Acinetobacter calcoaceticus is Adept at Colonizing the Gastrointestinal Tract and Stimulating Inflammation

Janiece S. Glover*[1], Brittney D. Browning[1], Taylor D. Ticer[1], Amy C. Engevik [1], Melinda A. Engevik[1]

[1]Medical University of South Carolina, Charleston, SC, USA

Introduction: Inflammatory bowel disease (IBD) is a life-long condition characterized by chronic inflammation of the gastrointestinal tract. IBD consists of two subtypes: Crohn's Disease and Ulcerative Colitis and collectively the disease affects 3 million individuals in the United States. IBD patients have an altered gut microbiota and *Acinetobacter* are one of the groups of microbes that have been shown to be increased in IBD patients. *Acinetobacter* levels significantly correlate with microbial pathways in actively inflamed tissue, suggesting a potential causal relationship between *Acinetobacter* and intestinal inflammation. Analysis of the IBD Transcriptome and Metatranscriptome Meta-Analysis (IBD TaMMA) platform, which houses 3,853 publicly available RNA-Seq datasets from 26 independent studies, revealed that *Acinetobacter calcoaceticus* was one of the top 10 highest elevated bacteria in Crohn's Disease patients. The majority of work with *Acinetobacter* has focused on *Acinetobacter baumannii* and to date no studies have examined *A. calcoaceticus* in the context of the gut or inflammation. We hypothesized that *A. calcoaceticus* would be capable of withstanding the conditions of the gastrointestinal tract, could colonize with other gut microbes and initiate inflammatory signals. **Methods & Results:** Using 2 commercially available strains and 4 clinical isolates, we found that all *A. calcoaceticus* strains were fairly resistant to high osmolarity (0.1, 0.5 and 1 M NaCl), ethanol (1, 2.5, and 5%) and hydrogen peroxide (0.05, 0.1 and 0.2%). In general, the clinical isolates were more resistant than the commercial strains. All strains were able to grow in pH 7, 6, 5 and 4 media; although a reduction in growth was observed at the lower pHs. Biolog phenotypic microarrays in minimal media lacking glucose revealed that all strains could use the following sugars: glucose, L-arabinose, D-galactose, D-mannose, D-fructose, GluNAC, and trehalose. Additionally, all *A. calcoaceticus* strains could colonize human fecal bioreactors; indicating that these microbes could colonize the setting of a complex gut microbiota. Finally, we sought to examine the interaction between *Acinetobacter* and the gut epithelium. Incubation of live *A. calcoaceticus* strains with inside-out intestinal organoids significantly increased pro-inflammatory cytokines (TNF, KC/IL-8, MCP-1 and IL-1α,) and decreased MUC2 and MUC13 transcripts, without altering tight junctions. **Conclusions:** Collectively, these data demonstrate that *A. calcoaceticus* is well adapted to the gastrointestinal tract and points to the potential for *Acinetobacter* to stimulate inflammation and contribute to the pathogenesis of IBD.