Abstract

Xanthomonas axonopodis pathovar citri (Xac) causes bacterial citrus canker, a serious disease of most citrus species. Xanthomonas campestris pv. campestris (Xcc) is the causal agent of black rot disease in cruciferous plants. In Xcc, cell-cell signaling is mediated by diffusible signal factor (DSF). Synthesis of DSF depends on RpfB and RpfF. DSF perception and signal transduction have been suggested to involve a two-component system comprising RpfC and RpfG. It has been proposed that these proteins participate in a signal transduction system linking changes in the environment to the synthesis of DSF and the expression of virulence genes. Although the cluster of the rpf genes in Xac has synteny with the corresponding cluster in Xcc, two genes (rpfH and rpfI) are absent in Xac. To investigate DSF-mediated regulation during Xac-Citrus limon interaction, we constructed two strains of Xac, one with a mutation in the rpfF gene, leading to an inability to produce DSF, and one with a mutation in the rpfC gene leading to an overproduction of DSF. These mutants also show decreased levels of extracellular cyclic β-(1,2)-glucans and decreased production of endoglucanase and protease extracellular enzymes. The Xac DSF-deficient rpfF and the DSF-hyper producing rpfC mutants are both severely compromised in their ability to cause canker symptoms in lemon leaves compared to the wild-type. Here we provide evidence that rpf genes in Xac are involved in controlling virulence factors mediated by DSF.

Keywords

canker, DSF, extracellular polysaccharide, quorum sensing