

Product Information

Anti-PADI4 (N-terminal)

produced in rabbit, IgG fraction of antiserum

Catalog Number **P4749**

Product Description

Anti-PADI4 (N-terminal) is produced in rabbit using as immunogen a synthetic peptide corresponding to amino acids 1-15 of human PADI4, conjugated to KLH via a C-terminal added cysteine residue. The immunizing peptide is specific to human. 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-PADI4 (N-terminal) specifically recognizes human PADI4. Applications include immunoblotting (67 kDa) and immunoprecipitation. Staining of the PADI4 band in immunoblotting is specifically inhibited by the immunizing peptide.

Covalent modifications of the amino-terminal and carboxy-terminal tails of histones, such as phosphorylation, acetylation, and methylation, play a critical role in the regulation of chromatin structure and function. The levels of histone acetylation and phosphorylation in the cell are regulated by pairs of opposing enzymes, such as acetylase/deacetylase and kinase/phosphatase action, respectively. Methylation of arginine and lysine residues is also regulated in a dynamic manner. Arginine methylation has been linked to transcriptional activation. PADs (also known as PADs), peptidyl-arginine deiminases, are a family of enzymes, which convert protein-bound arginine residues to citrulline residues in the presence of calcium ions, a process termed deimination.¹ PAD4 (also designated PADI4 or PAD type IV), one of the members of the PAD family, can, in addition, convert methylated-Arg to citrulline, releasing methylamine.² This deimination process is a mechanism for antagonizing the transcriptional induction mediated by arginine methylation.³ Indeed, PAD4 is recruited to the pS2 promoter (an estrogen responsive gene) following hormone induction when the gene is transcriptionally downregulated.^{2,3} Five types of mammalian PADs 1, 2, 3, 4, and 6 are found. PAD4 was initially named PAD5. However, since it is closely related to rodent PAD4 it was renamed human PAD4. The most noticeable difference between the isotypes is their tissue-specific expression. Expression of the PAD4 gene is tightly linked to myeloid differentiation.

The cDNA encoding human PAD4 was isolated from retinoic acids (RA)-induced HL-60 library.⁴ It is expressed in granulocytes and monocytes and expressed in the nucleus, whereas PAD 1, 2, and 3 are localized in the cytoplasm.⁵ PAD enzymes and their products, citrullinated proteins, are known to play a role in several human diseases, including rheumatoid arthritis (RA). Among the 5 isoforms of PADs, only PAD4 has been identified as an RA-susceptibility gene.⁶

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is 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

Immunoblotting: a working dilution of 1:500- 1:1,000 is recommended using extracts of DMSO-treated HL60 cells.

Immunoprecipitation: 2-4 µL of the antibody immunoprecipitates PADI4 from DMSO-treated HL60 cells.

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

References

1. Vossenaar, E.R., et al., *BioEssays*, **25**, 1106-1118 (2003).
2. Wang, Y., et al., *Science*, **306**, 279-283 (2004).

3. Cuthbert, G.L., et al., *Cell*, **118**, 545-553 (2004).
4. Nakashima, K., et al., *J. Biol. Chem.*, **274**, 27786-27792 (1999).
5. Nakashima, K., et al., *J. Biol. Chem.*, **277**, 49562-49568 (2002).
6. Suzuki, A., et al., *Nature Genet.*, **34**, 395-402 (2003).

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