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ProductInformation

ANTI-Par-4 (Prostate Apoptosis Response-4)
Developed in Rabbit
IgG Fraction of Antiserum

Product Number **P 5367**

Product Description

Anti-Par-4 (Prostate apoptosis response-4) is developed in rabbit using a synthetic peptide corresponding to the C-terminus of human Par-4 (amino acids 324-342) conjugated to KLH as immunogen. The Par-4 sequence is identical in rat Par-4. Whole antiserum is fractionated and then further purified by ion-exchange chromatography to provide the IgG fraction of antiserum that is essentially free of other rabbit serum proteins.

Anti-Par-4 (Prostate apoptosis response-4) may be used for the detection of Par-4 by immunoblotting (38 kDa). Staining of Par-4 in immunoblotting is specifically inhibited with the Par-4 immunizing peptide (amino acids 324-342).

Apoptosis is a crucial process in development, normal cellular differentiation, and in tissue homeostasis of all multicellular organisms.[1] Par-4 (**P**rostate **a**poptosis **r**esponse-**4**) is a 38 kDa pro-apoptotic protein identified in a screen for genes inducing apoptosis in prostate cancer cell lines.[1] Par-4 contains at its carboxyl terminus a leucine zipper domain and a death domain homologous to that found in other apoptotic genes such as Fas or TNF-α receptor-1.[2-4] Deletion of the leucine zipper domain results in loss of the pro-apoptotic function of Par-4 protein.

The *par-4* gene shows widespread expression in diverse tissue types and is exclusively induced by apoptotic stimuli by agents that elevate intracellular Ca^{2+} levels.[3, 5] In normal rat tissues, Par-4 is expressed in apoptotic and terminally differentiated cells.[6]

Par-4 is not induced by growth stimulation, growth arrest, oxidative stress, or necrotic signals. Functional studies indicate that Par-4 is not sufficient to cause apoptosis, but it can sensitize cells to the action of apoptotic agents. Par-4 interacts with and modulates the transcription and growth suppression function of the Wilm's tumor suppressor WT1.[2, 3] Oncogenic Ras causes down-regulation of Par-4 via the Raf-1-MEK-ERK pathway in immortalized fibroblasts.[7,8] It has been suggested that Par-4 can cause apoptosis by a p53-independent mechanism that may involve inhibition of downstream targets including protein kinase C-ζ and Bcl-2.[5, 9] Moreover, overexpression of Par-4 causes down-regulation of Bcl-2 in fibroblasts and prostate cancer cells. Restoration of Bcl-2 levels rescues the cells from the pro-apoptotic effects of Par-4, suggesting that Par-4 mediated apoptosis requires the inhibition of cell survival functions, such as Bcl-2. Par-4 plays an important role in neuronal apoptosis. It is upregulated in neuronal degeneration in Alzheimer's disease.[10] Par-4 levels are rapidly increased in hippocampal neurons in culture and *in vivo* following exposure to apoptotic or excitotoxic insults.[11, 12]

Reagent

Anti-Par-4 is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots at –20 °C. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

Product Profile

A minimum working dilution of 1:5,000 is determined by immunoblotting, using a whole cell extract of the human epitheloid carcinoma HeLa cell line.

A minimum working dilution of 1:1,000 is determined by immunoblotting, using a whole cell extract of the mouse fibroblast NIH-3T3 cell line.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration.

References

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