Pseudomonas aeruginosa cupD fimbrial genes in PAPI-1 are regulated by the RcsCB phosphorelay system

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Auto-agregation and adhesion of bacterial cells are important steps in the colonization of surfaces. The presence of multiple chaperone-usher fimbriae coding genes in various *Pseudomonas aeruginosa* strains may contribute to the success of this opportunistic pathogen in colonizing different substrates. However, little is known about the specificity and the regulation of expression of such appendages. We asked whether expression of *cupD*, a gene cluster present in the large pathogenicity island of strain PA14, is controlled by the *rcsCB* regulatory genes that were likely inserted in the chromosome in the same recombination event. Overexpression of the response regulator RcsB in PA14 promotes biofilm formation in glass tubes, due to higher *cupD* transcript levels from a transcription start site at position -31 from *cupD1* first codon. Short appendages were observed by transmission electron microscopy in cells overexpressing RcsB and are likely composed by CupD subunits, since the mRNA levels of other fimbrial genes are unaffected in those cells. The hybrid sensor histidine kinase RcsC has a negative effect on *cupD* mRNA levels in laboratory conditions, suggesting that it may act as a phosphatase in the absence of a specific signal, justifying the low basal *cupD* expression levels in wild-type cells. Unlike other *cup* gene clusters, *cupD* transcription is not affected by the MvaT HN-S protein, and does not seem to be controlled by PvrR, a response regulator with an EAL domain, coded in PAPI-1. These data supports the thought that expression of each *P. aeruginosa* Cup fimbriae depends on specific regulatory proteins, probably respondind to distinct environmental signals.

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