

The complete genome of *Xanthomonas albilineans* provides insights into pathogenicity of this sugarcane pathogen and allows further assessments of the large diversity within this species

Isabelle Pieretti¹, Stéphane Cociancich¹, Valérie Barbe², Sébastien Carrere³, Ralf Koebein⁴, Patrice Champoiseau¹, Arnaud Couloux², Armelle Darrasse⁵, Jérôme Gouzy³, Marie-Agnès Jacques⁵, Emmanuelle Lauber⁶, Charles Manceau⁵, Sophie Mangenot², Mélanie Marguerettaz⁷, Stéphane Poussier⁷, Béatrice Segurens⁴, Boris Szurek⁴, Valérie Verdier⁴, Matthieu Arlat⁸, Dean W Gabriel⁹, Philippe Rott¹, Monique Royer¹

(1) CIRAD, UMR BGPI, F-34398 Montpellier Cedex 5, France. (2) Génoscope, Centre national de séquençage, F-91057 Evry Cedex, France. (3) INRA, UMR LIPM, F-31326 Castanet-Tolosan Cedex, France. (4) IRD, UMR RPB, F-34394 Montpellier cedex 5, France. (5) INRA, UMR PaVé, F-49071 Beaucouzé, France. (6) CNRS, UMR LIPM, F-31326 Castanet-Tolosan Cedex, France. (7) Agrocampus Ouest centre d'Angers, UMR PaVé, F-49071 Beaucouzé, France. (8) Université Paul Sabatier, UMR LIPM, F-31326 Castanet-Tolosan Cedex, France. (9) University of Florida, Plant Pathology Department, Gainesville 32605, U.S.A.

contact: pieretti@cirad.fr



INTRODUCTION and OBJECTIVES

Xanthomonas albilineans is the causal agent of leaf scald, a lethal disease of sugarcane



Leaf scald symptoms, including white foliar stripes and bleaching caused by albicidin that blocks chloroplast differentiation

Xanthomonas albilineans, unlike other xanthomonads, is a xylem-limited pathogen which:

- produces the toxin albicidin, a potent DNA gyrase inhibitor
- experienced a genome erosion, lacks the *gum* gene cluster (xanthan) and the T3SS of the Hrp injectisome families [1]
- possesses a T3SS of the SPI-1 family [1-2]

Xanthomonas albilineans strains show a high genetic diversity:

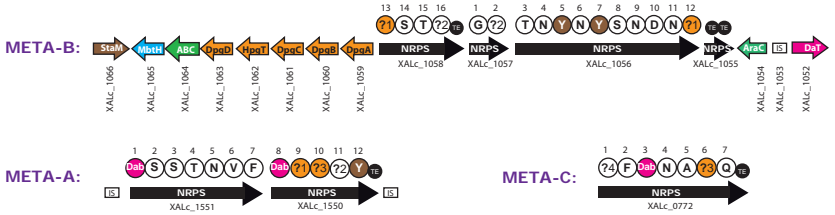
- 10 genetic groups identified by Pulsed Field Gel Electrophoresis (PFGE) [3]

All strains involved in disease outbreaks since the late 1980s and reported in several locations belong to the same genetic group, called PFGE-B. We used the genome sequence of strain GPE PC73 to describe specific pathogenicity-related features of *X. albilineans* and to further investigate the large diversity of this species.

RESULTS

X. albilineans encodes 10 large non ribosomal peptide synthetases genes (NRPS) grouped in 4 loci:

XALB1: albicidin biosynthesis gene cluster (3 NRPS genes previously described)

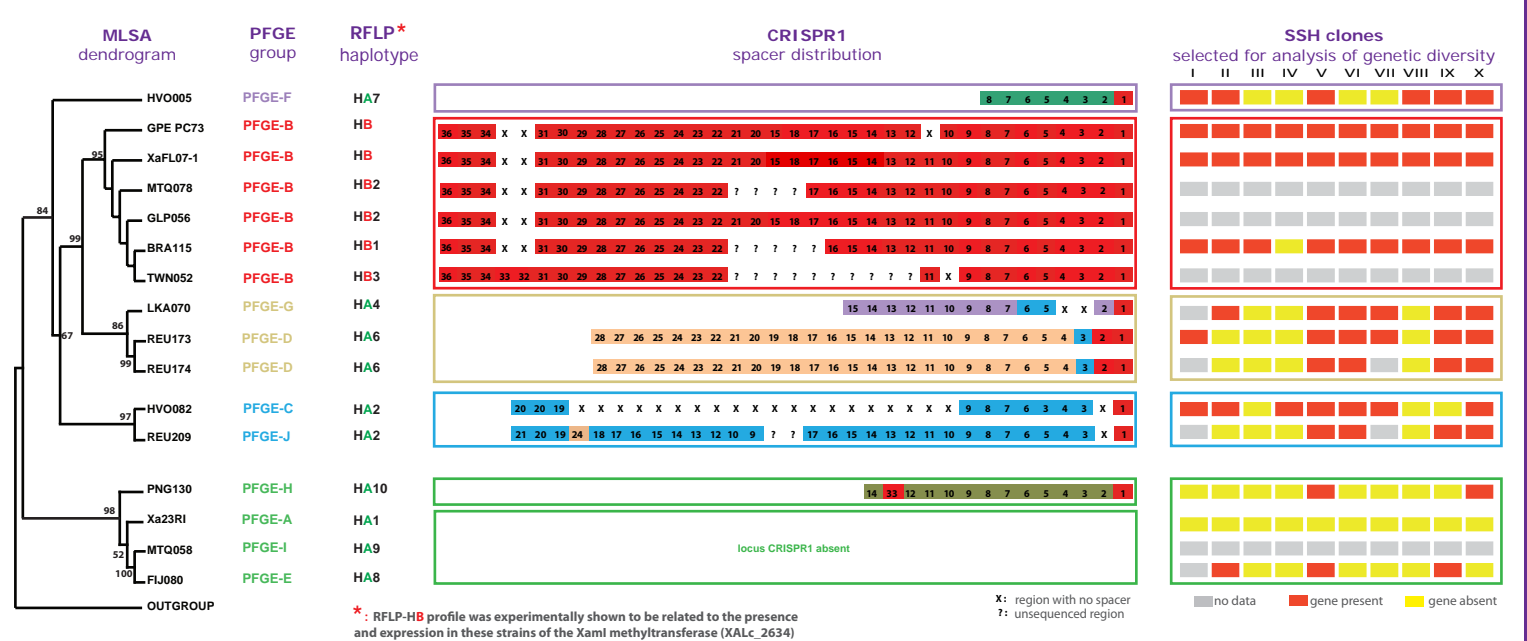


The NRPS genes are represented by black arrows, length of arrows is not proportional to the length of genes. NRPS modules are represented by circles. Large circles indicate complete NRPS modules (domains C, A and PCP). The amino acid predicted to be assembled by the corresponding module is indicated within each circle. The chronological order of amino acid incorporation is illustrated by the number above each circle. Small circles represent TE domains. Brown arrow: gene required for hydroxylation of tyrosine. Pink arrow: gene required for biosynthesis of Dab (2,4-Diaminobutyric acid). Orange arrows: genes required for biosynthesis of unknown substrates similar to Dpg (3,5-Dihydroxyphenyl-glycine). Brown circle: NRPS module predicted to be specific for hydroxytyrosine. Pink circle: NRPS module predicted to be specific to Dab. Yellow circle: NRPS module predicted to be specific of Dpg. Orange circles with ?1, 72, 73 and 74 indicate four different unpredicted amino-acid substrates.

X. albilineans encodes 5 cell wall degrading enzymes which harbour a long linker and a cellulose binding domain (CBD) at their C-termini which suggests that *X. albilineans* is adapted to the utilization of cell breakdown products as a carbon source:

Name	Accession	Linker size in amino acid (Aa)
Endoglucanase - EngXCA	XALC_2969	96 Aa CBD
	XALC_2967	26 Aa CBD
Cellobiosidase - CbhA	XALC_0484	152 Aa CBD
Cellulase - Cel5	XALC_0865	134 Aa CBD
Putative cell wall degrading enzyme	XALC_0874	62 Aa CBD

Large inter-strain variability in *X. albilineans* was confirmed using multi-locus sequence analysis (MLSA), spacer analysis of one clustered regularly interspaced short palindromic repeats (CRISPR1) and analysis of the diversity of suppression subtractive hybridization (SSH) markers



CONCLUSION AND PERSPECTIVES

X. albilineans is a highly distinctive xanthomonad harbouring specific cell wall degrading enzymes with long linkers and CBD suggesting its adaptation for the degradation of cell-wall breakdown products present in the xylem of sugarcane. NRPS genes are good candidates to produce small molecules potentially involved in interactions with host sugarcane cells and these molecules are currently being identified and characterized. MLSA analysis, coupled to analysis of SSH markers and spacer content in CRISPR1, revealed a large inter-strain variability in *X. albilineans*. No pathogenicity-related gene specific to PFGE-B strains was isolated by SSH. Further analysis of the XamI methyltransferase will be necessary to investigate its putative role in pathogenicity of PFGE-B strains associated with sugarcane leaf scald disease outbreaks.

REFERENCES

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