Obesity is becoming a growing public health problem in modern society, as fast food becomes more popular and exercise becomes a much lower priority. Obesity paves the way for a higher incidence of many other metabolic disease, including type 2 diabetes and non-alcoholic fatty liver disease (NAFLD). Through the Penn Undergraduate Summer Mentorship (PURM) program, I had the opportunity to work with Dr. Raymond Soccio in his lab, which sought to elucidate the mechanisms by which some people developed obesity and such metabolic diseases more easily than others.

My specific project involved finding different genes that were controlled by a gene called Peroxisome Proliferator Activated Receptor-Alpha (Ppara) that contained strain differing single nucleotide polymorphisms (SNPs) in two different strains of mice commonly used in endocrinology labs. Ppara is a nuclear receptor that is believed to be have heavy influence over fat metabolism and the chemical pathways involved in obesity, and is known to be active during fasting. Thus, looking for strain differential Ppara controlled genes in mice would allow us to confirm or reject the notion that normal genetic variation within a population through SNPs would make some individuals more easily prone to obesity related conditions over others.

Throughout my summer lab experience, I learned many different techniques that would ultimately allow me to reach a conclusion about this notion. These included quantitative polymerase chain reaction (qPCR), pyrosequencing, Bradford assaying, gel electrophoresis, Western blotting, RNA extraction, and cell culture and drug treatment. I also learned how to consult with microarray datasets, ChIP-seq tracks, and RNA-seq data in order to identify genes that were likely targets for Ppara.

Fortunately, my many experiments pointed me toward some promising results. I was able to identify one gene that matched the stated criteria called Cox6b2, which codes for a subunit of the last protein in the electron transport chain in mitochondria (cytochrome c oxidase). The next steps for my research, which I will continue after this summer, will be to measure Cox6b2 and Ppara levels in different cell lines to translate these results from mice to humans.

This research was especially exciting for me because of its vast implications in clinical medicine. Identifying genes with SNPs that played a role in the development of metabolic diseases would...
eventually allow for physicians to tailor their diagnoses and treatments to each individual’s genetic makeup, which is the concept behind personalized medicine. This would likely lead to better prognoses for patients in the future, and it was thrilling to witness and be a part of the biology and chemistry that lay the foundations for this concept in medicine. As an aspiring physician, this lab experience contributed to my education at Penn by giving me a different perspective of the profession that I hope to engage in one day.