Impromptu

Colloquia in Cellular Signaling

Venue: Medical University Vienna, Center for Physiology and Pharmacology,
Institute of Pharmacology, Waehringerstrasse 13a, 1090 Vienna, "Leseraum".
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MeCP2 recognizes cytosine methylated di- and tri-nucleotide sequences to tune transcription in the mammalian brain

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Abstract:

Rett Syndrome is a severe neurological disorder found in approximately 1:10.000 female births. The gene causing most cases of Rett Syndrome has been identified as methyl-CG binding protein 2 (MeCP2) which is an epigenetic reader protein, classically characterized as binding to CpG methylated (mCG) di-nucleotides. Although much research has focused on the binding capacities of MeCP2, its exact mode of action is still controversial. We could recently show that in addition to the classical mCG motif, frequently occurring mCAC tri-nucleotides are also bound by MeCP2. We additionally discover large genomic regions of high mCG + mCAC density that contain neuro-disease relevant genes sensitive to MeCP2 loss or overexpression. Our results re-emphasize MeCP2's original proposed function as a transcriptional repressor whose purpose is to maintain the delicate balance of neuronal gene expression.