Role of the acquisition of a Type 3 Secretion System in the emergence of novel pathogenic strains of Xanthomonas

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Résumé en anglais	Cases of emergence of novel plant pathogenic strains are regularly reported that hinder yield of crops and trees. However the molecular mechanisms underlying such emergence are still poorly understood. The acquisition by environmental non-pathogenic strains of novel virulence genes by horizontal gene transfer has been suggested as a driver for the emergence of novel pathogenic strains. In the present study, we tested such an hypothesis by transferring a plasmid encoding the Type 3 Secretion System (T3SS) and four associated Type 3 Secreted proteins (T3SPs) to the non-pathogenic strains of Xanthomonas CFBP 7698 and CFBP 7700, that lack genes encoding T3SS and any previously known T3SPs. The resulting strains were phenotyped on Nicotiana benthamiana using chlorophyll fluorescence imaging and image analysis. Wild-type non-pathogenic strains induced a HR-like necrosis, while strains complemented with the T3SS and T3SPs suppressed it. Such suppression depends on a functional T3SS. Among the T3SPs encoded on the plasmid, Hpa2, Hpa1, and to a lesser extend XopF1, collectively participate to the suppression. Monitoring the population sizes in planta showed that the sole acquisition of a functional T3SS by non-pathogenic strains impairs growth inside leaf tissues. These results provide functional evidence that the acquisition via horizontal gene transfer of a T3SS and four T3SPs by environmental non-pathogenic strains is not sufficient to make strains pathogenic. In the absence of canonical effector, the sole acquisition of a T3SS seems counter-selected, and further acquisition of type 3 effectors is probably needed to allow the emergence of novel pathogenic strains.
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