

ATRX Rabbit pAb

CatalogNo: YT0420

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse

Applications

- IF, ELISA

MW

- 283kD (Calculated)

Isotype

- IgG

Recommended Dilution Ratios

IF 1:200-1:1000

ELISA 1:10000

Not yet tested in other applications

Storage

Storage*

-15°C to -25°C/1 year (Do not lower than -25°C)

Formulation

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality

Polyclonal

Immunogen Information

Immunogen

The antiserum was produced against synthesized peptide derived from human ATRX. AA range: 111-160

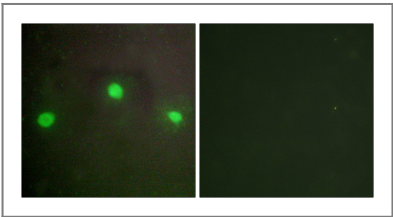
Specificity

ATRX Polyclonal Antibody detects endogenous levels of ATRX protein.

Target Information

Gene name	ATRX		
Protein Name	Transcriptional regulator ATRX		
	Organism	Gene ID	UniProt ID
	Human	546;	P46100;
	Mouse	22589;	Q61687;
Cellular Localization	Nucleus. Chromosome, telomere. Nucleus, PML body. Associated with pericentromeric heterochromatin during interphase and mitosis, probably by interacting with CBX5/HP1 alpha. Colocalizes with histone H3.3, DAXX, HIRA and ASF1A at PML-nuclear bodies. Colocalizes with cohesin (SMC1 and SMC3) and MECP2 at the maternal H19 ICR (By similarity). .		
Tissue specificity	Ubiquitous.		
Function	<p>Disease:Defects in ATRX are a cause of alpha-thalassemia myelodysplasia syndrome (ATMDS) [MIM:300448]. In this disorder, alpha-thalassemia occurs as an acquired abnormality in association with a multilineage myelodysplasia.,Disease:Defects in ATRX are the cause of mental retardation syndromic X-linked with hypotonic facies syndrome type 1 (MRXSHF1) [MIM:309580]; also called Carpenter-Waziri syndrome (CWS), Juberg-Marsidi syndrome (JMS), Smith-Fineman-Myers syndrome type 1 (SFM1). Clinical features include severe mental retardation, dysmorphic facies, and a highly skewed X-inactivation pattern in carrier women. Other more variable features include hypogonadism, deafness, renal anomalies, and mild skeletal defects.,Disease:Defects in ATRX are the cause of X-linked alpha-thalassemia/mental retardation syndrome (ATR-X) [MIM:301040]. ATR-X is an X-linked disorder comprising severe psychomotor retardation, facial dysmorphism, urogenital abnormalities, and alpha-thalassemia. An essential phenotypic trait are hemoglobin H erythrocyte inclusions.,Domain:Contains one Pro-Xaa-Val-Xaa-Leu (PxVxL) motif, which is required for interaction with chromoshadow domains. This motif requires additional residues -7, -6, +4 and +5 of the central Val which contact the chromoshadow domain.,Function:Could be a global transcriptional regulator. Modifies gene expression by affecting chromatin. May be involved in brain development and facial morphogenesis.,PTM:Phosphorylated upon DNA damage, probably by ATM or ATR.,similarity:Belongs to the SNF2/RAD54 helicase family.,similarity:Contains 1 GATA-type zinc finger.,similarity:Contains 1 helicase ATP-binding domain.,similarity:Contains 1 helicase C-terminal domain.,similarity:Contains 1 PHD-type zinc finger.,subcellular location:Associated with pericentromeric heterochromatin during interphase and mitosis, probably by interacting with HP1.,subunit:Probably binds EZH2. Binds annexin V in a calcium and phosphatidylcholine/phosphatidylserine-dependent manner (By similarity). Interacts directly with CBX5 via the PxVxL motif.,tissue specificity:Ubiquitous.,</p>		

Validation Data



Immunofluorescence analysis of A549 cells, using ATRX Antibody. The picture on the right is blocked with the synthesized peptide.

| Contact information

Orders: order.cn@immunoway.com
Support: support.cn@immunoway.com
Telephone: 400-8787-807(China)
Website: <http://www.immunoway.com.cn>
Address: 2200 Ringwood Ave San Jose, CA 95131 USA



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