

Currently, the possibility that DNA methylation is involved in pathomechanism of epilepsy development is not fully recognized, however there is growing body of evidence showing abnormalities in DNA methylation-related metabolic pathways in experimental models and in human epilepsy. Our recent unpublished data indicate alterations in the expression and functioning of MBD3 protein (a reader of DNA methylation marks) that are triggered by epileptogenic insult. The goal of this study is to disentangle the relationship between seizures, epileptogenesis/epilepsy and MBD3 expression. Our hypothesis is that MBD3 participates in the events leading to the decrease in seizure threshold by acting via epigenetic modification of gene expression. The results of our experiments will bring new data on the role of epigenetic mechanisms involved in epilepsy development. They may bring new ideas about the design of new anti-epileptic or anti-epileptogenic treatments.