Quorum-sensing (QS) is a cell-cell communication mechanism in bacteria that controls gene expression via secreted signaling molecules. Recently, a new QS mechanism was described in streptococci in which a small hydrophobic peptide (SHP) is produced, secreted, matured and re-imported into the cell to interact with a Rgg-like transcriptional regulator. This interaction controls the activity of Rgg and positively regulates the expression of its targets, among which is shp itself1.

Streptococcus agalactiae or Group B Streptococci (GBS) is a Gram positive bacterium that causes devastating infections mainly in newborns. The Rgg-like RovS transcriptional regulator controls the expression of several GBS virulence factors2. However, SHP was not identified in this previous work and experiments were done in growth conditions that probably inhibited rather than stimulated RovS activity. Consequently, we believe that RovS relevance in the pathogenicity of GBS has been underestimated.

A transcriptional fusion between promoter region of shp and lacZ in strain NEM316 of GBS, was used to identify good growth conditions for RovS targets expression.

The construction of ΔrovS deletion mutant in strain NEM316 showed that Psphp-lacZ fusion is not expressed in this genetic environment, demonstrating that RovS controls positively shp expression.

The addition of synthetic SHP in CDM + glucose 1% induces the expression of Psphp-lacZ fusion, showing that SHP is one of the components of this QS mechanism.

SHP/RovS QS system is involved in the persistence of strain NEM316 of GBS

Virulence results on 6 week-old mice showed that the rovS mutant, injected via intravenous route, displays lower ability to persist in liver after 24 h post-injection than the wild-type strain.

The secretomes of strain NEM316 and ΔrovS mutant were compared using a label free proteomic approach to identify targets regulated by the SHP/RovS QS system.

Identified new target, gbs1556, encodes a secreted protein with unknown function that presents a transglutaminase domain only presents in Streptococcus pyogenes, Streptococcus dysgalactiae and Streptococcus ictaluris, besides all sequenced GBS strains.

SHP/RovS QS system controls the expression of GBS1556 in NEM316 GBS strain

References


Conclusions

1. SHP/RovS QS system is active in GBS.
2. SHP/RovS QS mechanism seems to be involved in the persistence of GBS in vivo.
3. shp and gbs1556 are target genes of this QS mechanism.