Hypertension is a leading risk factor for heart attacks and end stage kidney failure. The renin angiotensin system (RAS) is a complicated system of proteins that bind to their respective receptors to regulate blood pressure (BP) through the control of sodium excretion. One of these key players is Angiotensin II (AII); it participates in the reabsorption of the filtered sodium from the lumen so it helps control salt balance. Its physiological function is mediated by binding another protein, the Angiotensin II receptor 1 (AT1R). This induces vasoconstriction and aldosterone secretion; both act in concert to raise BP. Several studies helped identify that the blocking of AT1 receptors lowered blood pressure. The expression of AT1R can be controlled at both the mRNA and protein levels. Preliminary studies have suggested that the expression of a minor microRNA, miR-155, negatively correlates with blood pressure in spontaneously hypertensive (SHR) compared with normotensive rats. Sequence data suggest that these small RNAs could possibly control the expression of AT1 proteins. There were four major goals in the proposed research. In part I, the objectives were to develop a Real Time Quantitative Polymerase Chain Reaction (qPCR) method to measure the AT1 receptor and then to use the validated method to measure the expression of AT1R mRNA in SHR male rats versus SHR female rats. Total RNA was isolated from rat kidney using the RNeasy Plus Mini Kit (Qiagen) and its quality and quantity was assessed with a spectrophotometer. Reverse transcription was performed to obtain cDNA from the RNA samples using High Capacity cDNA RT Kit (Life Technologies). The reverse primers, forward primers and probes for the targets; ß-actin and AT1R were designed, and relative quantitation was measured using the ??Ct method. Preliminary data suggested some small differences in the relative expression of AT1 in the female vs. male SHR kidneys. In part II, the question shifted to the possible control of AT1R expression at the protein level rather than at the mRNA level. The objectives were to develop a reverse transcription/qPCR method to measure miR-155 in kidney tissues, and then use this method to measure the expression of miR-155 in the hypertensive male and female rat kidneys. In part II, RNA enriched in miRNA was isolated using the mirVana kit (Life Technologies). The quality of enrichment of small RNAs was analyzed by gel electrophoresis. MicroRNAs were reverse transcribed to cDNA and the cDNA was amplified using Taqman micro RNA Assays and relative expression was measured using U2 snRNA as the endogenous control. Levels of miR-155 in hypertensive females will be compared with miR-155 levels in hypertensive male rats.