Campylobacter spp. infections are now the leading cause of human bacterial gastroenteritis in many developed countries. Following the ingestion of *Campylobacter* spp., the bacterium adheres to and invades the epithelial cells lining the gastrointestinal tract inducing a potent inflammatory response. Moderate to severe diarrhea results and in extreme cases this is a prelude to septicemia, post-infectious arthritis, Guillain-Barré syndrome, or Miller Fisher syndrome. Chickens are a natural host for *Campylobacter* species, and *Campylobacter jejuni* in particular. Transmission to humans most commonly occurs through consumption and handling of chicken meat products contaminated with this pathogen during slaughter and processing. Biofilm formation by *C. jejuni* is essential for its colonization abilities, and a deeper understanding of this process would help to reduce the incidence of *Campylobacter* spp. colonization in commercial poultry and subsequent human infections. Finding solutions to the campylobacteriosis problem has become imperative due to increasing antibiotic resistance in *Campylobacter* spp. and the severity of post-infectious disorders.

We have identified a specific modification in the *C. jejuni* ribosomal RNA that is necessary for the pathogen's ability to form biofilms. From our understanding of this mechanism, we propose that other modifications in the rRNA would also influence biofilm and virulence via modulation of *C. jejuni* protein expression. We propose to initially inactivate *C. jejuni* genes responsible for selected modifications, and follow the effects on biofilm formation and virulence. In mutant *C. jejuni* strains that have become significantly less virulent, we will determine the exact changes in their protein composition that are linked to altered rRNA modification. In this way, we will identify key features of the cellular proteome that are essential for pathogenicity. We believe that targeting rRNA modification is a powerful approach that will reveal cellular components which can be targeted to attenuate the growth, spread and virulence of *Campylobacter* spp.