**The RNA- Binding Protein ProQ promotes Antibiotic Persistence in *Salmonella***

Rizvanovic, A., Michaux, C., Panza, M., Iloglu, Z., Helaine, S., Wagner, E. G. H., & Holmqvist, E. *mBio* 2022;*13*(6), e0289122.

**Presenter:** Kamryn Escalante **Date/ Time:** 2023/ 09/ 07, 16:10 – 17:00

**Commentator:** Dr. Yi-Lin Cheng **Location:** Room 601, Med College Building

**Background:**

Antibiotic persistence is the presence of two subpopulations that consists of cells that are killed when exposed to antibiotic treatments while another part of the population consists of tolerant cell that are able to survive the exposure of antibiotic treatments. A persister cell is a tolerant cell that originated from a population that has the ability to display antibiotic persistence. Persister cells are dormant and can resume growth. Dormant bacteria are often tolerant to many antibiotics due to their growth arrest. After antibiotic removal, a population of the persisters are reestablished and can once again cause recurrent infections.

RNA- binding proteins (RBPs) plays a role in regulating virulence and stress response genes. In addition, it helps to ensure the infectivity and survival of bacterial pathogens inside their host. The RBP ProQ contributes to several cellar responses in *Salmonella* such as adaptation to chemical stress and bacterial virulence.

**Objective:**

To investigate how ProQ affects *Salmonella* growth and the implications it has on bacterial persistence and pathogenesis.

**Results:**

 Firstly, the authors investigate whether ProQ can affect the growth of *Salmonella.* A competition experiment was done using a combination of Δ*proQ* strain to compete against a wild-type strain. It was observed that the Δ*proQ* strain outcompeted its wildtype which indicates that ProQ can disturb *Salmonella* growth at a population level. Furthermore, the effect of ProQ on *Salmonella* growth was tested by a using a single cell level fluorescence dilution to determine whether ProQ contributes to the formation of growth arrest of the bacteria’s population. Majority of the cells in the population were growing cells, but also non growing cells that are dormant was also detected in the the wildtype and Δ*proQ* strain. On the contrary, the wild type strain revealed a higher fraction of non-growing cells than the Δ*proQ* population. This may signify that ProQ facilitate in the formation of growth arrested cells within the population. Also, it was observed that ProQ promotes persister formation in *Salmonella* in the presence of ciprofloxacin and ampicillin antibiotic treatment. In addition, the authors found that ProQ requires a functional flagellar pathway in order to form persister cells along with the type III protein secretion system (T3SS) that encodes on *Salmonell*a pathogenicity island 2 (SPI-2) contributes to antibiotic persistence. The authors were interested to know whether the same phenotypic effect can be observed *in vivo* and results showed that ProQ promotes persisters cells within *Salmonella* during a macrophage infection.

**Conclusion:** ProQpromotes persister formation in *Salmonella* by activating flagellar synthesis and the SPI-2-encodeded T3SS which can be observed both *in vitro* and *in vivo*.