

PAR4 Antibody
Purified Mouse Monoclonal Antibody
Catalog # AO1177a**Specification**

PAR4 Antibody - Product Information

Application	WB, IHC, E
Primary Accession	O96IZ0
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	IgG1

Description

Prostate apoptosis response 4 (Par4) is a 38kD protein originally identified as the product of a gene that is upregulated in prostate tumor cells undergoing apoptosis. It is a leucine zipper and death domain containing protein whose levels increase in neurons undergoing apoptosis as a result of trophic factor withdrawal or exposure to oxidative and metabolic insults. Par4 levels are reported to be increased in their lumbar spinal cord specimens further suggesting a role in neuronal degeneration. The tumor suppressor WT1 represses and activates transcription. The loss and/or imbalance of the dual transcriptional activity of WT1 may contribute to Wilms tumor. Par4 is a WT1 interacting protein that also functions as a transcriptional repressor.

Immunogen

Purified recombinant fragment of PAR4(aa1-330) expressed in E. Coli.

Formulation

Ascitic fluid containing 0.03% sodium azide.

PAR4 Antibody - Additional Information

Gene ID 5074

Other Names

PRKC apoptosis WT1 regulator protein, Prostate apoptosis response 4 protein, Par-4, PAWR, PAR4

Dilution

WB~~1/500 - 1/2000

IHC~~1/500 - 1/2000

E~~N/A

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

PAR4 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

PAR4 Antibody - Protein Information

Name PAWR

Synonyms PAR4

Function

Pro-apoptotic protein capable of selectively inducing apoptosis in cancer cells, sensitizing the cells to diverse apoptotic stimuli and causing regression of tumors in animal models. Induces apoptosis in certain cancer cells by activation of the Fas prodeath pathway and coparallel inhibition of NF-kappa-B transcriptional activity. Inhibits the transcriptional activation and augments the transcriptional repression mediated by WT1. Down-regulates the anti- apoptotic protein BCL2 via its interaction with WT1. Also seems to be a transcriptional repressor by itself. May be directly involved in regulating the amyloid precursor protein (APP) cleavage activity of BACE1.

Cellular Location

Cytoplasm. Nucleus. Note=Mainly cytoplasmic in absence of apoptosis signal and in normal cells. Nuclear in most cancer cell lines. Nuclear entry seems to be essential but not sufficient for apoptosis (By similarity). Nuclear localization includes nucleoplasm and PML nuclear bodies.

Tissue Location

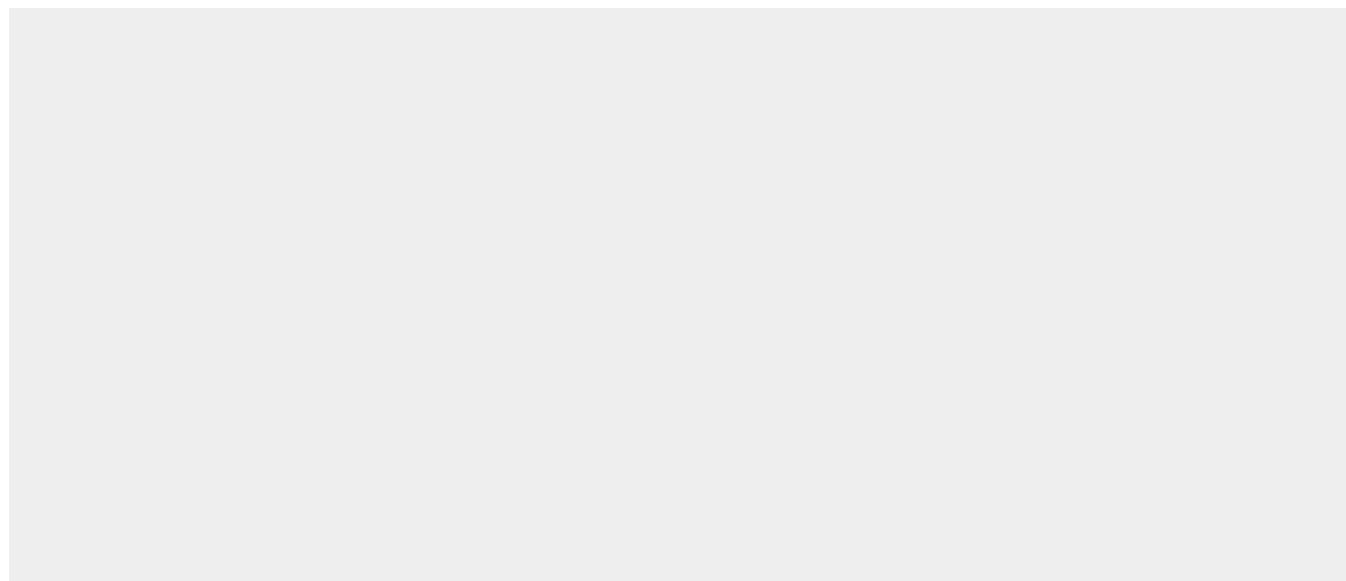
Widely expressed. Expression is elevated in various neurodegenerative diseases such as amyotrophic lateral sclerosis, Alzheimer, Parkinson and Huntington diseases and stroke. Down-regulated in several cancers.

PAR4 Antibody - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

PAR4 Antibody - Images



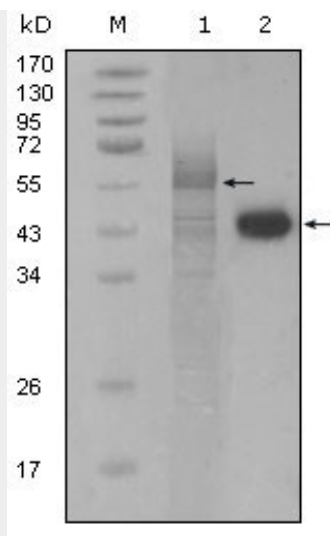


Figure 1: Western blot analysis using PAR4 mouse mAb against full-length Trx-Par4 recombinant protein (1) and Hela cell lysate (2).

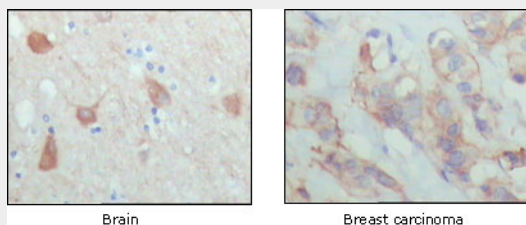


Figure 2: Immunohistochemical analysis of paraffin-embedded human brain and breast carcinoma, showing cytoplasmic and membrane localization with DAB staining using PAR4 antibody.

Figure 2: Immunohistochemical analysis of paraffin-embedded human brain (left) and breast carcinoma (right), showing cytoplasmic and membrane localization using PAR4 mouse mAb with DAB staining.

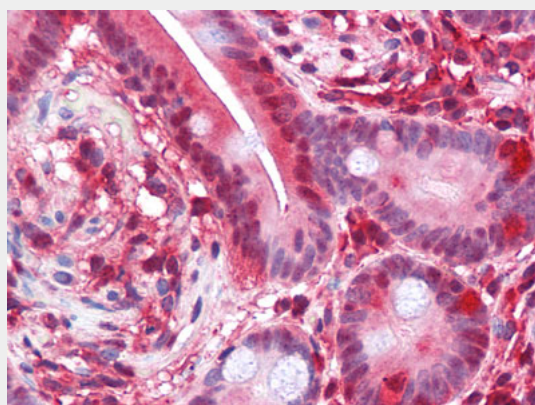


Figure 3: Immunohistochemical analysis of paraffin-embedded human Small Intestine tissues using PAR4 mouse mAb

PAR4 Antibody - References

1. J Biol Chem. 2004 Jul 2;279(27):28266-75.
2. Exp Hematol. 2004 Jul;32(7):649-56.
3. Mol Cell Biol. 2005 Feb;25(3):1146-61.
4. Psychiatr Genet. 2006 Oct;16(5):193-6.