GenomicComparison of Novel Mammiliicoccus and Staphylococcus

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Introduction

The ability to cause disease relies on an organism's ability to invade and successfully proliferate inside a host. *Staphylococcus aureus*, for example, has multiple mechanisms by which to enter the body, such as through epithelial breach, ingestion, and hitchhiking on the insertion of medical tools. Once inside, the bacteria will avoid if not directly attack immune cells, preventing immune response and resulting in collateral tissue damage (Cheung et al, 2021). The following assumption that non-pathogenic species lack disease-causing virulence factors has been argued, resulting in a diminished volume of descriptive literature. In an obvious turn of fate, cases of non-aureus disease have been recorded and are in fact on the rise (França et al, 2021). The bacteria responsible will continue to evolve to become more efficient pathogens, so the understanding of the mechanism of their viral factors will be vital to prevention and treatment. A recently named genus, Mamiliicoccus, used to be classified as Staphylococcus, but following 16S rRNA analysis were moved to their own genus (Madhaiyan et al, 2020).

Research Questions

The close relation of Mamiliicocci and Staphylococci means that their genomes and housed plasmids should be similar. Sequencing from the prior genus can be compared to known sequences of *S. aureus* to identify shared genes and possible shared virulence factors.

Methods

Twelve Mammiliicoccus samples previously gathered by Anand Karki, Ph.D., had been sent for Oxford Nanopore and Illumina sequencing. Returned FASTQs were then trimmed using Porechop for nanopore sequences and Trimmomatic for illumina sequences. Nanopore sequences were high coverage, some samples containing upwards of 130 passes, but after concatenating were merged with forward and reverse Illumina reads using Unicycler. 8 of the samples yielded usable configs. Moving forward, each config will have its plasmids removed. Each plasmid and remaining core genome will be imputed into BLAST to identify existing reference genomes. In addition, genomic data will be input to CARD to identify resistance genes, and through Virulence Factors of Pathogenic Bacteria to identify virulence factors.

Predictions

BLAST of study genomes are unlikely to share homology with known species of Staphylococcus due to its limited documentation. Therefore, primary focus will remain on genomic data gathered from *S. aureus*. Virulence factors should remain relatively across both genera, with some flagmark identifiers missing from study samples. Predicted differences include genes encoding for macrophage avoidance, attachment, and decreased antibiotic resistance. Conversely, a minor amount of novel virulence factor genes are expected, aiding to its continuance despite its decreased infectious adaptability.

References

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