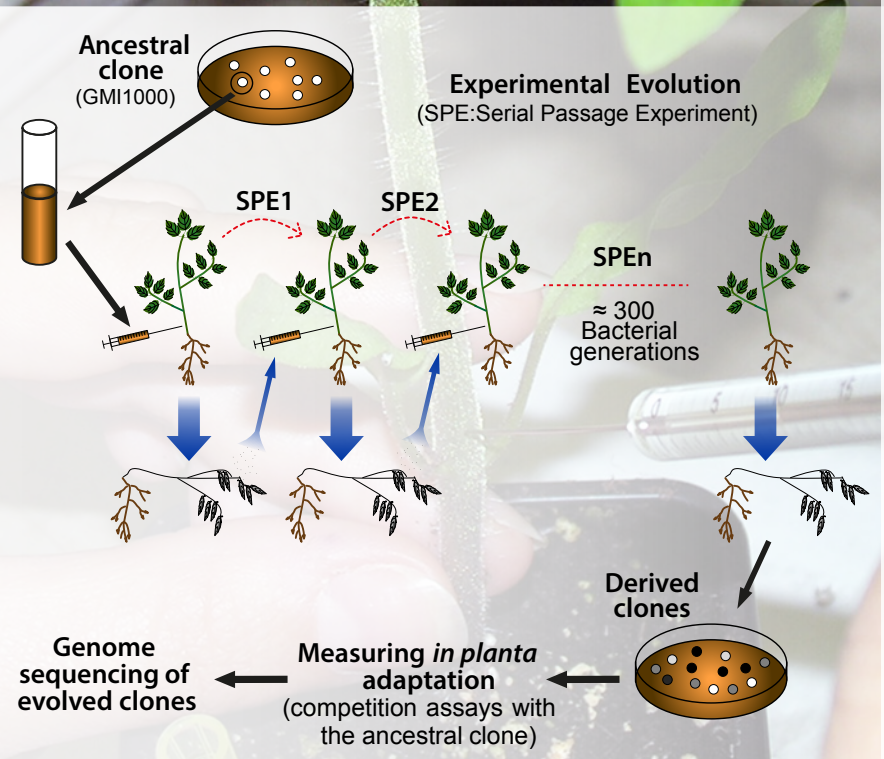
Multihost experimental evolution of the pathogen *Ralstonia solanacearum* unveils genes involved in adaptation to plants

Ralstonia solanacearum, the causal agent of a lethal bacterial wilt plant disease, infects an unusually wide range of hosts. These hosts can further be split into plants where *R. solanacearum* is known to cause disease (original hosts) and those where this bacterium can grow asymptomatically (distant hosts). Moreover, this pathogen is able to adapt to many plants as supported by field observations reporting emergence of strains with enlarged pathogenic properties.

To investigate the genetic bases of host adaptation, we conducted evolution experiments by serial passages of a single clone of the pathogen on three original and two distant hosts over 300 bacterial generations and then analyzed the whole-genome of nine evolved clones. Phenotypic analysis of the evolved clones showed that the pathogen can increase its fitness both on original and distant hosts although the magnitude of fitness increase was greater on distant hosts. Only few genomic modifications were detected in evolved clones compared to the ancestor but parallel evolutionary changes in two genes were observed in independent evolved populations. Independent mutations in the regulatory gene efpR were selected for in three populations evolved on beans, a distant host. Reverse genetic approaches confirmed that these mutations were associated with fitness gain on bean plants. This work provides a first step towards understanding the within-host evolutionary dynamics of *R. solanacearum* during infection and identifying bacterial genes subjected to in planta selection. The discovery of *EfpR* as a determinant conditioning host adaptation of the pathogen illustrates how experimental evolution coupled with whole-genome sequencing is a potent tool to identify novel molecular players involved in central life-history traits.



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