Non-typhoidal *Salmonella* serovarDublin is primarily associated with self-limiting gastrointestinal illness however, it has adapted to cause invasive disease in humans such as bacteremia and septicemia. In this study, whole genome sequencing (WGS) of human *Salmonella* Dublin strains revealed several virulence factors and mobile genetic elements (MGEs) that may contribute to bacterial virulence and enable the bacteria to cause invasive disease in humans including Gifsy-2 prophage, virulence plasmid and *Salmonella* pathogenicity islands; SPI-7 harbouring the Vi antigen,SPI-6 and SPI-19 harbouring two different T6SSs and the novel pathogenicity island ST313-GI*.* Interestingly, no genomic markers were detected that differentiate among invasive and non-invasive isolates therefore comparative transcriptomics will be carried out to identify differentially expressed genes in invasive strains compared with non-invasive strains aiming to identify novel virulence-attenuated strains with a potential for use as vaccine candidates for high-risk groups including children, elderly and immunocompromised patients.

Interestingly, non-typhoidal Salmonella serovars including Dublin have developed multi-drug resistance (MDR) against current antibiotics. Bacteriophage therapy is therefore the hope for the treatment of MDR bacterial infections however one of the key limitations to therapeutic use of phages, is the limited host range of many phages and the ease of development of bacterial resistance to phages. A solution is to develop one or a cocktail of engineered phage that overcome these limitations. An essential step towards this goal is understanding the complex dynamics of bacteria-phage interaction. We therefore use Anderson phage typing scheme as a valuable model system for study of phage-host interaction to characterize all bacterial antiviral systems (including clustered regularly interspaced short palindromic repeat (CRISPRs) loci and CRISPR-associated (Cas) proteins (CRISPR-Cas) immune systems, superinfection exclusion (Sie) and restriction-modification (R-M) systems) as well as phage evasion strategies (including anti-CRISPR). We aim to get new insights into phage biology and strategies for genetic modification of phages and designing effective broad spectrum engineered phages to overcome the limitations of bacteriophages as therapeutic agents.