Product Information Sheet

Polyclonal Anti- Caspase-3 (P10)

Catalogue No. PA1302

Lot No. 09H01

Ig type rabbit IgG

Size 100µg/vial

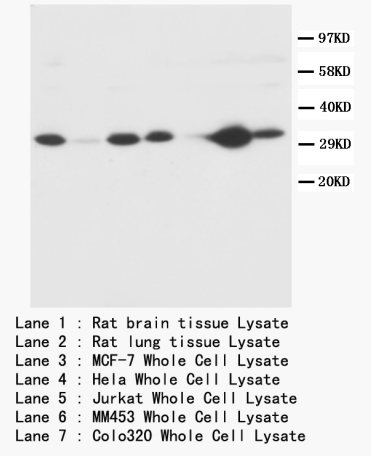
Specificity

Human, rat, mouse.

No cross reactivity with other proteins.

Recommended application

Western blot



Immunogen

A synthetic peptide corresponding to a sequence at the C-terminal of human Caspase-3 (P10), identical to the related rat and mouse sequence.

Purity

Immunogen affinity purified.

Application

	Concen-tration	Tested Species	Concluded Species	Antigen Retrieval
WB	1µg/ml	Hu, Rat	Ms	-
IHC-P	-	-	-	-
IHC-F	-	-	-	-
ICC	-	-	-	-

Other applications have not been tested.

Optimal dilutions should be determined by end user.

Contents

Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na_2HPO_4, 0.05mg Thimerosal, 0.05mg NaN_3.

Reconstitution

0.2ml of distilled water will yield a concentration of 500µg/ml.

Storage

At -20˚C for one year. After reconstitution, at 4˚C for one month. It can also be aliquotted and stored frozen at -20˚C for longer time.

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To reorder contact us at:

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BACKGROUND

Caspase 3 is a caspase protein which interacts with Survivin, XIAP, CFLAR, Caspase 8, HCLS1, Deleted in Colorectal Cancer, TRAF3 and GroEL. This gene which is located at 4q35 encodes a protein that is a member of the cysteine-aspartic acid protease (caspase) family. Sequential activation of caspases plays a central role in the execution-phase of cell apoptosis. Caspases exist as inactive proenzymes that undergo proteolytic processing at conserved aspartic residues to produce two subunits, large and small, that dimerize to form the active enzyme. This protein cleaves and activates caspases 6, 7, and 9; and the protein itself is processed by caspases 8, 9, and 10. It is the predominant caspase involved in the cleavage of amyloid-beta 4A precursor protein, which is associated with neuronal death in Alzheimer's disease. And the caspase-3 activation in heart failure sequentially cleaves SRF and generates a truncated SRF that appears to function as a dominant-negative transcription factor.[1] Additionally, the caspase-3 influence on bone mineral density should be considered in any in vivo application of caspase-3 inhibitors to the treatment of human disease.[2] In erythroid precursors undergoing terminal differentiation, Hsp70 prevents active CASP3 from cleaving GATA1 and inducing apoptosis.[3]

REFERENCE

1. Chang, J.; Wei, L.; Otani, T.; Youker, K. A.; Entman, M. L.; Schwartz, R. J. : Inhibitory cardiac transcription factor, SRF-N, is generated by caspase 3 cleavage in human heart failure and attenuated by ventricular unloading. *Circulation* 108: 407-413, 2003.
2. Miura, M.; Chen, X.-D.; Allen, M. R.; Bi, Y.; Gronthos, S.; Seo, B.-M.; Lakhani, S.; Flavell, R. A.; Feng, X.-H.; Robey, P. G.; Young, M.; Shi, S. : A crucial role of caspase-3 in osteogenic differentiation of bone marrow stromal stem cells. *J. Clin. Invest.* 114: 1704-1713, 2004.
3. Ribeil, J.-A.; Zermati, Y.; Vandekerckhove, J.; Cathelin, S.; Kersual, J.; Dussiot, M.; Coulon, S.; Moura, I. C.; Zeuner, A.; Kirkegaard-Sorensen, T.; Varet, B.; Solary, E.; Garrido, C.; Hermine, O. : Hsp70 regulates erythropoiesis by preventing caspase-3-mediated cleavage of GATA-1. *Nature* 445: 102-105, 2007.