CRISPR-associated sequence diversity within *Xanthomonas albilineans*, the causal agent of leaf scald of sugarcane

Isabelle Pieretti[1], Mélanie Marguerettaz[1], Stéphanie Bolot[2], Sébastien Carrere[2], Stéphane Cociancich[1], Jérôme Gouzy[2], Philippe Rott[1], Monique Royer[1].

(1) CIRAD, UMR BGPI, F-34398 Montpellier Cedex 5, France. (2) INRA, UMR LIPM, F-31326 Castanet-Tolosan Cedex, France.

contact: pieretti@cirad.fr

INTRODUCTION and OBJECTIVES

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

white foliar stripes — bleaching — necrosis

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

***Xanthomonas albilineans*, unlike other xanthomonads, is a xylem-inviding 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]

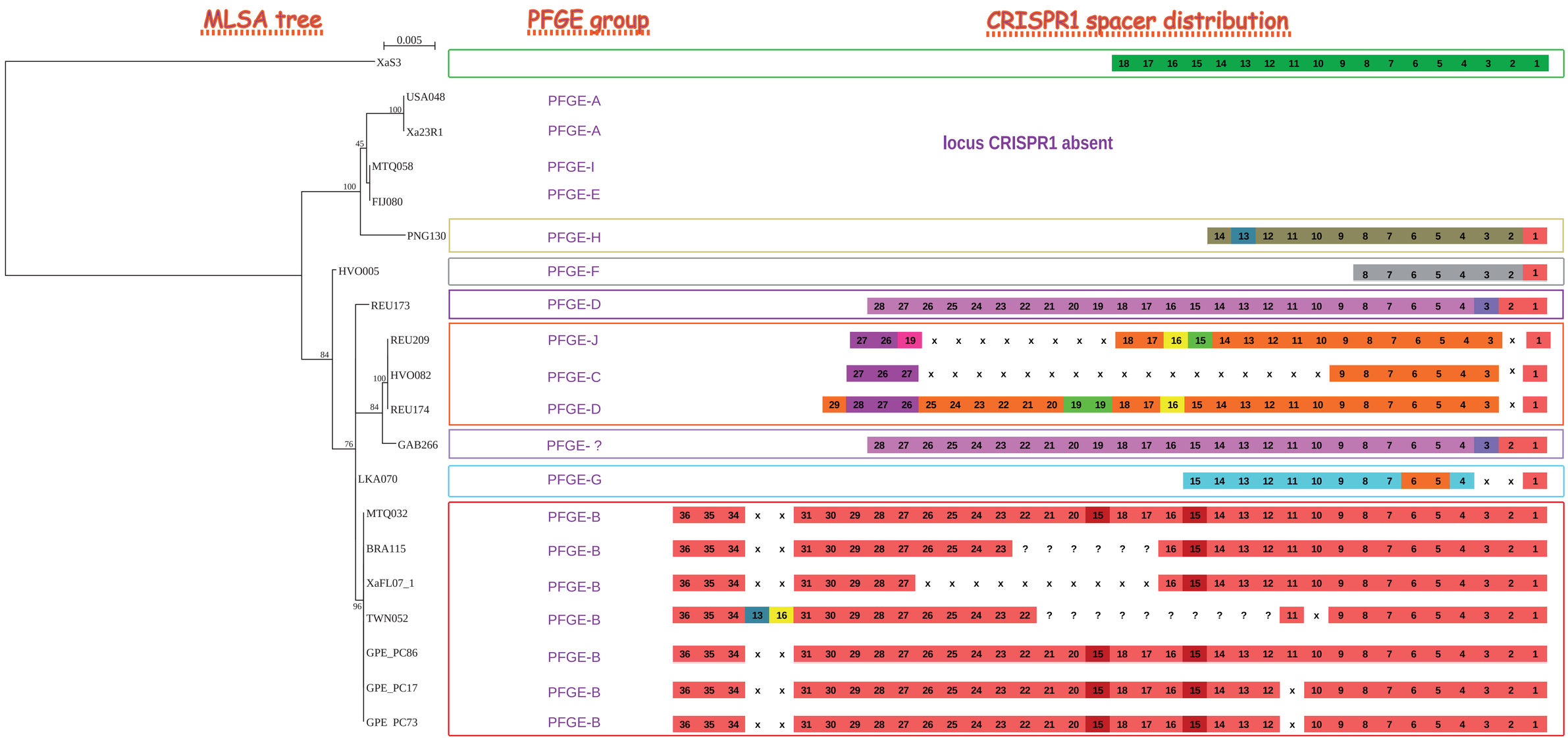
***Xanthomonas albilineans* strains show a high genetic diversity:**

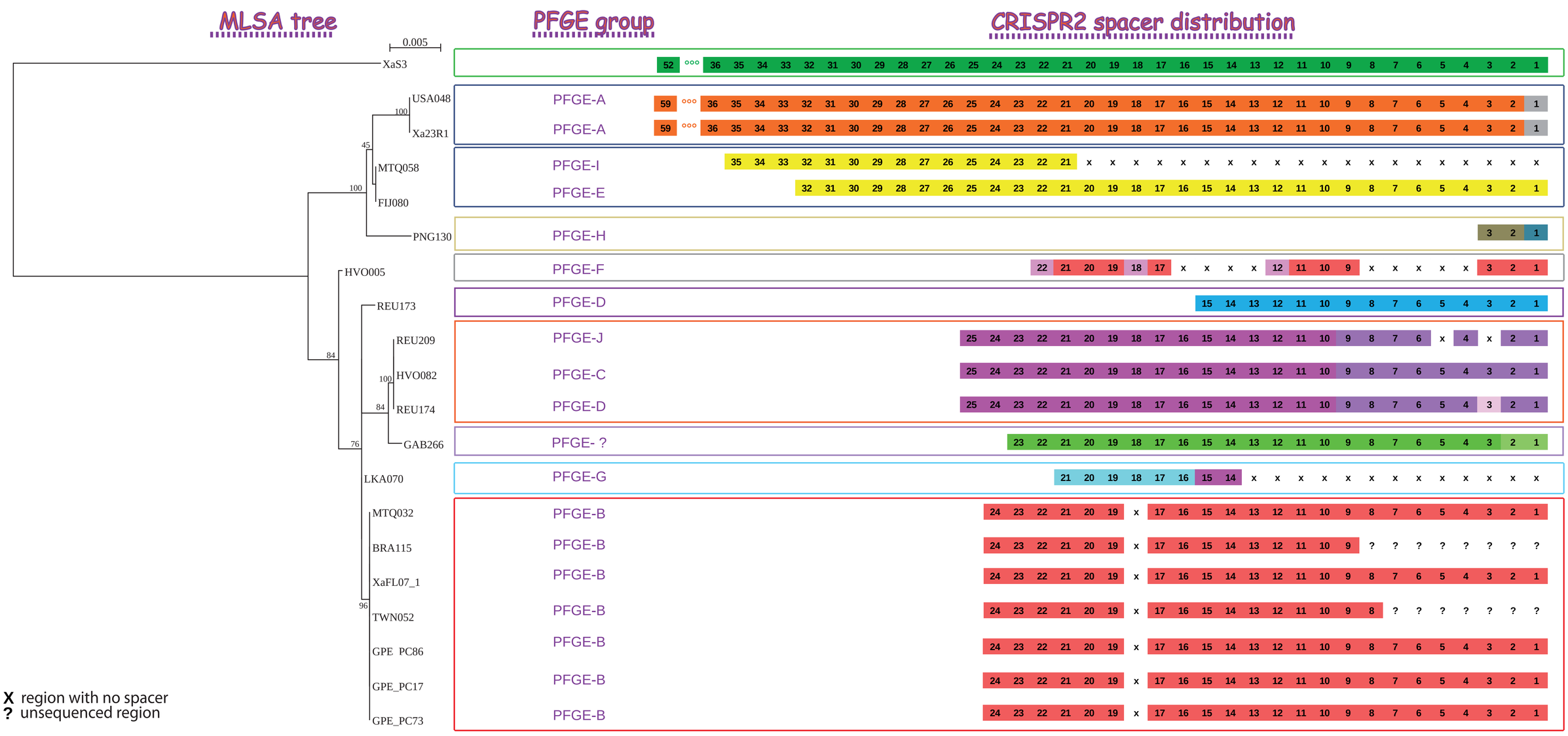
- 10 genetic groups identified by Pulsed Field Gel Electrophoresis (PFGE) [3]
- phylogenetic relationships between PFGE groups established by Multi Locus Sequence Analysis (MLSA) [2].

All strains involved in disease outbreaks since the late 1980s and reported in several locations belong to the same genetic group, called PFGE-B. The genome of *X. albilineans* contains two different CRISPR/cas systems (loci CRISPR1 and CRISPR2, respectively). Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) are composed of repeats separated by spacers. Analysis of the variability of these spacers is curently used to perform diversity or epidemiological studies in bacteria.

RESULTS

Analysis of the inter-strain variability of CRISPR-spacers in *X. albilineans* (CRISPR1 and CRISPR2) :





X region with no spacer
? unsequenced region

CONCLUSIONS

A large inter-strain variability of the CRISPR-spacers was observed not only between phylogenetically distant strains, but also between closely related strains. The variability is congruent with MLSA phylogenetic analysis, and CRISPR markers appear more polymorphic and discriminent than PFGE markers. Interstingly, analysis of the CRISPR1-spacer variability appears as a promising tool to study the phylogeny of PFGE-B strains and to better understand the occurence of the disease outbreaks since the late 1980s.

REFERENCES

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(3) Davis et al., 1997. Phytopathology, 87:316-324.