In the central nervous system, local mRNA translation in the synapto-dendritic compartment of neurons controls synaptic plasticity, which is thought to be the cellular/molecular basis of memory formation and consolidation. CPEB, a sequence-specific RNA binding protein, resides at synaptic sites of mammalian hippocampal and other neurons where it mediates translation by regulating mRNA poly(A) tail length. CPEB activity is controlled by phosphorylation, which is a result of synapse stimulation-dependent activation of the kinase Aurora A. CPEB regulates mRNA polyadenylation and translation by nucleating a number of factors including symplekin, a scaffold protein; Gld2, a poly(A) polymerase; PARN, a deadenylating enzyme; and neuroguidin, an eIF4E-binding protein. All of these factors reside in a complex in synapto-dendritic regions. While CPEB knockout (KO) mice show defects in synaptic plasticity and learning and memory, no neurologic function has been ascribed to these CPEB-interacting proteins. Accordingly, lentiviruses expressing shRNAs for Gld2, PARN, and neuroguidin were stereotactically injected into the hippocampus of rats. Two weeks after injection, the animals were sacrificed and the shRNAs were shown to have successfully reduced expression of the mRNAs encoding these proteins. Electrophysiological analysis demonstrated that two of the three knockdowns affected synaptic plasticity, specifically, long term potentiation. The significance of these results for translational control of neurologic function will be discussed.