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ProductInformation

Anti-MeCP2

Developed in Rabbit Affinity Isolated Antibody

Product Number M 9317

Product Description

Anti-MeCP2 is developed in rabbit using a synthetic peptide corresponding to the C-terminus of human MeCP2 (amino acids 465-478) conjugated to KLH as immunogen. The sequence is conserved in rat and mouse. Anti-MeCP2 is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-MeCP2 recognizes MeCP2 by immunoblotting (approx. 75 kDa). Staining of MeCP2 in immunoblotting is specifically inhibited by the MeCP2 immunizing peptide.

Chromatin, the physiological packaging structure of histone proteins and DNA, is a key element in the regulation of gene expression. Histones are subjected to post-translational modifications such as acetylation, phosphorylation, and methylation and play a major role in the regulation of transcription. DNA methylation is the major modification of eukaryotic genomes, which occurs at the fifth position of cytosine in CpG dinucleotide sequences. DNA methylation is associated with transcriptional repression. Silencing of transcription units has been found to occur in genes located on the inactive X-chromosome, in genes silenced by genomic imprinting, and in genes silenced in transformed cell lines and tumors. The DNA methylation system is composed of methyl-CpG-binding proteins as well as of DNA cytosine methyl transferases.

MeCP2 was the first methyl-CpG-binding protein to be isolated. ¹¹ This protein contains a methyl-CpG-binding domain (MBD) and a transcriptional repression domain (TRD). ¹¹ MeCP2 is capable of binding to a single symmetrically methylated CpG pair and was found to bind to chromosomes at sites known to contain methylated DNA. ¹² MeCP2 silences transcription by recruiting the histone deacetylase (HDAC) repressive machinery via recruitment of the Sin 3A corepressor,

thus removing acetyl groups from histones and consequently silencing genes. ¹³ Interestingly, mutations in MeCP2 were found in 65-77% of Rett syndrome patients, an X-linked dominant disorder that results in serious developmental defects. ^{14, 15}

Antibodies reacting specifically with MeCP2 may be used for the study of chromatin remodeling effects on gene expression.

Reagent

Anti-MeCP2 is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 1% bovine serum albumin (BSA) and 15 mM sodium azide.

Antibody concentration: Approx. 0.6 mg/ml

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

For immunoblotting, a working concentration of 0.5 - 1 μ g/ml is recommended using nuclear extracts of MCF7 breast carcinoma cell line.

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

References

- 1. Kornberg, R.D., et al., Cell, 98, 285-294 (1999).
- Strahl, B.D., and Allis, C.D., Nature, 403, 41-45 (2000).
- 3. Bird, A., and Wolffe, A.P., Cell, **99**, 451-454 (1999).
- Razin, A., and Szyf, M., Biochim. Biophys. Acta., 782, 331-342 (1984).
- Nur, I., et al., Nucleic Acids Res., 16, 9233-9251 (1988).

- 6. Li, M., et al., Gene, **301**, 43-51 (2002).
- 7. Razin, A., and Cedar, H., Cell, 77, 473-476 (1994).
- Riggs, A.D., and Pfeifer, G.P., Trends Genet., 8, 169-174 (1992).
- 9. Sakai, T., et al., Am. J. Hum. Genet., **48**, 880-888 (1991).
- Hendrich, B., and Bird, A., Mol. Cell. Biol., 18, 6538-6547 (1998).
- 11. Nan, X., et al., Cell, 88, 471-481 (1997).
- 12. Nan, X., et al., Mol. Cell. Biol., 16, 414-421 (1996).
- 13. Nan, X., et al., Nature, 393, 386-389 (1998).
- 14. Amir, R.E., et al., Nat. Genet., 23, 185-188 (1999).
- 15. Traynor, J., et al., BMC Med. Genet., **3**, 12-27 (2002).

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