Social behavior of Bacillus subtilis and Dickeya solani

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Abstract

Most natural environments harbor a stunningly diverse collection of microbial species, within these communities, bacteria communicate among themselves to carry out a wide range of complex social behaviors and win the 'survival game'. The mechanism of competition is mainly connected with producing, releasing, detecting, and responding to signaling compounds, enzymes and other interfering metabolites. Understanding the mode of action of those factors is crucial for biological control of plant diseases. Considering the ongoing reinforcement of the integrated plant protection policy in Europe, biological control of plant by non-pathogenic bacteria is a very promising alternative to the extended use of chemical agents.

My project aims to explain, on molecular level, the mechanism of interaction between the non-pathogenic *B. subtilis* MB73/2 and the plant pathogen *D. solani* IFB0102. Bacteria from *Dickeya* genera contribute to significant economic losses in potato plantation, these pathogens produce a variety of cell-wall degrading enzymes which are responsible for black-leg and soft-rot potato disease. This activity is regulated by AHL-based quorum sensing which activates genes involved in various aspects of bacteria physiology. Therefore, interference with this mechanism has potential as a means of controlling bacterial-mediated plant diseases.

First step of work focused on bacteria swarming motility, a flagella-driven directional movement which is quorum-sensing regulated. Inoculating both bacteria strains on two different points in the same Petri plate, *B. subtilis* MB73/2 spread all over the plate while IFB0102 is surrounded by a clear inhibition zone and directionally escape over the surface. This suggests a strong antagonism imposed by *B. subtilis* on *D. solani*. For swarming motility, *B. subtilis* MB73/2 requires surfactin, a surface-active compound with antibiotic activity. To rule out any effects of this compound on *D. solani*, we constructed a *B. subtilis* MB73/2 mutant strain with inactivation of *sfp* gene. Our results showed Dickeya is resistant to surfactin. Moreover, the mutant strain repeated the phenotype, suggesting that other factors are responsible of this interaction. To identify factors involves in this phenomenon, we are currently performing random mutagenesis of *B. subtilis* MB73/2 strain: mutants which don't show any growth inhibition or antagonistic effect will be selected and the whole-genome sequencing of selected mutants will be used to identify the genes responsible of the observed phenotype.