Prostate cancer remains the second leading cause of cancer deaths among American men. Most prostate cancer deaths arise from the advancement of localized tumor cells to distant sites through the circulatory system. Radiation therapy, one of only two curative prostate cancer treatments, utilizes high-energy photons to prompt cell death by inducing DNA damage, particularly double-strand breaks (DSBs). However, radiation resistance of cancer cells poses a significant challenge for treatment. Recent results from our lab has led us to hypothesize that protein arginine methyltransferase-5 (PRMT5) may contribute to radiation resistance through homologous recombination (HR). My research goal was to construct plasmids with genes involved in the regulation of HR, through a process known as molecular cloning. Transformation, restriction enzyme digest, and sequencing confirmed the constructed plasmids were correct. Ultimately, we can apply the plasmids to further experiments in determining whether PRMT5 contributes to radiation resistance of prostate cancer cells through these particular genes, or in other words, regulation of HR. A better understanding of PRMT5’s role in DNA damage response is important in strengthening therapeutic methods to sensitize prostate cancer cells to radiation therapy.