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Early-life stress (ELS) has long lasting effects on the brain. Maternal separation in mice persistently altered the offspring's hormonal responses to stress; this included elevated vasopressin (AVP) in the hypothalamus. Treatment with an AVP receptor antagonist was able to reverse the effects of early-life stress. The altered AVP expression was associated with sustained DNA hypomethylation of a region in the AVP gene enhancer that serves as a binding site for the methyl-CpG binding protein 2 (MeCP2). Neuronal activity was able to control the ability of MeCP2 to regulate transcription of the AVP gene and induce epigenetic marking. Thus, ELS can dynamically control DNA methylation in postmitotic neurons to generate stable changes in AVP expression that trigger neuroendocrine and behavioral alterations which are frequent features in depression.