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DISSECTING THE GENETIC BASIS OF SWARMING MOTILITY IN *PSEUDOMONAS AERUGINOSA* BY TRANSPOSON MUTAGENESIS TECHNIQUE

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Swarming motility plays important roles in the pathogenicity of *Pseudomonas aeruginosa*, a frequent opportunistic human pathogen. Bacterial swarming is coordinated by quorum sensing, a cell-to-cell communication mechanism which regulates virulence gene expression depending on cell population density. The global post-transcriptional regulator RsmA is essential for this phenotype. Intracellular levels of the signal molecule c-di-GMP appear to modulate motility and attachment phenotypes, as expressing c-di-GMP phosphodiesterases can compensate a loss of RsmA function. However, the molecular role of this signalling molecule and its interaction with RsmA and quorum sensing system is not yet well understood. It is hypothesized that elevated level of c-di-GMP inhibits swarming in *rsmA* mutants. The major goal of this study was to identify potential c-di-GMP receptors mediating this control. This study identified a set of genes potentially coding for proteins acting as such c-di-GMP receptors by using random transposon mutagenesis technique. Four genes disrupted by transposon insertion were found to restore swarming in an *rsmA* mutant: *rhlC*, encoding for a rhamnosyltransferase involved in the production of rhamnolipd biosurfactants; *mexT*, a transcriptional regulator controlling multi-antibiotic efflux systems, *cupA3* encoding a fimbrial biogenesis usher protein involved in surface attachment and PA3866 encoding a bacteriocin. In addition, the investigation of the roles of a variety quorum sensing signalling molecules on the restoration of swarming motility in a signal-less mutant was carried out. It was found that the fatty acid chain lengths of N-acyl-homoserine lactones (AHLs) have important roles in controlling the swarming network. AHLs with long fatty acid chain lengths were not found to act as biosurfactants during swarming.

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