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TOR1A Antibody (C-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP18209b

Specification

TOR1A Antibody (C-term) - Product Information

Application	**WB,E**
Primary Accession	O14656
Other Accession	NP_000104.1
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**37809**
Antigen Region	**267-295**

TOR1A Antibody (C-term) - Additional Information

Gene ID 1861

Other Names
Torsin-1A, Dystonia 1 protein, Torsin ATPase-1A, 364-, Torsin family 1 member A, TOR1A, DQ2, DYT1, TA, TORA

Target/Specificity
This TOR1A antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 267-295 amino acids from the C-terminal region of human TOR1A.

Dilution
WB~~1:1000

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
TOR1A Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

TOR1A Antibody (C-term) - Protein Information

Name TOR1A

Synonyms DQ2, DYT1, TA, TORA

Function Protein with chaperone functions important for the control of protein folding, processing, stability and localization as well as for the reduction of misfolded protein aggregates. Involved in the regulation of synaptic vesicle recycling, controls STON2 protein stability in collaboration with the COP9 signalosome complex (CSN). In the nucleus, may link the cytoskeleton with the nuclear envelope, this mechanism seems to be crucial for the control of nuclear polarity, cell movement and, specifically in neurons, nuclear envelope integrity. Participates in the cellular trafficking and may regulate the subcellular location of multipass membrane proteins such as the dopamine transporter SLC6A3, leading to the modulation of dopamine neurotransmission. In the endoplasmic reticulum, plays a role in the quality control of protein folding by increasing clearance of misfolded proteins such as SGCE variants or holding them in an intermediate state for proper refolding. May have a redundant function with TOR1B in non- neural tissues.

Cellular Location
Endoplasmic reticulum lumen. Nucleus membrane; Peripheral membrane protein. Cell projection, growth cone. Cytoplasmic vesicle membrane. Cytoplasmic vesicle, secretory vesicle. Cytoplasmic vesicle, secretory vesicle, synaptic vesicle. Cytoplasm, cytoskeleton. Note=Upon oxidative stress, redistributes to protusions from the cell surface (By similarity). Peripherally associated with the inner face of the ER membrane, probably mediated by the interaction with TOR1AIP1. The association with nucleus membrane is mediated by the interaction with TOR1AIP2.

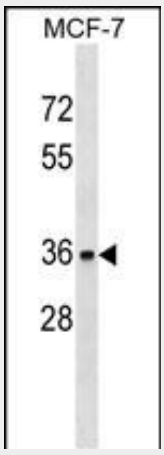
Tissue Location
Widely expressed. Highest levels in kidney and liver. In the brain, high levels found in the dopaminergic neurons of the substantia nigra pars compacta, as well as in the neocortex, hippocampus and cerebellum. Also highly expressed in the spinal cord

TOR1A Antibody (C-term) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

TOR1A Antibody (C-term) - Images



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TOR1A Antibody (C-term) (Cat. #AP18209b) western blot analysis in MCF-7 cell line lysates (35ug/lane).This demonstrates the TOR1A antibody detected the TOR1A protein (arrow).

TOR1A Antibody (C-term) - Background

The protein encoded by this gene is a member of the AAA family of adenosine triphosphatases (ATPases), is related to the Clp protease/heat shock family and is expressed prominently in the substantia nigra pars compacta. Mutations in this gene result in the autosomal dominant disorder, torsion dystonia 1. [provided by RefSeq].

TOR1A Antibody (C-term) - References

Sharma, N., et al. Mov. Disord. 25(13):2183-2187(2010)
Kaiser, F.J., et al. Ann. Neurol. 68(4):554-559(2010)
Gavarini, S., et al. Ann. Neurol. 68(4):549-553(2010)
Granata, A., et al. Eur. J. Neurol. 17 SUPPL 1, 81-87 (2010) :
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