In recent decades much of the research identifying the neural elements of the gut that control propulsive motility in the intestine has focused on the consistent, compliant guinea pig colon; but, as research shifts to transgenic, knock-in, and knock-out mice models, need has grown for a repeatable experimental measure to evaluate colonic motility in mice. My goals this semester were to develop a protocol that allowed for reliable and repeatable evaluation of propulsive motility in the mouse colon, and to test the hypothesis that propulsive motility is disrupted in a mouse model of colitis. Using an instrument called the Gastrointestinal Motility Monitor (GIMM) to monitor motility in the colon, I developed a relatively dependable procedure for evaluating colonic motility and exogenous pellet propulsion in a mouse model *ex vivo*. I established that it is critical to leave the cecum intact, indicating that there may be an important pacemaker in this region that initiates motility patterns. Employing the developed techniques, motility was compared between a control and an experimentally-induced colitis mouse model. Disrupted motility during endogenous clearing and exogenous pellet propulsion was exhibited in the DSS-inflamed colons when compared to the controls, indicating the importance of the myenteric plexus for uniform contraction. This research provides some of the first insights into *ex vivo* colonic motility in mice and establishes a reliable and repeatable experimental method to demonstrate *ex vivo* propulsion.