

bs-22098R**[Primary Antibody]****MeCP2 Rabbit pAb****BioSS**
ANTIBODIES

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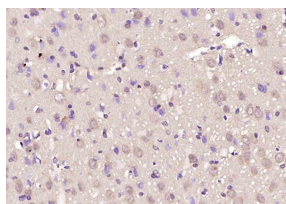
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— DATASHEET —

<p>Host: Rabbit</p> <p>Clonality: Polyclonal</p> <p>GeneID: 4204</p> <p>Target: MeCP2</p> <p>Immunogen: KLH conjugated synthetic peptide derived from human MeCP2: 421-486/486.</p> <p>Purification: affinity purified by Protein A</p> <p>Concentration: 1mg/ml</p> <p>Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.</p> <p>Background: DNA methylation is the major modification of eukaryotic genomes and plays an essential role in mammalian development. Human proteins MECP2, MBD1, MBD2, MBD3 and MBD4 comprise a family of nuclear proteins related by the presence in each of a methyl CpG binding domain (MBD). Each of these proteins, with the exception of MBD3, is capable of binding specifically to methylated DNA. MECP2, MBD1 and MBD2 can also repress transcription from methylated gene promoters. In contrast to other MBD family members, MECP2 is X-linked and subject to X inactivation. MECP2 is dispensible in stem cells, but is essential for embryonic development. MECP2 gene mutations are the cause of some cases of Rett syndrome, a progressive neurologic developmental disorder and one of the most common causes of mental retardation in females.</p>	<p>Applications: IHC-P (1:100-500) IHC-F (1:100-500) IF (1:100-500)</p> <p>Reactivity: Rat (predicted: Human, Mouse, Pig, Sheep, Cow, Dog, Horse)</p> <p>Predicted MW.: 52 kDa</p> <p>Subcellular Location: Nucleus</p>
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— VALIDATION IMAGES —

Paraformaldehyde-fixed, paraffin embedded (rat brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (MeCP2) Polyclonal Antibody, Unconjugated (bs-22098R) at 1:200 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.