CASSAVA BACTERIAL BLIGHT IN AFRICA: THE STATE OF KNOWLEDGE AND IMPLICATIONS FOR DESIGNING CONTROL STRATEGIES

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ABSTRACT

Introduced to Africa in the 1970s, cassava bacterial blight caused by *Xanthomonas campestris* pv. *manihotis* (XCM) is present in almost all cropping areas. In the past fifteen years, advances have been made in knowledge of the biology and molecular genetics of XCM, host-parasite relationships and epidemiology of the disease. This paper highlights these recent advances and focuses on the potential of the results obtained to facilitate the design of control strategies suitable for small-scale farmers in Africa.

Key Words: Xanthomonas campestris pv. *manihotis*, epidemiology, variability, host-pathogen interactions, control methods

RÉSUMÉ

Introduite en Afrique depuis les années 1970, la bactériose vasculaire du manioc dont l'agent causal est *Xanthomonas campestris* pv. *manihotis* y sévit dans la majorité des zones de culture de cette plante. Les études entreprises depuis une quinzaine d'années, dans divers pays africains, ont conduit à une bonne connaissance de l'étiologie et de l'épidémiologie de cette maladie. Cet article met en lumière les développement recents et souligne le potentiel des resultats obtenus pour la mise en place de stratégies de lutte adaptées aux utilieux paysans africains.

Mot Clés: Xanthomonas campestris pv. *manihotis*, épidémiologie, variabilité, relations hôte-parasite, méthodes de lutte
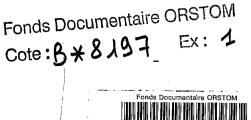
INTRODUCTION

Cassava bacterial blight incited by *Xanthomonas campestris* pv. *manihotis* (XCM), is currently present in almost all the cassava cropping areas in Africa but with very varied incidence (Williams *et al.*, 1973; Maraite and Meyer, 1975; Notteghem *et al.*,1980; Daniel *et al.*, 1981; Boher and Agbobli, 1992). The causal agent of the disease has been known since the beginning of the century in South America and it was introduced to Africa about twenty years ago.

In this paper, a description of the parasite cycle of XCM is followed by a review of knowledge of the biology of the parasite, interactions with the host and the epidemiology of the disease. The knowledge is used to draw up a list of possible control methods with the objective of integrated protection for cassava.

THE EPIDEMIOLOGICAL CYCLE OF *XANTHOMONAS CAMPESTRIS* PV. *MANIHOTIS*

The disease starts during the rainy season with the establishment of the parasite on foliage. Bacteria from contaminated plants or plant debris in the soil are carried to the leaves by rain water or

insects. The bacteria then multiply on the underside of the leaves, where they form microcolonies protected by mucus (Daniel and Boher, 1985a). This epiphytic multiplication contributes to the build up of inoculum sufficient to contaminate lamina tissue through stomata or the wounds that are frequently caused by high winds.

The bacteria colonise the intercellular spaces in the leaf mesophyl and multiply rapidly by division, producing large quantities of a fibrillar exopolysaccharide matrix. Multiplication of the bacteria and expansion of the matrix, combined with lysis of the middle lamella of the host tissue (Boher et al., 1995), account for the rapid colonisation of the lamina, leading to the formation of angular, translucent leaf spots. Leaf blight may occur as a result of a toxin produced by the parasite (Perreaux et al., 1982).

Progress is slowed by contact with the vein perifascicular parenchyma but bacteria can still reach and enter the xylem vessels by lysis of their cell wall. The vascular system of the petiole then provides Xanthomonas with a rapid pathway to the stem.

Blocking of the vessels by the bacterium and its matrix and/or by the gels produced by the plant, or by special structures such as tyloses, impedes sap flow and causes wilting of leaves and entire shoot apices. The bacterium may leave the vessels locally and form lysis pockets in the pith or phloem. In the latter, release of latex combined with bacterial growth causes lysis of neighbouring tissue, followed by exudation of a mixture of latex and bacteria at the surface of the aerial parts of the plant. These bacteria, dispersed in rainwater, can contaminate new leaves. The lytic pockets develop into cankers during lignification.

In the absence of rainfall, the parasite stops spreading in the tissues and the epiphytic populations disappear. The parasite can survive in stem and seed tissues and in plant debris which fall to the ground, but not in the soil (Daniel and Boher, 1985b).

THE BIOLOGY OF THE PATHOGEN

African isolates of XCM display remarkable stability of pathogenicity and limited variability in physiological and biochemical characteristics (Grousson et al., 1990). The strains from the different geographical zones are usually highly aggressive. Hypoaggressiveness has been demonstrated in a few isolates. Avirulent strains are very rare and result from spontaneous mutation. Genetic analysis of XCM isolates indicates that the information required for expression of pathogenicity is in a plasmid fragment of 13 kb.

Use of the restriction fragment length polymorphism (RFLP) technique and various probes specific to the DNA of the pathogen or of ribosomal RNA revealed only one type of profile in African isolates (Verdier et al., 1993). By contrast, isolates from South America can be classified in five groups on the basis of their RNA profiles. The uniform clonal structure of African XCM populations suggests that the bacterium was introduced recently to Africa. The greatest care should therefore be used when additional plant material is introduced from South America to avoid introducing other groups. It is particularly important to avoid using seed harbouring the parasite (Persley, 1979) and exchange of plant material using tissue culture techniques is recommended. Regular monitoring of XCM populations in Africa using molecular biology techniques should be considered in the future to detect any diversification that might result from plasmid exchanges.

The availability of specific probes opens up prospects for their application in the improvement of XCM characterisation techniques and detection of XCM in cuttings. However, it appears that the difficulties encountered in detecting the pathogen in plant material result more from the sampling method employed than from limitations due to the specificity or sensitivity of the probes and antibodies being used.

The low variability of the pathogenicity of XCM in Africa and its stability during in vitro culture ensure stable inoculum for indexing resistance by artificial contamination.

HOST REACTIONS TO PARASITE ATTACK: SEARCH FOR RESISTANCE

The great majority of the cassava cultivars planted in various local areas in Africa are sensitive or very sensitive to XCM. Several varieties derived mainly from inter-specific crosses between

Manihot glaziovii and *M. esculenta* display good resistance to the disease (Hahn, 1978; Hahn *et al.*, 1980). Intercellular and vascular development of XCM is slower in these varieties. At cell level, there is an accumulation of osmiophilic compounds in vacuoles and rapid lignification of the cell walls that are in contact with the parasite. The vascular companion cells rapidly form tyloses that obstruct the vessels. The steady progress of the bacterium is stopped and there is no formation of lytic pockets or exudate (Lambotte and Perreaux, 1979).

Search for resistance can be performed by observing the appearance of the symptoms in the field under strong parasite pressure for several crop cycles. This is an extremely reliable method as it takes into account the inoculum remaining in the stems which is the main source of natural contamination from one growth cycle to the next.

Screening techniques have been developed that are less demanding in time and plant material (Pacumbaba, 1987; Boher and Agbobli, 1992). Inoculation by pricking young shoots of cuttings enables categorisation of the behaviour of a variety in about two months (Boher and Daniel, 1985). An even faster method is the use of delicate manipulations to detect resistance in *in vitro* culture. This can be applied to individuals that have been genetically modified *in vitro*. However, these techniques are not very discriminating as they do not distinguish the intermediate types of resistance observed in the field.

Measurement of the area of the angular lesions that follow inoculation of the lamina should not be neglected as it enables both evaluation of the aggressiveness of an isolate and the observation of a hypersensitive reaction that may halt the parasite in the early phases of its development in the host. This hypersensitivity has not yet been observed in cassava infected with a wild strain but has been induced by avirulent XCM mutants and other *Xanthomonas* pathovars.

EPIDEMIOLOGY

A long rainy season with regular precipitation (alternating heavy rainfall and hot, dry, sunny days) is the main factor that enhances expression of the disease. In addition, poor soil aggravates the deterioration in health of cassava plants.

There is little or no incidence of disease in forest areas. Experience has shown that although conditions of temperature and moisture are little different in the forest and the nearby savannah, the forest biotope has generally more fertile soils and plants grow more vigorously.

Rainwater spreads the disease over short distances. Some leaf-eating insects can carry the bacterium further and contamination can occur through faeces or regurgitation. Two important features account for the rapid spread of the disease once it is introduced: 1) the internal contamination of plant material by bacteria that provides a large amount of inoculum through bacterial exudates produced on sprouts after bud-break; 2) the ability of the parasite to develop as an epiphyte on leaves, combined with spread of the initial inoculum by rainwater and insects.

The pathogen can thus contaminate a whole field very rapidly from only a few infected plants. The start of the epiphytic phase is too fast for it to be possible to prevent more general contamination by removing the first plants displaying symptoms.

Cassava is subjected to attack by several pathogens and pests and it is difficult to estimate harvest losses caused by cassava bacterial blight. Few data are available on this and better knowledge of the incidence of the disease in different ecosystems is required for it to be possible to appraise the economic appropriateness of certain control methods. Such assessments are currently being made in the four countries of the Ecological sustainable cassava plant protection (ESCaPP) project (Yaninek *et al.*, 1994).

POSSIBLE CONTROL METHODS

There are several possible interventions for disease control:

Modification of cultural practices

Improvement of crop nutrition. The soil organic content can be improved on smallholdings by digging in crop residues (this also restricts survival of the pathogen), applying manure or rotating cassava with legumes.

Potassium fertilizer (Adenidji and Obigbesan, 1976) increases resistance to XCM but is difficult for smallholders to obtain. Weeding is

recommended as some weed species including *Eupatorium odoratum* L., *Mariscus sumatrensis* (Retz) Raynal, and *Phyllanthus amarus* Schum. & Tonn.) can harbour the parasite.

Modification of the cropping cycle. Changing the planting date (earlier or later) and crop duration can reduce disease incidence.

Improvement of the quality of planting material. This can be achieved by careful selection of healthy stems when cuttings are made, but farmers are not accustomed to choosing their cuttings according to such criteria. However, they could be taught to recognise the symptoms and thus choose clean or little-contaminated stems for their new plantings as recommended for control of virus diseases of cassava (Thresh *et al.*, 1994).

Healthy plant material could be produced in controlled propagation sites. This would be important, especially in zones with low or medium parasite pressure. The production and distribution of high-quality cuttings is an essential stage in the improvement of cassava production and has proved worth-while in South America (Lozano and Wholey, 1974) and Asia. It is neglected in Africa and should receive more attention. The functioning and management of these propagation fields to supply small farmers remains to be organized and the experience of Otim-Nape *et al.* (1994) in producing virus-free planting material for use in Uganda is relevant. Use of such sites would facilitate improved control of health , the extension of new varieties and control of the introduction of new pathogens and pests. It would also limit the duration of heeling-in of plant material between successive crop cycles. Cassava nurseries to supply cuttings should preferably be sited in forest areas whenever possible to avoid cassava bacterial blight.

Production and distribution of resistant varieties. It has been mentioned above that a number of the varieties adopted widely in Africa have a considerable resistance to cassava bacterial blight and have remained resistant for many years. The genetic base of the resistance is currently limited and should be broadened by utilizing other species of *Manihot* and natural hybrids of

M. esculenta and *M. glaziovii* and introduced in a larger number of locally adapted varieties.

Is it possible to consider deploying another type of resistance such as the rapid blocking of parasite development by a hypersensitive reaction obtained from other species of cassava? The question is for geneticists to consider. However, such resistance would be monogenic or oligogenic and thus is likely to be less durable than the existing polygenic type.

The stems of resistant plants are not immune to contamination after several crop cycles and their use does not avoid the need for regular cleansing of plant material.

Use of biotechnology and biological control. The introduction into the genome of genes controlling the production of bactericides is being studied for other hosts of *Xanthomonas* and might be developed for cassava. This line of research is dependent on the development of a reliable technique for regeneration of cassava *in vitro* and the acceptance of the practice of genetic manipulation for routine use in the future.

Biological control is possible with the help of antagonistic bacteria such as fluorescent *Pseudomonas* (Lozano, 1986). This can be achieved with the help of avirulent strains of the parasite but seems likely to be used only for industrial- type plantations.

CONCLUSION

The divergence between the effective control obtained in research stations and laboratories and the limited progress in adopting such practices for use in smallholdings is striking. The search for new resistance factors does not preclude the development and above all, the promotion of cultural practices adapted to use by small-scale farmers in Africa and aimed at keeping the disease at an economically acceptable level and at maintaining a balanced agricultural system.

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