The natriuretic peptide hormones prevent *Pseudomonas aeruginosa* biofilm formation through a specific bacterial target.

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We have shown that the C-type Natriuretic Peptide (CNP), a peptide produced in the lung, reduces *Pseudomonas aeruginosa* biofilm formation. Herein, we clarified the effect of CNP and studied the effect of two other members of the natriuretic peptide family, the atrial natriuretic peptide (ANP) and the brain natriuretic peptide (BNP) on *P. aeruginosa* biofilm.

We observed that exposure of *P. aeruginosa* to graded concentrations of ANP, CNP or BNP (10⁻⁹ to 10⁻⁶ M) induced a dose-related inhibition of biofilm formation under dynamic conditions. In parallel, we showed that static biofilms formed by *P. aeruginosa* are impacted by BNP and CNP but more modestly as compared with the dynamic conditions. An *in silico* study comparing protein sequences of human natriuretic peptide receptors and *Pseudomonas* proteins revealed that the bacterial protein AmiC could be the sensor for natriuretic peptides. Using MicroScaleThermophoresis, we showed that both CNP and ANP bind AmiC with a K_D of 2 μM, whereas BNP has no affinity for AmiC. Interestingly, an *amiC* mutant strain was not impaired in biofilm formation in the presence of CNP at 10⁻⁷ M or less, while it remained sensitive to BNP.

We demonstrate that the natriuretic peptides strongly prevent biofilm formation through different mechanisms. Indeed, we showed that CNP and ANP which are the more expressed peptides in the lung act specifically through the AmiC sensor, whereas the BNP peptide seems to possess a non specific activity. This discrepancy should have major consequences to design drugs for biofilm treatment.

CLAMENS T., DESRIAC F., ROSAY T., CREPIN A., DUFOUR A., CORNELIS P., BOUFFARTIGUES E., CHEVALIER S., FEUILLOLEY M.G.J., LESOUHAITIER O.

The involvement of the *ami* operon in *Pseudomonas aeruginosa* virulence regulation and biofilm formation reveals new functions for the amidase AmiE. *16*th international conference on *Pseudomonas*, Liverpool, UK, September 5-9, 2017.

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DESRIAC F., CLAMENS T., ROSAY T., ENAULT J., ROQUIGNY L., BAZIRE A., DUFOUR A., **RACINE P.J., CORNELIS P., BOUFFARTIGUES E., CHEVALIER S., FEUILLOLEY M.G.J., LESOUHAITIER O**. The natriuretic peptide hormones prevent *Pseudomonas aeruginosa* biofilm formation through a specific bacterial target. *16*th international conference on *Pseudomonas*, Liverpool, UK, September 5-9, 2017.