While more commonly known as a stimulant and psychedelic, recent literature has indicated 3,4-methylenedioxymethamphetamine (MDMA) as a potential therapeutic for conditions such as post-traumatic stress disorder by attenuating IL-β reactivity, a proinflammatory cytokine, therefore decreasing fear-learning behaviors. Alternatively, MDMA has potential implications for the development of Alzheimer's Disease (AD), a deadly neurodegenerative disease, via increased Amyloid-β plaques, resulting from increased amyloid precursor protein (APP). Additionally, MDMA has been shown to increase cortisol levels, the primary stress hormone derived from corticotropin-releasing hormone (CRH), which has been linked to AD. Before MDMA can further be explored as a PTSD therapeutic, it is imperative to ensure that it will not increase cortisol levels, thereby exacerbating the conditions it is hypothesized to ameliorate, nor increase the propensity for AD. Given this conflicting evidence for the suitability of MDMA as a therapeutic, the current study aimed to provide insights into the effects of MDMA on mRNA expression. As APP and CRH are the most accurate genetic markers for their products, these genes are chosen to elucidate the effects of MDMA at a transcriptional level. The dorsal hippocampus (DH) is the brain region responsible for memory and learning processes, and deficits in both traits are hallmark symptoms of AD. As such, this brain region was selected for investigation. A preclinical model, consisting of 20 male rats, who were acutely administered either saline or MDMA subcutaneously (24 hours and 1 hour before sacrifice), was utilized. Two-step RT-qPCR was then performed to evaluate the effects of MDMA on APP and CRH gene expression in the DH. The results showed that MDMA increased expression of both APP and CRH. The current results support that MDMA could increase the likelihood of AD development through increases in APP and CRH, which have been linked to amyloid plaque formation.