

The Effector Protein PthA from *Xanthomonas citri* Shows DNA Binding Properties

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Citrus canker, caused by the bacterium *Xanthomonas axonopodis* pv. *citri* (Xac), is one of the most important diseases of citrus and is characterized by the formation of pustules-like lesions on the host surfaces, as a result of intense cell enlargement and division. The precise molecular mechanism by which Xac promotes cell hypertrophy and hyperplasia is not known. However, it has been shown that members of the PthA/AvrBs3 family of transcription factors are required to elicit canker. Through a series of microarray experiments employing cycloheximide (CHX), we were able to indentify major sweet orange genes that were strongly up regulated by Xac, early during infection. In particular, we highlight the up-regulation of pathogenesis-related (PR) proteins, transcription factor WRKY, chitinase and cyclophilin, which are either involved in defense responses or disease development. Notably, the promoter regions of such genes have TATA box-like sequences that are remarkably similar to the recently identified upa boxes found in the promoters of pepper genes directly bound and transactivated by AvrBs3, an effector protein from *Xanthomonas campestris* pv. *vesicatoria* that is 95% identical to PthA. We found however that the recombinant PthA proteins made in bacteria bound to all citrus promoters with no clear specificity to the TATA box-like sequences. Attempts to verify whether PthA proteins require host factors for specificity to DNA targets are in progress.

Key-words: *Xanthomonas citri*, citrus canker, PthA proteins

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