

Abstract

The *sil* System of *Streptococcus anginosus*: A Key Factor for Survival in Mixed Cultures [†]

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Quorum sensing is a density-dependent form of communication in bacteria. In streptococci, the quorum sensing system *sil* (*streptococcal invasion locus*) has been associated with virulence and bacteriocin production [1–3]. In this system, the autoinducing peptide SilCR is sensed by histidine kinase SilB, which as a response regulator phosphorylates transcription activator SilA. SilA alters gene expression, causing an increased expression of all *sil* genes, including *silCR*. SilCR is excreted and processed by SilD and SilE [1,4]. In *Streptococcus anginosus*, *sil* regulates the expression of Angicin, a bacteriocin active against various streptococci, listeria, and enterococci [3].

To further characterize the *sil* system in *S. anginosus*, deletion mutants of all *sil* genes were constructed and investigated for their influence on Angicin production. In most strains, the deletion of any gene led to a loss of antimicrobial activity. While a supplementation with synthetic SilCR restored the inhibitory activity of the *silCR* deletion mutant, it had no effect on any of the other mutants. To assess the role of *sil* for bacterial fitness, a *sil* deletion mutant was compared to the wildtype strain in coculture with rival species. In this setting, the bacteriocin-producing wildtype strain demonstrated a significantly enhanced survival, outperforming the *sil* deletion mutant.

Our study is the first to functionally characterize the *sil* system of *S. anginosus*, highlighting its crucial role in bacteriocin production and intra- and interspecies competition.

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