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Mouse Sirt1 Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP20849c

Specification

Mouse Sirt1 Blocking Peptide (C-term) - Product Information

Primary Accession Q923E4

Mouse Sirt1 Blocking Peptide (C-term) - Additional Information

Gene ID 93759

Other Names
NAD-dependent protein deacetylase sirtuin-1, 351-, Regulatory protein SIR2 homolog 1, SIR2-like protein 1, SIR2alpha, Sir2, mSIR2a, SirtT1 75 kDa fragment, 75SirT1, Sirt1, Sir2l1

Target/Specificity
The synthetic peptide sequence is selected from aa 566-581 of HUMAN Sirt1

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Mouse Sirt1 Blocking Peptide (C-term) - Protein Information

Name Sirt1

Synonyms Sir2l1

Function
NAD-dependent protein deacetylase that

Mouse Sirt1 Blocking Peptide (C-term) - Background

NAD-dependent protein deacetylase that links transcriptional regulation directly to intracellular energetics and participates in the coordination of several separated cellular functions such as cell cycle, response to DNA damage, metabolism, apoptosis and autophagy. Can modulate chromatin function through deacetylation of histones and can promote alterations in the methylation of histones and DNA, leading to transcriptional repression. Deacetylates a broad range of transcription factors and coregulators, thereby regulating target gene expression positively and negatively. Serves as a sensor of the cytosolic ratio of NAD(+)/NADH which is altered by glucose deprivation and metabolic changes associated with caloric restriction. Is essential in skeletal muscle cell differentiation and in response to low nutrients mediates the inhibitory effect on skeletal myoblast differentiation which also involves 5'-AMP-activated protein kinase (AMPK) and nicotinamide phosphoribosyltransferase (NAMPT). Component of the eNoSC (energy-dependent nucleolar silencing) complex, a complex that mediates silencing of rDNA in response to intracellular energy status and acts by recruiting histone-modifying enzymes. The eNoSC complex is able to sense the energy status of cell: upon glucose starvation, elevation of NAD(+)/NADP(+) ratio activates SIRT1, leading to histone H3 deacetylation followed by dimethylation of H3 at 'Lys-9' (H3K9me2) by SUV39H1 and the formation of silent chromatin in the rDNA locus. Deacetylates 'Lys-266' of SUV39H1, leading to its activation. Inhibits skeletal muscle differentiation by deacetylating PCAF and MYOD1. Deacetylates H2A and 'Lys-26' of HIST1H1E. Deacetylates 'Lys-16' of histone H4 (in vitro). Involved in NR0B2/SHP corepression function through chromatin remodeling: Recruited to LRH1 target gene promoters by NR0B2/SHP thereby stimulating histone H3 and H4 deacetylation leading to transcriptional

links transcriptional regulation directly to intracellular energetics and participates in the coordination of several separated cellular functions such as cell cycle, response to DNA damage, metabolism, apoptosis and autophagy (PubMed:11250901, PubMed:11672522, PubMed:12651913, PubMed:12887892, PubMed:12960381, PubMed:15175761, PubMed:15220471, PubMed:15632193, PubMed:15744310, PubMed:15788402, PubMed:16098828, PubMed:16366736, PubMed:16790548, PubMed:16892051, PubMed:17098745, PubMed:17347648, PubMed:17620057, PubMed:<a href="http://www.uniprot.org/ci

repression. Proposed to contribute to genomic integrity via positive regulation of telomere length; however, reports on localization to pericentromeric heterochromatin are conflicting. Proposed to play a role in constitutive heterochromatin (CH) formation and/or maintenance through regulation of the available pool of nuclear SUV39H1. Upon oxidative/metabolic stress decreases SUV39H1 degradation by inhibiting SUV39H1 polyubiquitination by MDM2. This increase in SUV39H1 levels enhances SUV39H1 turnover in CH, which in turn seems to accelerate renewal of the heterochromatin which correlates with greater genomic integrity during stress response. Deacetylates 'Lys-382' of p53/TP53 and impairs its ability to induce transcription-dependent proapoptotic program and modulate cell senescence. Deacetylates TAF1B and thereby represses rDNA transcription by the RNA polymerase I. Deacetylates MYC, promotes the association of MYC with MAX and decreases MYC stability leading to compromised transformational capability. Deacetylates FOXO3 in response to oxidative stress thereby increasing its ability to induce cell cycle arrest and resistance to oxidative stress but inhibiting FOXO3-mediated induction of apoptosis transcriptional activity; also leading to FOXO3 ubiquitination and protesomal degradation. Appears to have a similar effect on MLLT7/FOXO4 in regulation of transcriptional activity and apoptosis. Deacetylates DNMT1; thereby impairs DNMT1 methyltransferase-independent transcription repressor activity, modulates DNMT1 cell cycle regulatory function and DNMT1-mediated gene silencing. Deacetylates RELA/NF-kappa-B p65 thereby inhibiting its transactivating potential and augments apoptosis in response to TNF-alpha. Deacetylates HIF1A, KAT5/TIP60, RB1 and HIC1. Deacetylates FOXO1, which increases its DNA binding ability and enhances its transcriptional activity leading to increased gluconeogenesis in liver. Inhibits E2F1 transcriptional activity and apoptotic function, possibly by deacetylation. Involved in HES1- and HEY2-mediated transcriptional repression. In cooperation with MYCN seems to be involved in transcriptional repression of DUSP6/MAPK3 leading to MYCN stabilization by phosphorylation at 'Ser-62'. Deacetylates MEF2D. Required for antagonist-mediated transcription suppression of AR-dependent genes which may be linked to local

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modulate chromatin function through
deacetylation of histone H3. Represses HNF1A-
mediated transcription. Required for the
repression of ESRRG by CREBZF. Modulates
AP-1 transcription factor activity. Deacetylates
NR1H3 AND NR1H2 and deacetylation of
NR1H3 at 'Lys-434' positively regulates
transcription of NR1H3:RXR target genes,
promotes NR1H3 proteosomal degradation and
results in cholesterol efflux; a promoter
clearing mechanism after reach round of
transcription is proposed. Involved in lipid
metabolism. Implicated in regulation of
adipogenesis and fat mobilization in white
adipocytes by repression of PPARG which
probably involves association with NCOR1 and
SMRT/NCOR2. Deacetylates ACSS2 leading to
its activation, and HMGCS1. Involved in liver
and muscle metabolism. Through
deacteylation and activation of PPARGC1A is
required to activate fatty acid oxidation in
skeletel muscle under low-glucose conditions
and is involved in glucose homeostasis.
Involved in regulation of PPARA and fatty acid
beta-oxidation in liver. Involved in positive
regulation of insulin secretion in pancreatic
beta cells in response to glucose; the function
seems to imply transcriptional repression of
UCP2. Proposed to deacetylate IRS2 thereby
facilitating its insuline-induced tyrosine
phosphorylation. Deacetylates SREBF1 isoform
SREBP-1C thereby decreasing its stability and
transactivation in lipogenic gene expression.
Involved in DNA damage response by
repressing genes which are involved in DNA
repair, such as XPC and TP73, deacetylating
XRCC6/Ku70, and faciliting recruitment of
additional factors to sites of damaged DNA,
such as SIRT1-deacetylated NBN can recruit
ATM to initiate DNA repair and
SIRT1-deacetylated XPA interacts with RPA2.
Also involved in DNA repair of DNA double-
strand breaks by homologous recombination
and specifically single- strand annealing
independently of XRCC6/Ku70 and NBN.
Transcriptional suppression of XPC probably
involves an E2F4:RBL2 suppressor complex
and protein kinase B (AKT) signaling.
Transcriptional suppression of TP73 probably
involves E2F4 and PCAF. Deacetylates WRN
thereby regulating its helicase and
exonuclease activities and regulates WRN
nuclear translocation in response to DNA
damage. Deacetylates APEX1 at 'Lys-6' and
'Lys-7' and stimulates cellular AP endonuclease
activity by promoting the association of APEX1

deacetylation of histones and can promote alterations in the methylation of histones and DNA, leading to transcriptional repression (By similarity). Deacetylates a broad range of transcription factors and coregulators, thereby regulating target gene expression positively and negatively (By similarity). Serves as a sensor of the cytosolic ratio of NAD(+)/NADH which is altered by glucose deprivation and metabolic changes associated with caloric restriction (By similarity). Is essential in skeletal muscle cell differentiation and in response to low nutrients mediates the inhibitory effect on skeletal myoblast differentiation which also involves 5'-AMP-activated protein kinase (AMPK) and nicotinamide phosphoribosyltransferase (NAMPT) (PubMed:12887892, PubMed:18477450). Component of the eNoSC (energy-dependent nucleolar silencing) complex, a complex that mediates silencing of rDNA in response to intracellular energy status and acts by recruiting histone-modifying enzymes (By similarity). The eNoSC complex is able to sense the energy status of cell: upon glucose starvation, elevation of NAD(+)/NADP(+) ratio activates SIRT1, leading to histone H3 deacetylation followed by dimethylation of H3 at 'Lys-9' (H3K9me2) by SUV39H1 and the formation of silent chromatin in the rDNA locus (PubMed:18004385). Deacetylates 'Lys-266' of SUV39H1, leading to its activation (By similarity). Inhibits skeletal muscle differentiation by deacetylating PCAF and MYOD1 (PubMed:12887892). Deacetylates H2A and 'Lys-26' of H1-4 (By similarity). Deacetylates 'Lys-16' of histone H4 (in vitro) (By similarity). Involved in NR0B2/SHP corepression function through chromatin remodeling: Recruited to LRH1 target gene promoters by NR0B2/SHP thereby stimulating histone H3 and H4 deacetylation leading to transcriptional repression (By similarity). Proposed to contribute to genomic integrity via positive to XRCC1. Increases p53/TP53-mediated transcription-independent apoptosis by blocking nuclear translocation of cytoplasmic p53/TP53 and probably redirecting it to mitochondria. Deacetylates XRCC6/Ku70 at 'Lys-537' and 'Lys- 540' causing it to sequester BAX away from mitochondria thereby inhibiting stress-induced apoptosis. Is involved in autophagy, presumably by deacetylating ATG5, ATG7 and MAP1LC3B/ATG8. Deacetylates AKT1 which leads to enhanced binding of AKT1 and PDK1 to PIP3 and promotes their activation. Proposed to play role in regulation of STK11/LBK1-dependent AMPK signaling pathways implicated in cellular senescence which seems to involve the regulation of the acetylation status of STK11/LBK1. Can deacetylate STK11/LBK1 and thereby increase its activity, cytoplasmic localization and association with STRAD; however, the relevance of such activity in normal cells is unclear. In endothelial cells is shown to inhibit STK11/LBK1 activity and to promote its degradation. Deacetylates SMAD7 at 'Lys-64' and 'Lys- 70' thereby promoting its degradation. Deacetylates CIITA and augments its MHC class II transactivation and contributes to its stability. Deacteylates MECOM/EVI1. Isoform 2 is shown to deacetylate 'Lys-382' of p53/TP53, however with lower activity than isoform 1. In combination, the two isoforms exert an additive effect. Isoform 2 regulates p53/TP53 expression and cellular stress response and is in turn repressed by p53/TP53 presenting a SIRT1 isoform-dependent auto-regulatory loop. Deacetylates PML at 'Lys-487' and this deacetylation promotes PML control of PER2 nuclear localization. During the neurogenic transition, repress selective NOTCH1-target genes through histone deacetylation in a BCL6-dependent manner and leading to neuronal differentiation.

Mouse Sirt1 Blocking Peptide (C-term) - References

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Muth V.,et al.EMBO J. 20:1353-1362(2001).
McBurney M.W.,et al.Mol. Cancer Res. 1:402-409(2003).
McBurney M.W.,et al.Mol. Cell. Biol. 23:38-54(2003).

regulation of telomere length; however, reports on localization to pericentromeric heterochromatin are conflicting (PubMed:21187328). Proposed to play a role in constitutive heterochromatin (CH) formation and/or maintenance through regulation of the available pool of nuclear SUV39H1 (By similarity). Upon oxidative/metabolic stress decreases SUV39H1 degradation by inhibiting SUV39H1 polyubiquitination by MDM2 (By similarity). This increase in SUV39H1 levels enhances SUV39H1 turnover in CH, which in turn seems to accelerate renewal of the heterochromatin which correlates with greater genomic integrity during stress response (By similarity). Deacetylates 'Lys-382' of p53/TP53 and impairs its ability to induce transcription- dependent proapoptotic program and modulate cell senescence (PubMed:11672522, PubMed:12960381). Deacetylates TAF1B and thereby represses rDNA transcription by the RNA polymerase I (PubMed:11250901). Deacetylates MYC, promotes the association of MYC with MAX and decreases MYC stability leading to compromised transformational capability (By similarity). Deacetylates FOXO3 in response to oxidative stress thereby increasing its ability to induce cell cycle arrest and resistance to oxidative stress but inhibiting FOXO3-mediated induction of apoptosis transcriptional activity; also leading to FOXO3 ubiquitination and protesomal degradation (By similarity). Appears to have a similar effect on MLLT7/FOXO4 in regulation of transcriptional activity and apoptosis (By similarity). Deacetylates DNMT1; thereby impairs DNMT1 methyltransferase-independent transcription repressor activity, modulates DNMT1 cell cycle regulatory function and DNMT1- mediated gene silencing (By similarity). Deacetylates RELA/NF-kappa-B p65 thereby inhibiting its transactivating potential and augments apoptosis in

response to TNF-alpha (By similarity). Deacetylates HIF1A, KAT5/TIP60, RB1 and HIC1 (PubMed:17620057). Deacetylates FOXO1, which increases its DNA binding ability and enhances its transcriptional activity leading to increased gluconeogenesis in liver (PubMed:15220471, PubMed:15788402). Inhibits E2F1 transcriptional activity and apoptotic function, possibly by deacetylation (PubMed:16892051). Involved in HES1- and HEY2-mediated transcriptional repression (By similarity). In cooperation with MYCN seems to be involved in transcriptional repression of DUSP6/MAPK3 leading to MYCN stabilization by phosphorylation at 'Ser-62' (By similarity). Deacetylates MEF2D (By similarity). Required for antagonist-mediated transcription suppression of AR-dependent genes which may be linked to local deacetylation of histone H3 (By similarity). Represses HNF1A- mediated transcription (PubMed:21176092). Required for the repression of ESRRG by CREBZF (By similarity). Deacetylates NR1H3 and NR1H2 and deacetylation of NR1H3 at 'Lys-434' positively regulates transcription of NR1H3:RXR target genes, promotes NR1H3 proteosomal degradation and results in cholesterol efflux; a promoter clearing mechanism after reach round of transcription is proposed (PubMed:17936707). Involved in lipid metabolism (By similarity). Implicated in regulation of adipogenesis and fat mobilization in white adipocytes by repression of PPARG which probably involves association with NCOR1 and SMRT/NCOR2 (PubMed:15175761). Deacetylates p300/EP300 and PRMT1 (PubMed:15632193,

PubMed:28883095). Deacetylates ACSS2 leading to its activation, and HMGCS1 deacetylation (PubMed:16790548). Involved in liver and muscle metabolism (By similarity). Through deacetylation and activation of PPARGC1A is required to activate fatty acid oxidation in skeletal muscle under low-glucose conditions and is involved in glucose homeostasis (PubMed:15744310, PubMed:17347648). Involved in regulation of PPARA and fatty acid beta-oxidation in liver (PubMed:19356714). Involved in positive regulation of insulin secretion in pancreatic beta cells in response to glucose; the function seems to imply transcriptional repression of UCP2 (PubMed:16098828, PubMed:16366736, PubMed:17901049). Proposed to deacetylate IRS2 thereby facilitating its insulin-induced tyrosine phosphorylation (PubMed:17901049). Deacetylates SREBF1 isoform SREBP-1C thereby decreasing its stability and transactivation in lipogenic gene expression (By similarity). Involved in DNA damage response by repressing genes which are involved in DNA repair, such as XPC and TP73, deacetylating XRCC6/Ku70, and facilitating recruitment of additional factors to sites of damaged DNA, such as SIRT1-deacetylated NBN can recruit ATM to initiate DNA repair and SIRT1-deacetylated XPA interacts with RPA2 (By similarity). Also involved in DNA repair of DNA double-strand breaks by homologous recombination and specifically single-strand

annealing independently of XRCC6/Ku70 and NBN. Transcriptional suppression of XPC probably involves an E2F4:RBL2 suppressor complex and protein kinase B (AKT) signaling (By similarity). Transcriptional suppression of TP73 probably involves E2F4 and PCAF (By similarity). Deacetylates WRN thereby regulating its helicase and exonuclease activities and regulates WRN nuclear translocation in response to DNA damage (By similarity). Deacetylates APEX1 at 'Lys-6' and 'Lys-7' and stimulates cellular AP endonuclease activity by promoting the association of APEX1 to XRCC1 (By similarity). Increases p53/TP53-mediated transcription-independent apoptosis by blocking nuclear translocation of cytoplasmic p53/TP53 and probably redirecting it to mitochondria (By similarity). Deacetylates XRCC6/Ku70 at 'Lys-537' and 'Lys-540' causing it to sequester BAX away from mitochondria thereby inhibiting stress-induced apoptosis (By similarity). Is involved in autophagy, presumably by deacetylating ATG5, ATG7 and MAP1LC3B/ATG8 (PubMed:18296641, PubMed:21189328). Deacetylates AKT1 which leads to enhanced binding of AKT1 and PDK1 to PIP3 and promotes their activation (By similarity). Proposed to play role in regulation of STK11/LBK1-dependent AMPK signaling pathways implicated in cellular senescence which seems to involve the regulation of the acetylation status of STK11/LBK1 (PubMed:18687677). Can deacetylate STK11/LBK1 and thereby increase its activity, cytoplasmic localization and association with STRAD; however, the relevance of such activity in normal cells is unclear (By similarity). In endothelial cells is shown to inhibit STK11/LBK1 activity and to promote its degradation (By similarity). Deacetylates SMAD7 at 'Lys-64' and 'Lys- 70' thereby promoting its degradation (PubMed:17098745). Deacetylates CIITA and augments its MHC class II transactivation and contributes to its

stability (By similarity). Deacetylates MECOM/EVI1 (By similarity). Deacetylates PML at 'Lys-487' and this deacetylation promotes PML control of PER2 nuclear localization (By similarity). During the neurogenic transition, represses selective NOTCH1-target genes through histone deacetylation in a BCL6-dependent manner and leading to neuronal differentiation (By similarity). Regulates the circadian expression of several core clock genes, including ARNTL/BMAL1, RORC, PER2 and CRY1 and plays a critical role in maintaining a controlled rhythmicity in histone acetylation, thereby contributing to circadian chromatin remodeling (PubMed:18662546, PubMed:18662547, PubMed:19299583). Deacetylates ARNTL/BMAL1 and histones at the circadian gene promoters in order to facilitate repression by inhibitory components of the circadian oscillator (PubMed:18662546, PubMed:18662547, PubMed:19299583). Deacetylates PER2, facilitating its ubiquitination and degradation by the proteosome (PubMed:18662546). Protects cardiomyocytes against palmitate-induced apoptosis (PubMed:21622680). Deacetylates XBP1 isoform 2; deacetylation decreases protein stability of XBP1 isoform 2 and inhibits its transcriptional activity (By similarity). Deacetylates PCK1 and directs its activity toward phosphoenolpyruvate production promoting gluconeogenesis (PubMed:30193097). Involved in the CCAR2-mediated regulation of PCK1

and NR1D1 (By similarity). Deacetylates CTNB1 at 'Lys-49' (By similarity). In POMC (pro-opiomelanocortin) neurons, required for leptin-induced activation of PI3K signaling (PubMed:20620997). In addition to protein deacetylase activity, also acts as protein-lysine deacylase: acts as a protein depropionylase by mediating depropionylation of Osterix (SP7) (PubMed:30026585). Deacetylates SOX9; promoting SOX9 nuclear localization and transactivation activity (PubMed:26910618). Involved in the regulation of centrosome duplication. Deacetylates CENATAC in G1 phase, allowing for SASS6 accumulation on the centrosome and subsequent procentriole assembly (By similarity).

Cellular Location
Nucleus, PML body {ECO:0000250|UniProtKB:Q96EB6}. Cytoplasm Nucleus. Note=Colocalizes in the nucleus with XBP1 isoform 2. Recruited to the nuclear bodies via its interaction with PML. Colocalized with APEX1 in the nucleus. May be found in nucleolus, nuclear euchromatin, heterochromatin and inner membrane (By similarity). Shuttles between nucleus and cytoplasm (PubMed:17197703) {ECO:0000250|UniProtKB:Q96EB6, ECO:0000269|PubMed:17197703}

Tissue Location
Widely expressed. Weakly expressed in liver and skeletal muscle.

Mouse Sirt1 Blocking Peptide (C-term) - Protocols

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

- Blocking Peptides