Hypothesis: Repeated exposure to tobacco smoke will exacerbate pre-existing systemic hypertension in spontaneously hypertensive (SH) rats while simultaneously inducing classical anatomic lesions analogous to human COPD, including emphysema, small airway remodeling, and excessive mucin secretion. In contrast, normotensive Wistar Kyoto (WKY) rats will not demonstrate elevations in systemic blood pressure and will have less prominent structural changes associated with tobacco smoke exposure.

Proposed research to accomplish: For my project, I was supposed to learn how to do a telemetry device implantation surgery on my own. I was given 6 animals to practice surgery on, and 12 experimental animals that were to be used in the study. Of the 12 animals, 3 were normotensive WKY rats exposed to tobacco smoke, 5 were SH rats exposed to tobacco smoke, and 4 were control SH rats exposed to air. There were 36 other SH and WKY rats in the study, but did not have a telemetry device implanted due to financial constraints. After surgeries were completed, I was to wait 3 weeks for them to recover and establish baseline blood pressure and heart rate values. Once they began tobacco smoke exposure, I had to maintain constant data collection.

Discussion of results—what actually happened: The first speed bump I ran into was that the IACUC amendment had not gone through yet when school ended, so I had to wait until the beginning of July when it was finally approved to start practicing the surgeries. Overall, the surgeries went very well. The first animal unfortunately did not survive, but my other 5 practice animals and 12 experimental animals all survived, and are still alive today. I started exposing them to smoke in the beginning of August. A couple weeks ago, the rats were not doing well, so we decided to halt the smoke treatments for 3 weeks. This essentially changed the project to mimic what would happen if a smoker tried to quit smoking, and after a couple weeks went back to smoking. It will be interesting to see what kind of changes occur with their blood pressures and heart rate, as well as histologically.

So far, with telemetric data we have gathered, we know that baseline systemic measures of blood pressure are significantly different in spontaneously hypertensive (SH) rats and normotensive Wistar Kyoto (WKY) rats. Whereas repeated exposure to tobacco smoke by SH rats resulted in an initial elevation in systolic pressure that was attenuated with continued exposure, WKY rats had significant elevation in systolic pressure during all smoke exposure days. Diastolic pressures were significantly elevated in SH rats with repeated exposure to tobacco smoke. Both SH and WKY rats have significantly reduced mean pulse pressure and increased heart rate due to tobacco smoke exposure, suggesting early signs of heart failure. These findings demonstrate telemetric approaches can be implemented to detect and document novel and progressive cardiovascular impacts of tobacco smoke under experimental settings.