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	KAT3B/p300 is a transcriptional adapter protein (300 kDa)which is characterized by

three cysteine and histidine rich regions and its C-terminus specifically binds the adenovirus E1A protein. KAT3B and associated proteins are components of TATA-binding protein (TBP) complexes. Protein kinase A mediated CREB phosphorylation results in the binding of CREB to a 265 kDa nuclear protein designated KAT3A/CBP (for CREB-binding protein). KAT3B and KAT3A are homologous to each other.

Function:

Functions as histone acetyltransferase and regulates transcription via chromatin remodeling. Acetylates all four core histones in nucleosomes. Histone acetylation gives an epigenetic tag for transcriptional activation. Mediates cAMP-gene regulation by binding specifically to phosphorylated CREB protein. Also functions as acetyltransferase for nonhistone targets. Acetylates 'Lys-131' of ALX1 and acts as its coactivator in the presence of CREBBP. Acetylates SIRT2 and is proposed to indirectly increase the transcriptional activity of TP53 through acetylation and subsequent attenuation of SIRT2 deacetylase function. Acetylates HDAC1 leading to its inactivation and modulation of transcription. Acts as a TFAP2A-mediated transcriptional coactivator in presence of CITED2. Plays a role as a coactivator of NEUROD1-dependent transcription of the secretin and p21 genes and controls terminal differentiation of cells in the intestinal epithelium. Promotes cardiac myocyte enlargement. Can also mediate transcriptional repression. Binds to and may be involved in the transforming capacity of the adenovirus E1A protein. In case of HIV-1 infection, it is recruited by the viral protein Tat. Regulates Tat's transactivating activity and may help inducing chromatin remodeling of proviral genes. Acetylates FOXO1 and enhances its transcriptional activity.

Subunit:

Interacts with phosphorylated CREB1. Interacts with HIF1A; the interaction is stimulated in response to hypoxia and inhibited by CITED2. Interacts (via N-terminus) with TFAP2A (via N-terminus); the interaction requires CITED2. Interacts (via CH1 domain) with CITED2 (via C-terminus). Interacts with CITED1 (unphosphorylated form preferentially and via C-terminus). Interacts with ESR1; the interaction is estrogen-dependent and enhanced by CITED1. Interacts with DTX1, EID1, ELF3, FEN1, LEF1, NCOA1, NCOA6, NR3C1, PCAF, PELP1, PRDM6, SP1, SP3, SPIB, SRY, TCF7L2, TP53, DDX5, DDX17, SATB1, SRCAP, TTC5, JMY and TRERF1. The TAZ-type 1 domain interacts with HIF1A. Probably part of a complex with HIF1A and CREBBP. Part of a complex containing CARM1 and NCOA2/GRIP1. Interacts with ING4 and this interaction may be indirect. Interacts with ING5. Interacts with the C-terminal region of CITED4. Interacts with HTLV-1 Tax and p30II. Interacts with and acetylates HIV-1 Tat. Non-sumoylated EP300 preferentially interacts with SENP3. Interacts with SS18L1/CREST. Interacts with ALX1 (via homeobox domain). Interacts with NEUROD1; the interaction is inhibited by NR0B2. Interacts with TCF3. Interacts (via CREB-binding domain) with MYOCD (via C-terminus). Binds to HIPK2. Interacts with ROCK2 and PPARG. Forms a complex made of CDK9, CCNT1/cyclin-T1, EP300 and GATA4 that stimulates hypertrophy in cardiomyocytes. Interacts with IRF1 and this interaction enhances acetylation of p53/TP53 and stimulation of its activity. Interacts

with FOXO1; the interaction acetylates FOXO1 and enhances its transcriptional activity. Interacts with DDIT3/CHOP.

Subcellular Location:

Cytoplasm. Nucleus. Note=In the presence of ALX1 relocalizes from the cytoplasm to the nucleus. Co-localizes with ROCK2 in the nucleus.

Post-translational modifications:

Acetylated on Lys at up to 17 positions by intermolecular autocatalysis. Deacetylated in the transcriptional repression domain (CRD1) by SIRT1, preferentially at Lys-1020. Citrullinated at Arg-2142 by PADI4, which impairs methylation by CARM1 and promotes interaction with NCOA2/GRIP1. Methylated at Arg-580 and Arg-604 in the KIX domain by CARM1, which blocks association with CREB, inhibits CREB signaling and activates apoptotic response. Also methylated at Arg-2142 by CARM1, which impairs interaction with NCOA2/GRIP1. Sumoylated; sumoylation in the transcriptional repression domain (CRD1) mediates transcriptional repression. Desumoylated by SENP3 through the removal of SUMO2 and SUMO3. Probable target of ubiquitination by FBXO3, leading to rapid proteasome-dependent degradation. Phosphorylated by HIPK2 in a RUNX1-dependent manner. This phosphorylation that activates EP300 happens when RUNX1 is associated with DNA and CBFβ. Phosphorylated by ROCK2 and this enhances its activity. Phosphorylation at Ser-89 by AMPK reduces interaction with nuclear receptors, such as PPARγ.

DISEASE:

Note=Defects in EP300 may play a role in epithelial cancer.
Note=Chromosomal aberrations involving EP300 may be a cause of acute myeloid leukemias. Translocation t(8;22)(p11;q13) with KAT6A.
Defects in EP300 are the cause of Rubinstein-Taybi syndrome type 2 (RSTS2) [MIM:613684]. A disorder characterized by craniofacial abnormalities, postnatal growth deficiency, broad thumbs, broad big toes, mental retardation and a propensity for development of malignancies. Some individuals with RSTS2 have less severe mental impairment, more severe microcephaly, and a greater degree of changes in facial bone structure than RSTS1 patients.

SWISS:
Q09472

Gene ID:
2033

Database links:

[Entrez Gene: 2033](#)Human

[Entrez Gene: 328572](#)Mouse

[Entrez Gene: 170915](#)Rat

[Oimim: 602700](#)Human

[SwissProt: Q09472](#)Human

[SwissProt: B2RWS6](#)Mouse

[Unigene: 517517](#)Human

[Unigene: 655211](#)Human

[Unigene: 258397](#)Mouse

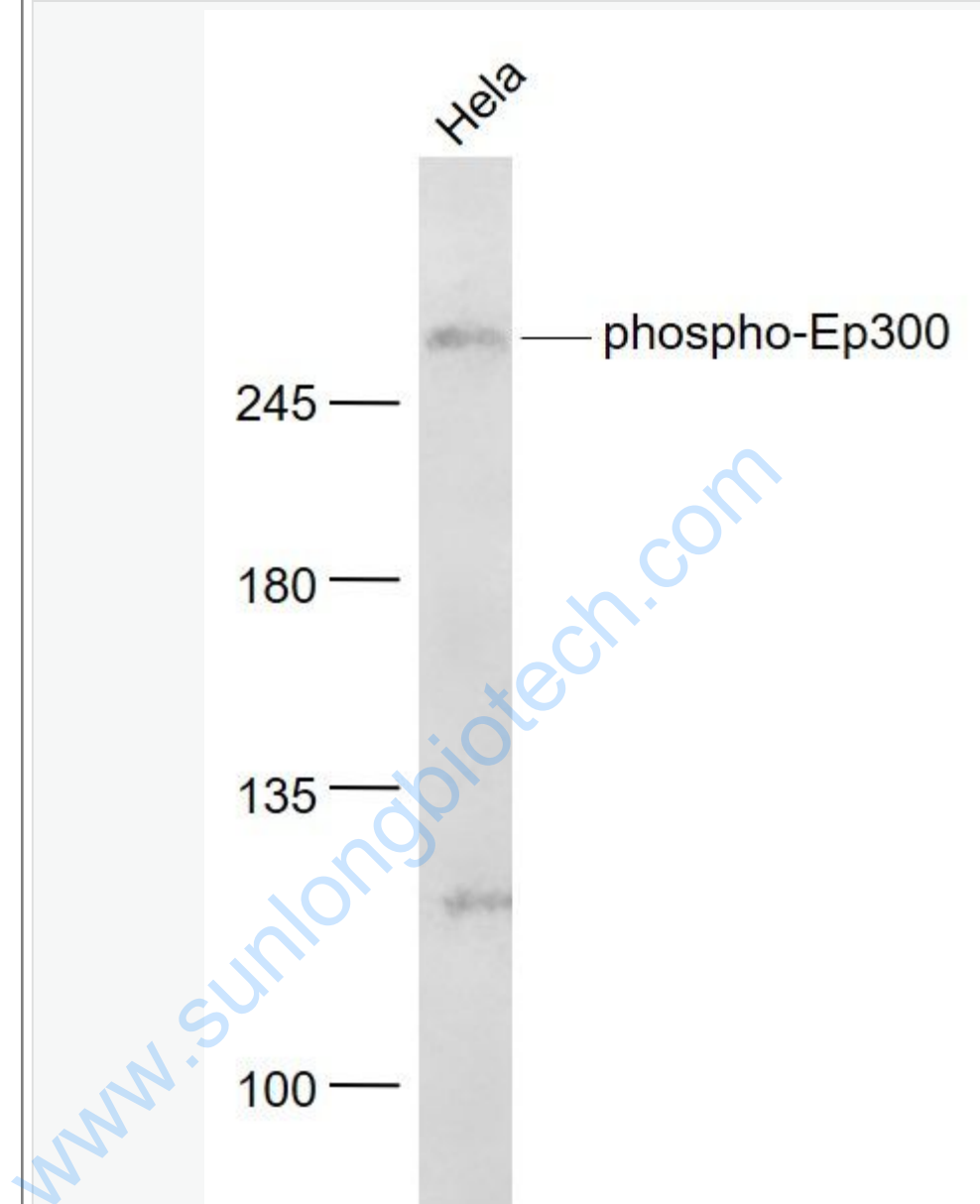
[Unigene: 12447](#)Rat

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

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Picture:



Sample:

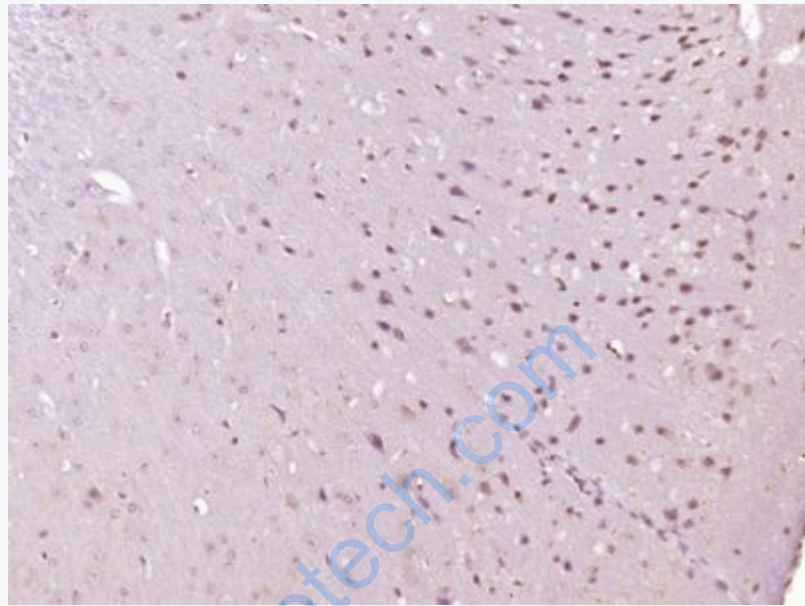
HeLa(Human) Cell Lysate at 30 ug

Primary: Anti- phospho-Ep300 (Ser1834) (SL5339R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 266 kD

Observed band size: 266 kD



Paraformaldehyde-fixed, paraffin embedded (Mouse brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (phospho-Ep300 (Ser1834)) Polyclonal Antibody, Unconjugated (SL5339R) at 1:400 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.