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TYPE II SECRETION SYSTEM: A KEY FACTOR FOR *Serratia marcescens* BACTERIAL COMPETITION

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Serratia marcescens is an opportunistic pathogen with a wide host range that in humans causes nosocomial infections and has a high colonizing capacity. The Type II secretion system (T2SS) is a multiprotein secretion mechanism present in various organisms and often linked to virulence. In our clinical *S. marcescens* RM66262 strain, we identified a chromosomally encoded T2SS. This system is commonly found in clinical isolates but is absent in many non-clinical strains, such as the environmental reference strain *S. marcescens* Db11. Our experimental evidence demonstrated that T2SS plays a role in intra and interspecies bacterial competition. T2SS-deficient mutants showed significantly reduced competitiveness against *Escherichia coli*, *Pseudomonas aeruginosa* and distinct *S. marcescens* strains, suggesting that T2SS is a survival strategy in complex bacterial environments. To understand the regulation of T2SS, we constructed a reporter plasmid containing the upstream region of the T2SS operon fused to GFP. By competition assays using the strain with the reporter plasmid, we determined that T2SS transcriptional expression is induced in response to the attack from species that can actively compete with *S. marcescens*. To further explore the role of the T2SS in bacterial competition, we restored T2SS function in a *gspD*-deficient mutant strain and performed competition assays. The results confirmed that the competitive disadvantage of the mutant was due to the absence of the major GspD secretin, which precluded the assembly of the T2SS complex. In *S. marcescens*, the Type VI Secretion System (T6SS) has already been demonstrated to play a role in bacterial competition. We demonstrate that a double mutant lacking both T2SS and T6SS has no ability to eliminate other bacteria, highlighting that both systems are necessary for full competition capability. Preliminary results also indicated that bacteria-free supernatant from a strain with a functional T2SS could kill prey bacteria, whereas the supernatant from a T2SS-deficient mutant could not, suggesting that the competitive effect is due to secreted effector proteins. Therefore, we performed SDS-PAGE to compare extracellular proteins from the wild-type and the mutant strain and found a differential protein pattern with distinct bands that could correspond to T2SS-secreted proteins. In conclusion, our findings suggest that, in *S. marcescens*, regulated T2SS expression serves as a survival strategy during bacterial competition, thereby enhancing its proliferation capacity across diverse ecological niches.

Palabras clave: *Serratia marcescens* - Bacterial Competition - Type II Secretion System (T2SS) - Effector Proteins - Secretion System