

# PDCD10 Rabbit pAb

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Catalog # AP58526

## Product Information

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<b>Application</b>	IHC-P, IHC-F, IF
<b>Reactivity</b>	Rat
<b>Predicted</b>	Human, Mouse, Chicken, Pig, Horse, Rabbit
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Calculated MW</b>	22 KDa
<b>Physical State</b>	Liquid
<b>Immunogen</b>	KLH conjugated synthetic peptide derived from human PDCD10
<b>Epitope Specificity</b>	145-212/212
<b>Isotype</b>	IgG
<b>Purity</b>	affinity purified by Protein A
<b>Buffer</b>	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
<b>SUBCELLULAR LOCATION</b>	Cytoplasm. Golgi apparatus membrane; Peripheral membrane protein; Cytoplasmic side. Cell membrane; Peripheral membrane protein; Cytoplasmic side. Note=Partially co-localizes with endogenous PXN at the leading edges of migrating cells.
<b>SIMILARITY</b>	Belongs to the PDCD10 family.
<b>SUBUNIT</b>	Homodimer. Interacts (via C-terminus) with CCM2 and PXN. Interacts (via N-terminus) with MST4, STK24 and STK25. Interacts with GOLGA2. Identified in a complex with CCM1 and CCM2. Interacts with KDR/VEGFR2. Interaction with KDR/VEGFR2 is enhanced by stimulation with VEGFA.
<b>DISEASE</b>	Defects in PDCD10 are the cause of cerebral cavernous malformations type 3 (CCM3) [MIM:603285]. Cerebral cavernous malformations (CCMs) are congenital vascular anomalies of the central nervous system that can result in hemorrhagic stroke, seizures, recurrent headaches, and focal neurologic deficits. CCMs have an incidence of 0.1%-0.5% in the general population and usually present clinically during the 3rd to 5th decade of life. The lesions are characterized by grossly enlarged blood vessels consisting of a single layer of endothelium and without any intervening neural tissue, ranging in diameter from a few millimeters to several centimeters.
<b>Important Note</b>	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
<b>Background Descriptions</b>	This gene encodes an evolutionarily conserved protein associated with cell apoptosis. The protein interacts with the serine/threonine protein kinase MST4 to modulate the extracellular signal-regulated kinase (ERK) pathway. It also interacts with and is phosphorylated by serine/threonine kinase 25, and is thought to function in a signaling pathway essential for vascular development. Mutations in this gene are one cause of cerebral cavernous malformations, which are vascular malformations that cause seizures and cerebral hemorrhages. Multiple alternatively spliced variants, encoding the same protein, have been identified. [provided by RefSeq, Jul 2008].

## Additional Information

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<b>Other Names</b>	Programmed cell death protein 10, Cerebral cavernous malformations 3 protein, TF-1 cell apoptosis-related protein 15, PDCD10, CCM3, TFAR15
<b>Target/Specificity</b>	Ubiquitous.
<b>Dilution</b>	IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
<b>Storage</b>	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.

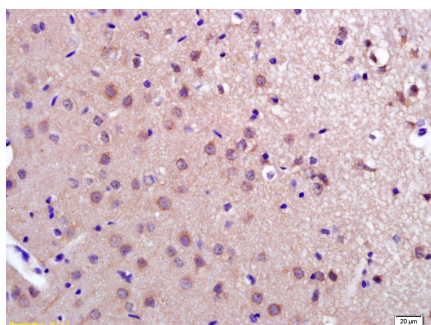
## Background

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## Images

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Tissue/cell: rat brain tissue; 4% Paraformaldehyde-fixed and paraffin-embedded;  
Antigen retrieval: citrate buffer ( 0.01M, pH 6.0 ), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum,C-0005) at 37°C for 20 min;  
Incubation: Anti-PDCD10 Polyclonal Antibody, Unconjugated(AP58526) 1:200, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining

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