Background

The phenomenon of how bacteria communicate is called quorum sensing (QS), where the receptor histidine sensory kinase QseC causes down stream genetic regulation through the response regulator QseB from the response of autoinducers released from bacteria, or even in response to epinephrine and norepinephrine. We have shown that competition whether based on nutrients or niche, is what drives pathogenesis, and previous research shows the connection of QS with motility, pathogenicity and carbon utilization leading to the indication that the utilization of carbon sources is through QS.

Methods

Since no one had previously measured how QS affects bacterial physiology of *E. coli*, we measured competitive colonization within the murine model and the growth rates of the strain *E. coli* MG1655 Δ*qseC* on individual carbon sources with n-morpholino propane sulfonic acid.

Results

There was a significant competitive advantage for colonization without the sensory kinase QseC. We found that QS is not limited to the induction of virulence and motility as shown in previous studies, but that downstream central carbon metabolism is heavily influenced. Specifically, *E. coli* MG1655 Δ*qseC* has a shorter generation time than the wild type on both catabolite repressing and non-catabolite repressing sugars.

Conclusions

The generation time decrease found with the QseC mutant demonstrates the connection between carbon metabolism, QS, intestinal adaptation, and colonization. Understanding the mechanisms behind QS, will lead to greater understanding of how both commensals and pathogens interact within the intestinal microbiome.