Various studies have supported the role of p53 genetic mutation and its influence on micro-RNA expression as driving forces behind lung tumor growth and individual susceptibility to the disease. With this project, I aim to determine which of two p53 mutations (p53-175H and p53-273H) is the most oncogenic, through the investigation of their differential effects on micro-RNA expression and lung tumor growth in mice. I hypothesize that the p53-175H mutant cells will promote the greatest tumor growth and that my study will support previous conclusions of this mutation’s oncogenic function, as well the influence of p53 mutations on micro-RNA expression. The experimental design of this project involves two groups of experimental mice, one exhibiting each strain of p53 mutation, as well as a non-mutant control group. The progression of lung cancer of the two mutant groups will be compared to one another and to the control group in terms of produced tumor size and micro-RNA expression. Analyzing the micro-RNA array of each mouse in every group and the specific researched functions of these micro-RNAs will allow the inference of oncogenic function relative to p53 mutation. Few research studies have focused on the implications of specific p53 mutations on oncogenesis in mice. In the past, relevant studies have more commonly investigated the general role of p53 mutation in influencing micro-RNA expression, the effect of various p53 mutations, and different types of p53 mutations observed in tumor growth. By more closely concentrating on the distinct impacts of two p53 mutations, this project will initiate further progress in the development of medical innovations, treatments, and medications designed for a patient’s specific mutation or genetic predisposition. This project’s applicability to other genetically-influenced diseases is one of its most notable factors, allowing for wide application throughout the fields of public health and healthcare.