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The Revisited Infection Cycle of *Xanthomonas albilineans*, the Causal Agent of Leaf Scald of Sugarcane

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ABSTRACT

Leaf scald, a sugarcane disease caused by *Xanthomonas albilineans*, has been largely studied since its discovery in 1911. Numerous data were obtained and reported, from isolation, transmission and diagnosis of the pathogen to the complete sequence of its genome. Although *X. albilineans* was thought to be exclusively transmitted by infected cuttings and contaminated cutting implements for more than 80 years, the existence of an epiphytic life of this pathogen was reported in the late 1990s. We highlight herein the role of the epiphytic populations of *X. albilineans* in plant and field contamination and the impact of environmental conditions, especially rainfall, on these populations of the pathogen. Data obtained from experimental research in Guadeloupe showed the capacity of *X. albilineans*, when rainfall is sufficient, to spread by aerial means within the sugarcane canopy in a field or between fields. This new epidemiological trait helps *X. albilineans* in contaminating rapidly healthy sugarcane plants and, in certain geographical locations, it can be much more important than disease spread by cutting implements. These new epidemiological data of sugarcane leaf scald lead us to propose a revisited infection cycle for *X. albilineans*.

Keywords: epidemiology, epiphytic survival, rainfall
Abbreviations: ELISA, enzyme-linked immunosorbent assay; **CFU**, colony forming units; **ITS**, intergenic transcribed spacer; **PCR**, polymerase chain reaction; **PFGE**, pulse-field gel electrophoresis; **RFLP**, restriction fragment length polymorphism; **XAS**, *Xanthomonas albilineans* selective medium

CONTENTS

INTRODUCTION: DISCOVERY AND DESCRIPTION OF LEAF SCALD OF SUGARCANE

Leaf scald, caused by the bacterial pathogen *Xanthomonas albilineans*, was first recorded in Australia in 1911 by North. It is one of the major diseases of sugarcane (*Saccharum* spp.) and has been reported in at least 66 countries worldwide (Rott and Davis 2000). This vascular disease caused severe yield losses when noble canes (*Saccharum officinarum*) were widely cultivated in the early decade of the 20th century, and disease impact was reduced by cultivating resistant varieties. However, outbreaks periodically occurred, especially in the Caribbean basin in the 1990s, and these outbreaks were attributed to pathogen variation. First description of variability in *X. albilineans* was based on immunological properties and three serological variants, called serovars, were identified in a world collection of different isolates of the pathogen (Rott *et al.* 1994). Subsequently, genetic and pathogenic variations were also reported but no correlation was found between genetic and pathogenicity markers so far (Davis *et al*. 1997; Daugrois *et al.* 2003; Champoiseau *et al.* 2006b; Rott *et al.* 2010). Recent sequencing of the genome of *X. albilineans* revealed that this pathogen does not possess a Hrp (hypersensitive response and pathogenicity) type III secretion system that is generally found in plant pathogenic bacteria (Pierretti *et al.* 2009). Additionally, several new candidate pathogenicity factors that could play a role in plant colonization and symptom expression were identified using transposon mutagenesis (Rott *et al.* 2011).

Symptoms of systemically infected sugarcane

Symptoms of systemically infected sugarcane are well described, and two different forms (chronic and acute) of the disease are known to occur (Rott and Davis 2000). The

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Table 1 Description of foliar symptoms of sugarcane leaf scald resulting from aerial spread of *Xanthomonas albilineans*.

Location (reference)	Date of first description	Size of chlorotic symptom	Size of necrotic symptom	Description
Guadeloupe (Daugrois *et al.* 2003)	1993	2-4 mm wide	3-20 cm long and 0.5-1 cm wide	Elongated necrotic stripe continued by a yellowish stripe parallel to the veins
Florida (Comstock 2001)	1994	3 mm maximum	Up to 25 cm long	Tan-brown necrotic lesions with a pencil-line extending from the edge of the necrotic lesions
Mauritius (Autrey *et al.* 1995)	1989-1991	2-4 mm wide	-	Cream to yellow stripe with reddish flecks; stripe turning necrotic

chronic form is characterized by different symptoms such as white to yellow chlorotic stripes that can be thin like a pencil-line or reach several millimeters wide. Emerging leaves may also show extensive white chlorosis. When disease progresses, leaf stripes and chlorosis turn into necrosis and leaves dry out and wilt. Leaf extremities curl inwards, giving a scalded appearance to the plant and the name "leaf scald" to the disease. A common symptom in mature cane is the abnormal development of side shoots all along the stalk, and the side shoots at the bottom of the stalk are generally the most developed. The acute form is characterized by a sudden wilting of mature stalks, but seems to be limited to highly susceptible sugarcane cultivars. Systemically infected canes do not always exhibit symptoms, and stalks can remain infected by the pathogen for several months without showing symptoms. These apparently healthy but infected plants are in a latency phase (Ricaud and Ryan 1989), which comes to an end for reasons that are not elucidated so far.

Leaf scald management

Leaf scald is best managed by planting healthy seed-cane and resistant cultivars. Healthy seed-cane can be obtained from plants issued from disease-free tissue culture propagation (Flynn and Anderlini 1990; Feldmann *et al.* 1994), or from hot-water treated plants. A 48-h soak in cold running water (15-25°C) followed by a 3-h hot water soak at 50°C can clean sugarcane cuttings from *X. albilineans* (Egan and Sturgess 1980). Use of resistant cultivars to manage leaf scald is highly recommended (Walker 1987), but screening of resistant cultivars is difficult because of the occurrence of latent infections and variation of the pathogen. Therefore, improved diagnostic techniques that allow detection of the pathogen in symptomless plants must be used (Comstock and Irey 1992). Leaf scald management could also be improved by using endosymbiontes such as *Gluconacetobacter diazotrophicus*, a bacterium antagonistic to *X. albilineans* (Blanco *et al.* 2005).

Leaf scald diagnosis

Various diagnostic methods can be used for detection and/or identification of *X. albilineans*, including isolation on selective culture media, plant inoculation, and biochemical, immunological and molecular assays. When immunofluorescence, ELISA, and latex flocculation were compared, direct ELISA was the most sensitive method and it resulted in the detection of bacteria at the low concentration of 10^4 cells per ml (Autrey *et al*. 1990). When immunoassays such as ELISA and tissue blotting were compared to isolation of *X. albilineans* on Wilbrink's medium, ELISA was less effective than the two other techniques (Comstock and Irey 1992). Although isolation of *X. albilineans* with selective media is more time consuming than other techniques, it has proved to be very efficient in detecting the pathogen in diseased and symptomless plants (Davis *et al.* 1994). The *Xanthomonas albilineans* selective medium (XAS), based on Wilbrink's medium and developed by Davis and collaborators (1994), contains several antibiotics and fungicides that facilitate selective isolation of *X. albilineans* in culture and identification of this relatively slow growing bacterium.

A first PCR-based diagnostic test to identify *X. albilineans* used primers targeting genes involved in biosynthesis of the toxin albicidin produced by *X. albilineans*. This test proved to be very efficient in detecting the pathogen *in vitro* and in sugarcane juice, especially in multiplex PCR (Davis *et al*. 1998). Specific PCR amplification of a 288 bp DNA product of *X. albilineans* was also obtained using primers based on multiple sequence alignments of ITS sequences (Pan *et al.* 1999). Additionally, primers designed from DNA repetitive (BOX) and enterobacterial repetitive intergenic consensus (ERIC) sequences generated fingerprints of *X. albilineans* that permitted a clear differentiation of this pathogen from the other bacteria (Lopes *et al*. 2001).

Disease spread and pathogen transmission

Leaf scald is known to be transmitted mechanically by knives and harvesters, and by planting infected cuttings originating from symptomless plants (Ricaud and Ryan 1989). The pathogen was also found in the rhizosphere of infected roots, suggesting the possibility of transmission of *X. albilineans* by root-to-root contact (Klett and Rott 1994). However, other means of leaf scald pathogen spread have been suspected to occur, especially since the leaf scald outbreaks that occurred in the late 1980s and early 1990s.

EVIDENCE FOR AERIAL TRANSMISSION OF *X. ALBILINEANS* AND ASSOCIATED SYMPTOMS

Disease symptoms that could not be attributed to mechanical transmission were first observed in 1989, in Mauritius, on maize that was grown between rows of sugarcane. These maize plants exhibited foliar stripes from which *X. albilineans* was isolated (Autrey *et al.* 1995). At the same time, unusual leaf symptoms were also found on newly released sugarcane cultivar M695/69. These symptoms were described as yellow stripes, resembling closely to those of gumming disease caused by *X. axonopodis* pv. *vasculorum*, and turning necrotic with time (**Table 1**). *X. albilineans* was isolated from the leaf blade up to 30 cm down from the edge of the necrotic tissue. Because the pathogen was also isolated from guttation droplets on leaves, the authors hypothesized that *X. albilineans* was aerially transmitted and that epidemics of leaf scald observed during the 1989-1991 period were primarily due to aerial transmission followed by mechanical spread of the pathogen.

Presence of *X. albilineans* in guttation droplets of sugarcane and sweet corn was first reported in Brazil by Sordi and Tokeshi (1986), and Klett and Rott (1994) provided proof for the presence of *X. albilineans* in the leaf canopy of sugarcane in Guadeloupe. In this later geographical location, the pathogen was isolated from leaf surface water (dew or rain) sampled from sugarcane early in the morning, but *X. albilineans* also was trapped at night with culture plates placed between inoculated sugarcane rows, thus demonstrating aerial spread of the pathogen.

In the 1990s, tan brown necrotic lesions were observed on sugarcane leaves in Florida (Comstock 2001). Symptoms were similar to those described previously (**Table 1**), and variations in number of lesions were observed between sugarcane cultivars. *X. albilineans* was isolated from the necrotic lesions and from the leaf blade up to 30 cm down the leaf blade from the necrotic lesion. However, the patho-

gen was found only up to 15 cm down the leaf midrib. Stalks with foliar lesions were investigated for systemic infection, and only stalks with foliar symptoms extending to the midrib were found infected. It was therefore hypothesized that only a small percentage of sugarcane stalks turn into systemic infection after leaf infection by *X. albilineans* (Comstock 2001). Interestingly, the two most susceptible sugarcane cultivars showed the highest extent of necrotic symptoms.

In Guadeloupe, symptoms that were attributed to aerial transmission of leaf scald were first observed in a sugarcane nursery field in 1993 (J.-H. Daugrois, unpublished data). These symptoms consisted of 3-20 cm long necrotic leaf stripes, originating or not from the leaf margin, and from which a yellowish stripe ran down the leaf parallel to the main vein (**Table 1**). The necrotic stripes were 0.5-1 cm wide, and the chlorotic stripes were 0.2-0.4 cm wide. *X. albilineans* was isolated from necrotic leaves, but not from the stalks. In Guadeloupe, year 1993 was followed by two years of relative drought, and no leaf scald symptom was observed in sugarcane nurseries during that period. With the return to average rainfall in 1996, elongated necrotic leaf symptoms attributed to leaf scald were again observed in a nursery plot established with disease-free tissue cultured plants of cultivar B69566, which is susceptible to leaf scald (Rott *et al.* 1995). *X. albilineans* was isolated from symptomatic leaf samples, but the pathogen was not isolated from the stalk fragments attached to symptomatic leaves. However, three months later, *X. albilineans* was isolated from 6 of 108 (5%) stalks sampled from plants that previously exhibited leaf symptoms attributed to aerial transmission of leaf scald (J.-H. Daugrois and L. Costet, unpublished data). As it was observed in Florida, only few plants with leaves exhibiting leaf scald symptoms, due to aerial transmission of *X. albilineans,* resulted in systemic stalk infection. However, subsequent research showed that sugarcane stalk infection after aerial transmission of the pathogen was linked to climate conditions, and especially rainfall, as described below.

ENVIRONMENTAL CONDITIONS FAVORING CONTAMINATION OF PLANT LEAF SURFACE BY *X. ALBILINEANS*

Contamination of the plant canopy before appearance of disease symptoms is known to occur with pathogenic bacterial species belonging to the genus *Xanthomonas*. Short distance spread of several pathogenic xanthomonads occurs either by dissemination of bacteria by water splashing (Gottwald *et al.* 1989; Milus and Mirlohi 1993; Pruvost *et al.* 1999) or by passive transport of the bacteria by the canopy fauna (Gottwald *et al.* 2007). Additionally, epiphytic life of xanthomonads is promoted by high relative humidity, periodic rainfall and mild to warm temperature (Stall *et al.* 1993). Rain was shown to favor bacterial growth on infected plants (Pietrarelli *et al.* 2006), and subsequent dispersal of bacteria on the leaf canopy (Bock *et al.* 2005). Additionally, tropical climatic events such as tropical storms and hurricanes are able to spread xanthomonads over long distances (Pruvost *et al.* 1999; Gottwald *et al.* 2002).

Conditions that favor epiphytic life and spread of xanthomonads are present in numerous tropical and subtropical areas where sugarcane is grown, including Guadeloupe. Therefore, a study of canopy contamination by *X. albilineans* was set-up with three disease-free and three *X. albilineans*-infected sugarcane plots of susceptible cultivar B69566 grown under different climatic conditions in Guadeloupe. Unlike the two others, one of the three surveyed disease-free plots was not near known inoculum sources of the pathogen. The study was based on assessment of *X. albilineans* populations in water droplets on the leaf surface just after sunrise. In the humid area, when inoculum sources of the pathogen were present either within the plot or in the vicinity, *X. albilineans* was detected on leaves of two month-old canes early in the rainy season. Pathogen population densities increased up to mean values of 10^6 and 10^7 CFU (colony forming units) per ml of water sampled on the leaf surface. When environmental conditions were favorable (rain and presence of inoculum), *X. albilineans* populations reached maximum density values within 3-5 weeks (**Fig. 1**). Surprisingly, pathogen population densities decreased when rainfall decreased or when sugarcane matured (**Fig. 1**). Furthermore, one can expect that the epiphytic population densities of *X. albilineans*, including bacterial populations on both the leaf surface and within the leaf, would be higher than those present in water droplets, because part of the bacterial population is most likely able to reach endophytic sites. Indeed, external and internal leaf associated phytopathogenic bacteria are known to form a continuum due to ingression and egression processes (Beattie and Lindow 1999). Ingression and egression processes are associated to continuum between the inner and outer spaces of the leaves and are associated to leaf lesions and stomata (Smith and Singel 2005).

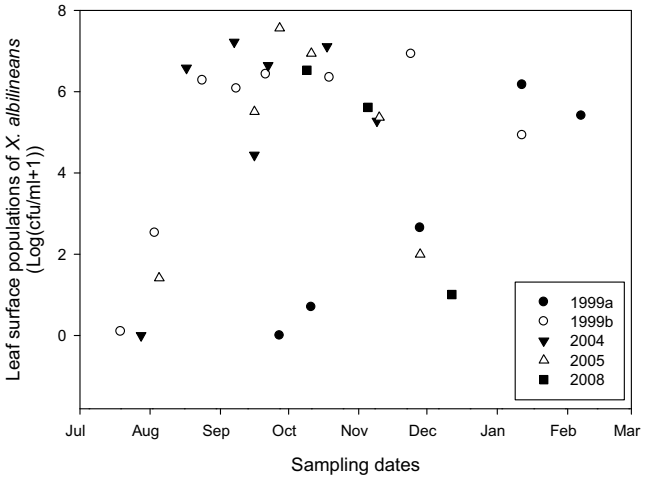

Fig. 1 Population densities of *Xanthomonas albilineans* at the leaf surface of sugarcane cultivar B69566. Bacteria were sampled from water droplets during the first half (July to December) of plant cane crops in 1999, 2004, 2005 and 2008, and each sample consisted of 30 to 60 individual droplets collected from different plants. Field plots were set-up with either disease-free tissue cultured plants (in 1999) or sugarcane cuttings taken from a nursery plot (in 2004, 2005 and 2008). Data were collected from field plots located away (data set 1999a) or in the vicinity (data sets 1999b, 2004, 2005 and 2008) of known inoculum sources of the pathogen. Each sample was plated on XAS medium and population densities of *X. albilineans* were assessed by counting individual colonies after 5 days of growth at 28°C. Each data point corresponds to the mean of bacterial populations from 3 to 40 samples at each sampling date.

In Guadeloupe, leaf canopy colonisation by *X. albilineans* is linked to total rainfall during the first seven months of sugarcane growth (**Fig. 2**), and to the presence of *X. albilineans* inoculum sources in the vicinity of the sugarcane plants. Because stomata opening, which was shown to be promoted by water reserve in the soil (Underwood *et al.* 2007), is also linked to rainfall, one can assume that stomata opening plays a role in *X. albilineans* ingression and egression processes and subsequent pathogen populations on the leaf surface. Therefore, *X. albilineans* may need to face stomatal innate immunity as it was described for *X. campestris* pv. *campestris* (Gudesblat *et al*. 2009). First consequence of leaf canopy contamination is the appearance of necrotic lesions 4-6 weeks after *X. albilineans* detection on leaves, indicating an invasion of internal leaf tissue by the pathogen. In the absence of inoculum sources in the vicinity of sugarcane plants, leaf and stalk contamination was shown to occur after tropical climatic events such as tropical storms and hurricanes (Daugrois *et al*. 2003; Champoiseau *et al.* 2009).

X. albilineans produces a toxin named albicidin that is involved in symptom development, but this toxin exhibits

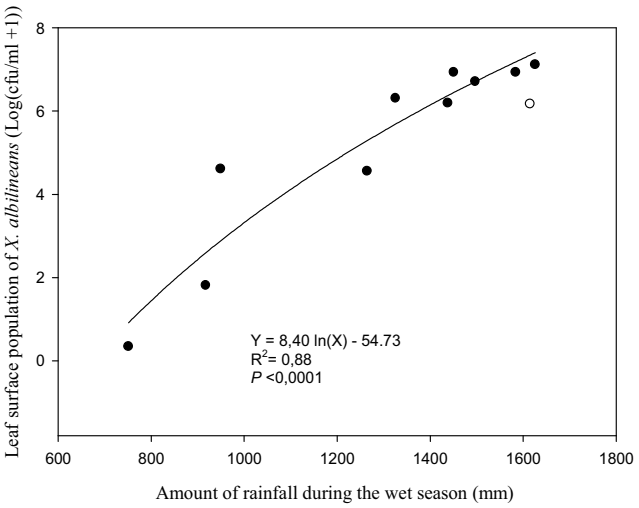

Fig. 2 Maximum average population densities of *Xanthomonas albilineans* at the leaf surface of sugarcane cultivar B69566 at the end of the rainy season (October to December), in relation to total rainfall during the rainy season (June-December). Bacterial population densities were assessed as described in Figure 1. Data are from 6 field plots set up in 1999 (3 fields), 2004 (1 field), 2005 (1 field) and 2008 (1 field), and sampled as follow: 6 fields sampled in plant crop (1999, 2004, 2005 and 2008), 3 fields sampled in first ratoon crop (2000), and 2 fields sampled in second ratoon crop (2001). The open circle represents data from the 1999 plant cane crop of the plot located away from *X. albilineans* inoculum sources. These latter data were not used for the regression calculation.

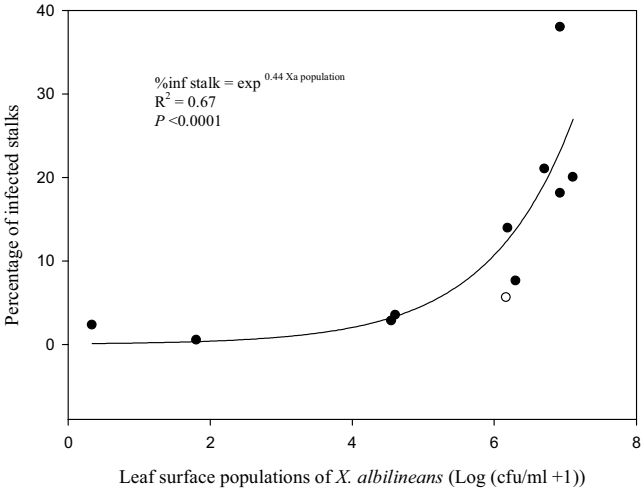

Fig. 3 Relationship between *Xanthomonas albilineans* leaf surface population densities during the wet season (June-December) and the number of infected stalks of sugarcane cultivar B69566 at harvest time (May-June). Bacterial population densities were assessed as described in **Fig. 1**. At the end of the crop cycle, 90 to 300 sugarcane stalks were sampled for diagnosis of *X. albilineans* by stalk blot isolation with XAS medium. Data are from 6 field plots set up in 1999 (3 fields), 2004 (1 field), 2005 (1 field) and 2008 (1 field), and sampled as follows: 6 fields sampled in plant crop (1999, 2004, 2005 and 2008), 3 fields sampled in first ratoon crop (2000), and 2 fields sampled in second ratoon crop (2002). The open circle represents data from the 1999 plant cane crop of the plot located away from *X. albilineans* inoculum sources. These latter data were not used for the calculation of the regression.

also antimicrobial properties. Additionally, virulent strains of *X. albilineans* are able to invade the leaf canopy and replace leaf surface populations of other epiphytic bacteria such as avirulent strains of *X. albilineans* (Daugrois *et al*. 2003). Therefore, albicidin most likely contributes to pathogen competition with other epiphytic bacteria and invasion of sugarcane xylem (Birch 2001). However, no relationship was found between variation in albicidin production, variation in virulence of *X. albilineans*, and ability to colonize the sugarcane leaf canopy (Champoiseau *et al*. 2006a).

The ability of *X. albilineans* populations to survive and increase on the leaf canopy with sufficient humidity or rain seems to be associated with a specific genetic group of *X. albilineans*. Indeed, all strains associated with the leaf scald outbreaks that occurred in the late 1980s and in the 1990s, especially in Florida and Guadeloupe, belong to the same genetic group, namely PFGE (pulse-field gel electrophoresis)-B, as determined by a restriction fragment length polymorphism (RFLP) study of the whole genome of the bacterium (Davis *et al.* 1997), or ALB-RFLP-B as determined by RFLP of genes (49 kb) involved in biosynthesis of the toxin albicidin (Champoiseau *et al.* 2006a). Surprisingly, although these strains emerged recently in Florida, strains belonging to these genetic groups existed prior to leaf scald outbreaks in other locations, such as in Martinique where the oldest strains of group PFGE-B were isolated in 1932 and 1952 (Davis *et al.* 1997). This observation supports the hypothesis that recent outbreaks of the disease were due to pre-existing and re-emerging strains of *X. albilineans* that may have acquired new pathogenic traits that confer better ability to survive epiphytically and/or to invade the host after aerial spread. However, no functional genomic analysis was able to confirm this hypothesis so far. Surprisingly, *X. albilineans* populations issued from aerial spread showed high variation in aggressiveness (severity of disease symptoms caused after sugarcane inoculation), although no genetic diversity within these isolates was found (Champoiseau *et al.* 2006b). In addition, variation in pathogenicity of the bacteria was not related to geographic or *in planta* locations.

AERIAL SPREAD OF *X. ALBILINEANS* AND SUBSEQUENT STALK INFECTION

As mentioned above, the leaf scald outbreaks that occurred about 25 years ago appear to be associated with changes in the epidemiology of the disease. Until the 1980s, the leaf scald pathogen was thought to be exclusively transmitted by infected cuttings and harvesting tools. Occurrence of aerial transmission of the pathogen associated with specific strains of *X. albilineans* raises several questions, especially regarding potential changes in the impact of the disease and its management. One of the first issues of aerial spread of *X. albilineans* is related to subsequent stalk infection. In Guadeloupe, stalk infection of sugarcane cultivar B69566

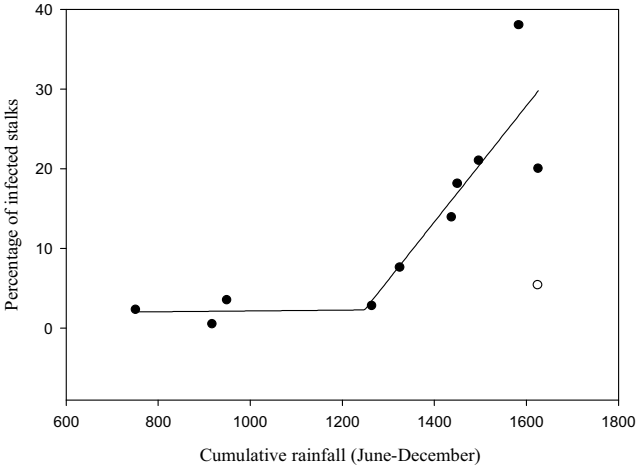

Fig. 4 Relationship between rainfall and stalk infection of sugarcane cultivar B69566 by *Xanthomonas albilineans*. Stalk infection at harvest was assessed as described in **Fig. 3**. The open circle represents data from the 1999 plant cane crop of the plot located away from *X. albilineans* inoculum sources. These latter data were not used for the calculation of the regression.

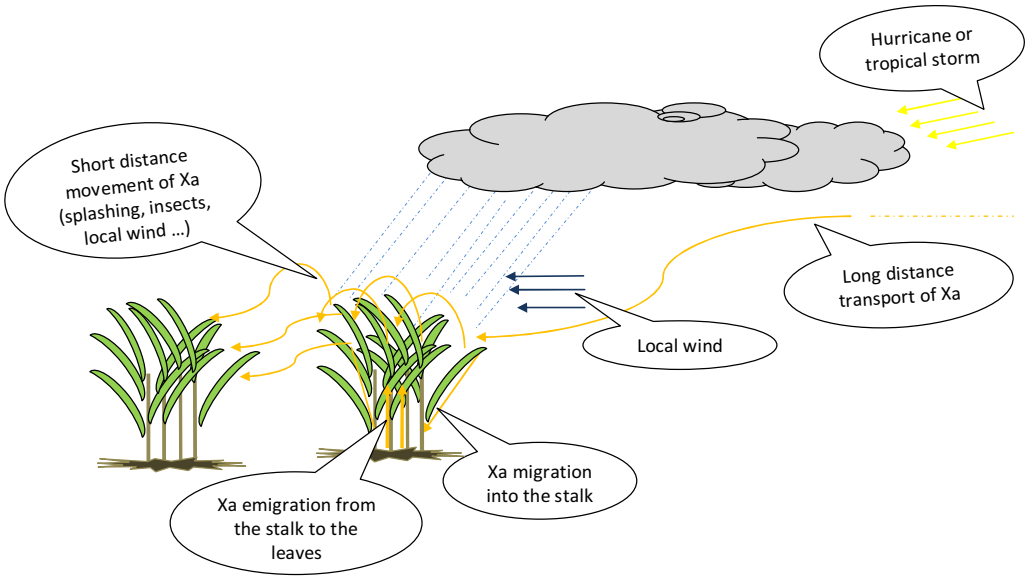

Fig. 5 Schematic representation of the natural spread of *Xanthomonas albilineans* (Xa) and subsequent contamination of a healthy sugarcane plot by the causal agent of leaf scald.

(susceptible to leaf scald) was determined at the end of the crop season by the stalk blot assay using XAS medium (Davis *et al.* 1994). Percentage of infected stalks in ten surveys performed between 1999 and 2008 ranged from 0.5 to 38%, and stalk infection was significantly correlated to epiphytic populations of *X. albilineans* by an exponential regression with a coefficient of determination R^2 of 0.67 and a *P*-value of < 0.0001 (**Fig. 3**).

Because leaf surface population densities of *X. albilineans* are linked to rainfall, the effect of rain on stalk infection was also analyzed. The number of infected stalks was correlated to the amount of rainfall during the wet season when plant growth was maximal (Champoiseau *et al*. 2009). Further analysis conducted with additional data collected from 2004 to 2008 showed that the highest correlation between infected stalks and amount of rainfall during the wet season was obtained with a piecewise equation containing two linear functions (**Fig. 4**). The first linear function indicated that below rainfall value of 1250 mm there is no significant effect of rain on stalk infection of cv. 'B69566' (*P*-value = 0.73). In contrast, the second linear function starting at the rainfall value of 1250 mm showed a significant correlation between rainfall and stalk infection (*P*-value = 0.018). Based on these data, rainfall is associated with the number of stalks infected by *X. albilineans* after aerial spread of the pathogen only when cumulative rainfall reaches a threshold

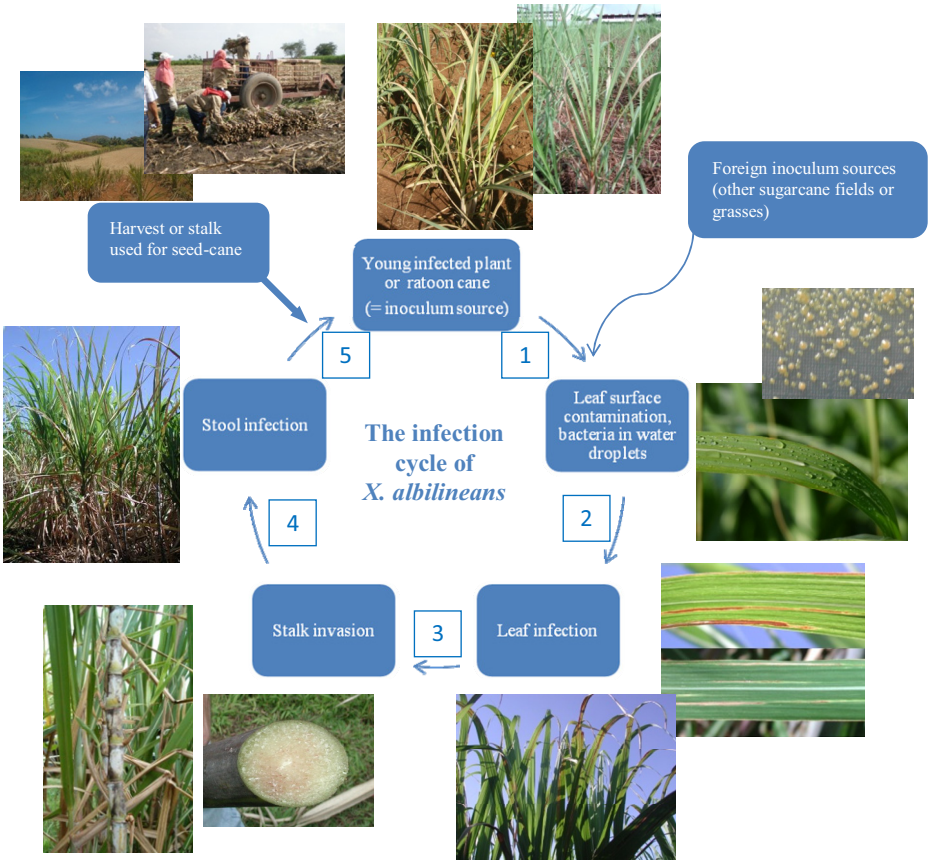

Fig. 6 The revisited infection cycle of *Xanthomonas albilineans*.

value. Therefore, rainfall appears to be critical and a key factor for increase of *X. albilineans* populations on the leaf surface of sugarcane and subsequent colonization of the stalk by the pathogen (**Fig. 5**). It should therefore be possible to predict field contamination by *X. albilineans* and to assess the risks of development of leaf scald epidemics based on rainfall. However, these results need to be confirmed with a wider range of data, including data regarding impact of sugarcane genetic diversity on the epiphytic spread of *X. albilineans* and associated plant contamination.

THE UPDATED AND NEW INFECTION CYCLE OF *X. ALBILINEANS*

Since the first report of the presence of *X. albilineans* in guttation droplets of sugarcane and sweet corn leaves (Sordi and Tokeshi 1986), numerous observations and research data from diverse sugarcane growing locations led us to propose a new infection cycle for *X. albilineans* (**Fig. 6**). In contrast to earlier belief, the leaf scald pathogen can live outside its host plant. *X. albilineans* can be aerially transmitted from undetermined inoculum sources outside the field, or from another contaminated sugarcane field (sugarcane canopy), and reach healthy sugarcane plants under the influence of various climatic factors (**Fig. 6-1**) (Daugrois *et al.* 2003; Champoiseau *et al.* 2009). The pathogen then colonizes the leaf surface, enters the leaves, most likely through open stomata or wounds, and progresses within the xylem, and symptoms may appear (**Fig. 6-2**). From the leaves, *X. albilineans* can migrate into the stalk (**Fig. 6-3**) and infect the stool that may show scalding or side shoots (**Fig. 6-4**). At harvest, the pathogen can be transmitted to healthy plants by means of harvesting tools, but some infected canes may also grow from stools that were previously infected by aerial means. The pathogen can also be propagated by infected cuttings (**Fig. 6-5**). Stalks growing from these aerially or mechanically inoculated stools or from infected planting material are colonized via the vascular system by *X. albilineans* which can later exude from systemically infected leaves. Exuded populations of the pathogen are inoculum sources for a new disease cycle (**Fig. 6-1**).

CONCLUSION

Numerous new data recently became available regarding the epidemiology of leaf scald of sugarcane and the infection cycle of *X. albilineans*, and these data can be used to manage leaf scald disease. As an example, one should avoid transferring sugarcane cuttings from a humid area to a dry area, and should only produce seed-cane in a geographical area with low rainfall. However, there are still numerous aspects of disease epidemiology that need to be elucidated such as mechanisms by which *X. albilineans* spreads aerially, colonizes the leaf surface of sugarcane plants, and invades the vascular system of its host. The genome sequence of *X. albilineans* strain GPE PC73 from Guadeloupe that was recently described (Pieretti *et al.* 2009) should contribute to further decipher the infection cycle of this pathogen. Interestingly, the genome of *X. albilineans* contains a type III secretion system that usually is only found in animal pathogens or symbionts. This later observation suggests that the leaf scald pathogen also has an animal host that remains to be identified to further understand the epidemiological traits of this unique plant pathogen.

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