10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

HIC1 Rabbit pAb

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Catalog # AP56020

Product Information

Application	WB
Primary Accession	Q14526
Reactivity	Mouse
Predicted	Human, Rat, Chicken, Dog, Pig, Horse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	76508
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human HIC1
Epitope Specificity	501-650/733
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Nucleus.
SIMILARITY	Belongs to the krueppel C2H2-type zinc-finger protein family. Hic subfamily.Contains 1 BTB (POZ) domain.Contains 5 C2H2-type zinc fingers.
SUBUNIT	Self-associates. Interacts with HIC2. Interacts with CTBP1 and CTBP2. Interacts with TCF7L2 and ARID1A. Interacts with MTA1 and MBD3; indicative for an association with the NuRD complex.
Post-translational modifications	Acetylated on several residues, including Lys-333. Lys-333 is deacetylated by SIRT1.Sumoylated on Lys-333 by a PIAS family member, which enhances interaction with MTA1, positively regulates transcriptional repression activity and is enhanced by HDAC4.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Hypermethylated in cancer (HIC-1) was originally identified as a target of p53-induced gene expression. HIC-1 is deleted in the genetic disorder Miller-Dieker syndrome (MDS), and the expression of HIC-1 is also frequently suppressed in leukemia and various cancers due to the hypermethylation of specific DNA regions and the resulting transcriptional silencing. These and other studies indicate that HIC-1 acts as a putative tumor suppressor protein that mediates transcriptional repression. HIC-1 is ubiquitously expressed in adult tissues and its structure is defined by five zinc fingers and an N-terminal broad complex POZ (or BTB) domain. In several BTB/POZ containing proteins, including BCL-6 and the promyelocytic leukemia zinc-finger (PLZF) oncoprotein, this domain interacts with the SMRT/N-CoR-mSin3A HDAC complex and is directly involved in repressing and silencing gene transcription. When this domain is deleted, as with the oncogenic PLZF-RAR chimera of promyelocytic leukemias, this transcriptional repression is attenuated. Conversely, HIC-1 does not interact with components of the HDAC complex, suggesting that HIC-1-induced transcriptional repression is unassociated with the POZ/BTB domain.

Additional Information

Gene ID	3090
Other Names	Hypermethylated in cancer 1 protein, Hic-1, Zinc finger and BTB domain-containing protein 29, HIC1, ZBTB29
Target/Specificity	Ubiquitously expressed with highest levels found in lung, colon, prostate, thymus, testis and ovary. Expression is absent or decreased in many tumor cells.
Dilution	WB=1:500-2000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. 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.

Protein Information

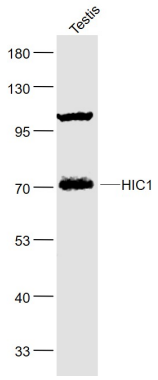
Name	HIC1
Synonyms	ZBTB29
Function	Transcriptional repressor (PubMed: 12052894, PubMed:15231840). Recognizes and binds to the consensus sequence '5-[CG]NG[CG]GGGCA[CA]CC-3' (PubMed:15231840). May act as a tumor suppressor (PubMed:20154726). Involved in development of head, face, limbs and ventral body wall (By similarity). Involved in down- regulation of SIRT1 and thereby is involved in regulation of p53/TP53- dependent apoptotic DNA-damage responses (PubMed:16269335). The specific target gene promoter association seems to be depend on corepressors, such as CTBP1 or CTBP2 and MTA1 (PubMed:12052894, PubMed:20547755). In cooperation with MTA1 (indicative for an association with the NuRD complex) represses transcription from CCND1/cyclin-D1 and CDKN1C/p57Kip2 specifically in quiescent cells (PubMed:20547755). Involved in regulation of the Wnt signaling pathway probably by association with TCF7L2 and preventing TCF7L2 and CTNNB1 association with promoters of TCF-responsive genes (PubMed:16724116). Seems to repress transcription from E2F1 and ATOH1 which involves ARID1A, indicative for the participation of a distinct SWI/SNF-type chromatin-remodeling complex (PubMed:18347096, PubMed:19486893). Probably represses transcription of ACKR3, FGFBP1 and EFNA1 (PubMed:16690027, PubMed:19525223, PubMed:20154726).
Cellular Location	Nucleus.
Tissue Location	Ubiquitously expressed with highest levels found in lung, colon, prostate, thymus, testis and ovary. Expression is absent or decreased in many tumor cells

Background

Hypermethylated in cancer (HIC-1) was originally identified as a target of p53-induced gene expression. HIC-1 is deleted in the genetic disorder Miller-Dieker syndrome (MDS), and the expression of HIC-1 is also frequently suppressed in leukemia and various cancers due to the hypermethylation of specific DNA regions and the resulting transcriptional silencing. These and other studies indicate that HIC-1 acts as a putative

tumor suppressor protein that mediates transcriptional repression. HIC-1 is ubiquitously expressed in adult tissues and its structure is defined by five zinc fingers and an N-terminal broad complex POZ (or BTB) domain. In several BTB/POZ containing proteins, including BCL-6 and the promyelocytic leukemia zinc-finger (PLZF) oncoprotein, this domain interacts with the SMRT/N-CoR-mSin3A HDAC complex and is directly involved in repressing and silencing gene transcription. When this domain is deleted, as with the oncogenic PLZF-RAR chimera of promyelocytic leukemias, this transcriptional repression is attenuated. Conversely, HIC-1 does not interact with components of the HDAC complex, suggesting that HIC-1-induced transcriptional repression is unassociated with the POZ/BTB domain.

Images



Sample:
Testis (Mouse) Lysate at 40 ug
Primary: Anti-HIC1 (AP56020) at 1/1000 dilution
Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution
Predicted band size: 76 kD
Observed band size: 72 kD

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.