We recently mapped and validated *Hnrnph1* (heterogeneous nuclear ribonucleoprotein H1) as a quantitative trait gene for reduced methamphetamine (MA) behavioral sensitivity. Mice with heterozygous deletion of a small region in the first coding exon of *Hnrnph1* (*Hnrph1*+/−) showed reduced sensitivity to the stimulant, rewarding, reinforcing effect of MA as well as a decrease in MA-induced dopamine release relative to the wildtype. There is very little known about the mRNA targets in the brain or in vivo function of this RNA binding protein. Given that our data suggested a drug-induced cell biological mechanism by which *Hnrnph1* deletion affects MA neurobehavioral responding, we examined the change in hnRNP H RNA targets in response to MA. We optimized and performed cross-linking immunoprecipitation coupled with high-throughput sequencing (CLIP-seq) to understand hnRNP H-RNA interaction in the striatum (a brain region involved in addiction) of both wildtype and *Hnrnph1*+/− at baseline and in response to an acute dose of MA (2 mg/kg i.p.). Briefly, CLIP-seq involved the use of ultraviolet irradiation to generate covalent bond between RNA and proteins that are in close contact. Antibody specific to hnRNP H was then used to immunoprecipitate the protein-RNA complex followed by RNA extraction and reverse transcription of the extracted RNA into a cDNA library to be sequenced. This is the first CLIP-seq study to examine drug-induced changes in protein-RNA interactions in a specific functionally relevant brain region and will shed light on the molecular mechanism through which hnRNP H regulates methamphetamine-induced dopamine release and addictive behaviors.