Background: Extraintestinal Pathogenic *Escherichia coli*, or ExPEC, is a genetically diverse class of bacteria that can survive in various host niches. ExPEC is most commonly found in the GI tract as a commensal, but it can disseminate to the urinary tract and blood and cause disease. *E. coli* has been shown to utilize the mucus layers present within the gut for nutrients, but how ExPEC may consume mucus is currently unknown. A previous genetic screen suggested that the *fbp* gene within the ExPEC cystitis isolate F11 promoted bacterial growth in mucus. The *fbp* gene codes for the Fructose Bisphosphatase (FBPase), the rate-limiting enzyme in gluconeogenesis. Our goal was to determine if and to what extent *fbp* affected ExPEC’s ability to consume mucus and colonize the gut.

Methods: A knockout of the *fbp* gene was created using the Lambda Red Recombinase system. Following the knockout, an *in vitro* assay was utilized that simulates the conditions of the GI tract by growing the bacteria in a mucus-supplemented minimal medium under microaerobic conditions. The bacteria were grown in competition (wild type against the ∆*fbp* mutant), and the cultures were titrated at 24 and 48 hours. An *in vivo* assay was also performed with seven adult Balb/c mice, in which the mice were orally gavaged with a mixture of wild type and ∆*fbp* bacteria and the bacterial counts in the feces were determined over time.

Results/Conclusions: On the second day of the *in vitro* assays, there was a statistically significant drop in bacterial counts for the ∆*fbp* mutant in mucus. This suggests that the *fbp* gene is necessary for F11 to utilize mucus *in vitro*. However, in the *in vivo* experiments using Balb/c mice there was no statistically significant difference between wild type and ∆*fbp* fecal titers, indicating that *fbp* may not be essential for gut colonization by ExPEC.

Future Experiments: The *in vivo* data had a high standard deviation, which may be due to the mouse gut microbiota competing with F11. A potential future experiment is to treat the mice with a broad-spectrum antibiotic (e.g. streptomycin) first, to decrease competitive effects of the microbiota before performing the *in vivo* experiment in order to reduce variability and possibly better resolve mutant phenotypes.