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**Resolving the Repression Pathway of Virulence Gene hilA in Salmonella**

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**Intro/Background:**
Salmonella is a virulent species of bacteria that causes 1.35 million infections yearly (CDC). As antibiotic resistance becomes an increasingly urgent problem, it is crucial that we develop alternative treatment options.

HilA is the main transcriptional regulator of SPI-1 (UniProt). SPI-1 plays an important role in the invasion of Salmonella into epithelial cells (Frontiers).

Preliminary evidence suggests that the marA protein indirectly represses hilA expression.

**Objective:**
Further resolve the repression of hilA by MarA, by isolating and genotyping mutants that have had this process disrupted.

**Methodology:**
Transposon mutagenesis was conducted on a salmonella strain that overexpresses marA, and expresses hilA with a lacZ reporter. This would introduce a DNA sequence that would disrupt different genes throughout the bacterial genome at random.

These mutants were plated on X-gal media. Any that had a gene involved in hilA repression disrupted would grow blue.

The hilA activity of these mutants was then measured via B-galactosidase assays. Mutations with high hilA expression were reinserted into the wild-type strain via transduction.

**Results:**
Though this project is still in progress, we have identified several mutants that exhibit increased amounts of B-gal activity (and subsequently, hilA activity) compared to the wild-type salmonella strain.

**Conclusion:**
Variable B-gal activity implies there are different routes to repress hilA, and that a pathway may be involved. This data supports evidence that MarA indirectly represses hilA.

Sequencing will allow us to resolve potential genes and gene products that are important for this process.

**References:**