# Séminaire

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CONFÉRENCIER INVITÉ

**Vendredi 30 Novembre 2018 à 11h**

Salle des séminaires

Institut de biologie structurale - 71 avenue des Martyrs CS 10090 38044 Grenoble Cedex 9 - T.+33 (0)4 57 42 85 00

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## The T6SS (Type Six Secretion System) of *Pseudomonas aeruginosa*, an anti-eukaryotic and antibacterial weapon

*Pseudomonas aeruginosa* encodes three Type Six Secretion Systems (T6SS). They are independent and inject effectors to affect various components in the target cell. While H1-T6SS is so far only involved in antibacterial activity, H2-T6SS and H3-T6SS are able to attack both prokaryotic and eukaryotic cells. Altogether, T6SSs of P. aeruginosa are important to fight other bacteria for an ecological niche including presumably the lungs, and are essential in the pathogenicity process.

We demonstrated that H2-T6SS promotes the internalization of *P. aeruginosa* into non-phagocytic cells. Although considered an extracellular pathogen, P. aeruginosa is able to transiently invade epithelial cells by hijacking host kinases. We then identified the evolved VgrG2b as an H2-T6SS effector4. Once injected into epithelial cells, the C-terminal extension of VgrG2b targets the gamma-tubulin ring complex, a microtubule-nucleating multiprotein complex. Remarkably this interaction is followed by a microtubule-dependent internalization of the pathogen. Our observations document one of the first mechanisms of a bacterial pathogen whose entry is mediated by the microtubule network. Indeed, most pathogens modulate the actin cytoskeleton of eukaryotic cells to facilitate the invasion of host cells.

Moreover we broadened the effector role of VgrG2b to a structural component of the H2-T6SS during its antibacterial function by showing that VgrG2b is required for the delivery into target bacteria of Tle3, a novel lipolytic toxin. We are currently deciphering the Tle3 secretion process mechanism.

*Hôte : Cécile Morlot (IBS/Groupe Pneumocoque)*

L'accès au campus EPN nécessite un avis de rendez-vous
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