

Summary

Production Name	Ataxin-1 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,IHC-P,IF-P,IF-F,ICC/IF,ELISA
Reactivity	Human,Mouse

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	ATXN1
Alternative Names	ATXN1; ATX1; SCA1; Ataxin-1; Spinocerebellar ataxia type 1 protein
Gene ID	6310.0
SwissProt ID	P54253.The antiserum was produced against synthesized peptide derived from human Ataxin 1. AA range:742-791

Application

Dilution Ratio	WB 1:500-2000, IHC-P 1:100-1:300, IF-P/IF-F/ICC/IF 1:200-1:1000, ELISA 1:5000.Not yet tested in other applications.
Molecular Weight	87kDa

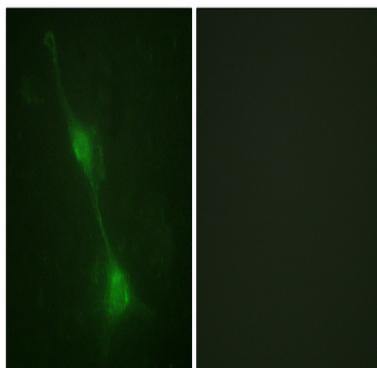
Background

ataxin 1(ATXN1) Homo sapiens The autosomal dominant cerebellar ataxias (ADCA) are a heterogeneous group of neurodegenerative disorders characterized by progressive degeneration of the cerebellum, brain stem and spinal cord. Clinically, ADCA has been divided into three groups: ADCA types I-III. ADCAI is genetically heterogeneous, with five genetic loci, designated spinocerebellar ataxia (SCA) 1, 2, 3, 4 and 6, being assigned to five different chromosomes. ADCAII, which always presents with retinal degeneration (SCA7), and ADCAIII often referred to as the 'pure' cerebellar syndrome (SCA5), are most likely homogeneous disorders. Several SCA genes have been cloned and shown to contain CAG repeats in their coding regions. ADCA is caused by the expansion of the CAG repeats, producing an elongated polyglutamine tract in the corresponding protein. The expanded repeats are variable in size and unstable, usually increasing in size when transmitted. At least 2 isoforms are produced. Defects in ATXN1 are the cause of spinocerebellar ataxia type 1 (SCA1) [MIM:164400]; also known as olivopontocerebellar atrophy I (OPCA I or OPCA1). Spinocerebellar ataxia is a clinically and genetically heterogeneous group of cerebellar disorders. Patients show progressive incoordination of gait and often poor coordination of hands, speech and eye movements, due to cerebellum degeneration with variable involvement of the brainstem and spinal cord. SCA1 belongs to the autosomal dominant cerebellar ataxias type I (ADCA I) which are characterized by cerebellar ataxia in combination with additional clinical features like optic atrophy, ophthalmoplegia, bulbar and extrapyramidal signs, peripheral neuropathy and dementia. SCA1 is caused by expansion of a CAG repeat in the coding region of ATXN1. Longer expansions result in earlier onset and more severe clinical manifestations of the disease. The AXH domain is required for interaction with CIC. Binds RNA in vitro. May be involved in RNA metabolism. The expansion of the polyglutamine tract may alter this function. The self-association seems to be necessary to form nuclear aggregates. Ataxin-1 entry. The poly-Gln region of ATXN1 is highly polymorphic (4 to 39 repeats) in the normal population and is expanded to about 40-83 repeats in spinocerebellar ataxia 1 (SCA1) patients. Belongs to the ATXN1 family. Contains 1 AXH domain. Colocalizes with USP7 in the nucleus. Interacts with CIC (By similarity). Interacts with ANP32A, PQBP1, UBIN, ATXN1L, USP7 and ZNF804A. Widely expressed throughout the body.

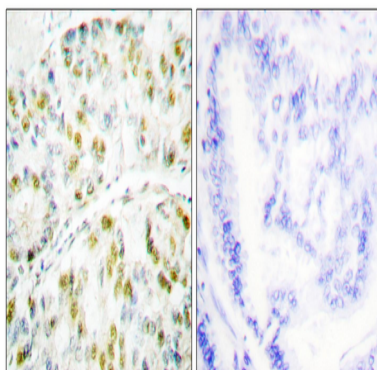
Research Area

Image Data

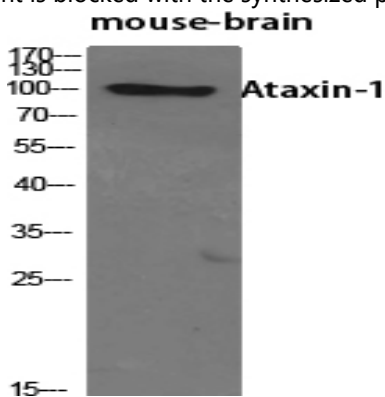
Product Name: Ataxin-1 Rabbit Polyclonal Antibody
Catalog #: APRab07251



Immunofluorescence analysis of NIH/3T3 cells, using Ataxin 1 Antibody. The picture on the right is blocked with the synthesized peptide.



Immunohistochemistry analysis of paraffin-embedded human lung carcinoma tissue, using Ataxin 1 Antibody. The picture on the right is blocked with the synthesized peptide.



Western Blot analysis of various cells using Ataxin-1 Polyclonal Antibody diluted at 1: 500

Note

For research use only.