
Molecular mechanisms of virulent bacteriophages infecting *Pseudomonas aeruginosa*

Laurent Debarbieux*¹, Anne Chevallereau , Mathieu De Jode , Elsa Brambilla , and Gouzel Karimova

¹Molecular Biology of Gene in Extremophiles Unit – Institut Pasteur de Paris – France

Résumé

Pseudomonas aeruginosa bacteriophages PAK_P3 and PAK_P4 belong to two distantly related genera of *Caudovirales* viruses. Outside the cluster of genes coding for structural proteins that are almost identical, the rest of the genome is highly divergent. Nevertheless, few open reading frames display a strong conservation within bacteriophages belonging to these two genera suggesting they may code for conserved functions. In this work we examined one of these conserved ORFs which displays a phenotype of growth defect when expressed ectopically in the bacterial host. Interestingly, this phenotype was also observed when expressed in two other *P. aeruginosa* hosts as well as in *Escherichia coli*. Using a two-hybrid system in *E. coli* we identified the anti- σ E factor RseA as a putative interacting partner of the product of this ORF. The *P. aeruginosa* homologous protein of RseA is named AlgU, while the σ E factor is named MucA. Both AlgU and MucA are well studied proteins in *P. aeruginosa* as being associated to the general stress response that regulates in particular the biosynthesis of alginate, which is linked to the mucoid phenotype often found in clinical isolates from cystic fibrosis patients. We experimentally verified that the product of this ORF is interacting directly with AlgU and are proposing a mechanism used by the bacteriophage to modulate the stress response in order to achieve its infectious cycle.

*Intervenant