INVITED REVIEW

THE POPULATION STRUCTURE OF SOME PLANT PATHOGENIC BACTERIA: AN ECOLOGICAL AND ADAPTIVE PERSPECTIVE

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SUMMARY

In the last decades, the population structure of some plant pathogenic bacteria has been assessed by using neutral markers. However, the dynamics of such populations as well as host plant and environment selection, gene flow and genetic drift have been analysed to a lesser extent. Insights into the sequential adaptation occurring between the crop and the micro-organism can also contribute to achieve a more effective control of the disease in the long run. In this review the possible centre of origin, often in relation to the geographic history of the crop, is discussed for some phytopathogenic bacteria, such as *Xanthomonas axonopodis* pv. *citri*, *Xanthomonas axonopodis* pv. *cassavae*, *Xanthomonas oryzae* pv. *oryzae*, *Erwinia amylovora* and *Xylella fastidiosa*, The rapid genetic change of bacteria through lateral gene flow and the efficient exploration of new adaptive solutions by exploiting the "contingency genes" of mutator clones, might also explain the occurrence of new diseases. Host selection, changes in environmental conditions and the introduction of new agronomic techniques, can play an important role in structuring the bacterial populations and in dramatically altering the equilibrium between the host plant and the pathogen. The prediction of such disturbances of equilibrium is fundamental for disease management.

Key words: phytopathogenic bacteria, host selection, lateral gene flow, geographic history of crops, sequential evolution, disease management.

"It is an advantage to a host species to be biochemically diverse, and even to be mutable as regards genes concerned in disease resistance".

J.B.S. Haldane, 1949

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INTRODUCTION

In recent years, the widespread availability of molecular tools has provided plant pathologists with many neutral markers (genetic markers that are conserved within taxa, but different between taxa; Gurtler and Mayall, 2001) with which they can assess the population structure of pathogens. In particular, considerable achievements have been made in elucidating the population structure of some phytopathogenic bacteria since the seminal paper of Leung *et al.* (1993). The literature shows that most studies have, partially or totally, assessed the facets that such investigations have to clarify: i) the amount of genetic variation among individuals in a population; ii) the ways in which this variation is partitioned in time and space; and iii) the genetic relationships among the individuals within and between lineages (Leung *et al*., 1993). Other important aspects, however, influencing the overall population structure of bacterial pathogens, such as host and environmental selection, gene flow (the exchange of genes between populations), and genetic drift (the change in genotype or gene frequencies due to chance alone), have been analysed to a lesser extent. Also studies regarding genetic or evolutionary dynamics (Maynard Smith *et al*., 1993, 2000) have been rarely performed. Consequently, for most plant pathogenic bacteria we still do not know if their current population structure is clonal (the role of recombination of large chromosomal segments is not high enough to randomize genomes or break up clonal associations), panmictic (the random association between loci yielding chromosomal diversity among bacterial lineages) or epidemic (significant association between loci arising only because of the recent epidemic phase that is favoured by selection) *sensu* Maynard Smith *et al*. (1993). Studies on population structure can also establish the source of inoculum over long and short distances, track the movement or population dynamics of different genotypes and, in particular cases, disclose the origin of a plant pathogen (Milgroom, 1997).

The aim of this review, after definition of possible ecological interactions in an adaptive context between the host plant and the pathogen, is: i) to point out the centre of origin of some plant pathogenic bacteria; ii) to

frame the occurrence of new diseases in an adaptive context; iii) to briefly examine current knowledge on the population structure of some phytopathogenic bacteria especially in relation to genetic diversity of the hosts and to changes in climate and weather conditions. Unfortunately, for some hosts the extensive breeding that occurred in the last century and the subsequent world-wide distribution of few cultivars, disturbs studies aiming to clarify the "natural" adaptation of the pathogen to a host plant in a particular area. However, for some host plants still cultivated in a more traditional way (i.e. by exploiting the same local plant germplasm since ancient times) it is still possible to infer the dynamics between the plant and the micro-organism in an only slightly modified ecosystem.

PLANT PATHOGENIC BACTERIA IN AN ECOLOGICAL AND ADAPTIVE CONTEXT

Many plant pathogenic bacteria seem strictly co-adapted to their host plants, and, to a certain extent, to depend on them. In fact, if they killed their hosts, they would probably die off as well. Consequently, except when they are in an epidemic phase, the pathogenic behaviour of most phytopathogenic bacteria would seem to be evolved to not completely destroy the host. To reinforce this view, there is also the apparently weak capacity of survival when they are not in direct contact with the host. Plant pathogenic bacteria do not produce resting spores, and their capacity to survive starving over long periods in other environments other than the plant or plant parts would seem limited (Goto, 1992). To what extent a viable but non-culturable state of bacterial plant pathogens can be postulated is still far from clear (Grey and Steck, 2001). To radicalise these concepts, if the crop is abandoned, the associated phytopathogenic bacteria would probably go extinct. The postulated survival strategy "aimed" at not-eliminating the host (i.e. group selection), however, is almost universally rejected by evolutionary biologists (Ridley, 1996). In fact, while the long-term interest of pathogenic species dictates that it should not destroy its resource, it is postulated that natural selection will favour individual pathogens that reproduce themselves in the greatest number over those that restrain themselves in the interest of preserving their host. In this view natural selection will favour individual pathogens that can consume as much of the host as possible, as fast as possible, before any other pathogen takes advantage of the resource. Consequently, the interaction between the host plant and the pathogen involves, in any ecological niche, the resistance and tolerance genes displayed by the plant to counteract the various virulence and avirulence genes possessed by the micro-organism (Gandon *et al.*, 2002). If this is the case, as far as microbial adaptation to the host is concerned, we can refer to: i) co-evolution (i.e. when two species influence one another's evolution over a prolonged period) between the host and pathogen in natural environments (i.e. wild vegetation), yielding a final equilibrium between the host and the microbe(s) with rare occurrence of epidemics (Dinoor and Eshed, 1984; Price, 1992); and to ii) sequential evolution, when the change in one lineage (the host plant) selects for change in the other lineage (the bacterium). In fact, the pathogen has to change to keep up when the host germplasm changes (introduction of new cultivars). In areas where the environment is particularly conducive for the pathogen and severe outbreaks frequently occur (see also next paragraph), the natural occurrence of harmful micro-organisms can dramatically reduce the economic viability of the host crop.

THE CENTRE OF ORIGIN OF PLANT PATHOGENIC BACTERIA

When a high level of phenotypic and genetic diversity is exhibited by a cultivated host plant or by its wild relatives in a certain area where archaeological and cultural evidence is available, it is possible to indicate the origin (i.e. the area where the wild progenitor(s) of the crop is still living) of a cultivated plant (De Candolle, 1882; Vavilov, 1926; Harlan, 1971). In contrast, for bacterial species showing strict association with a host plant it is not possible with certainty to establish its geographical origin or to assess when the coevolutionary process started. The matter is further complicated by the endophytic life stage that some plant bacterial species possess and by the latency of the infection process. In fact, in cases of strong influence by the environment, a host species introduced from another area and hosting cells of the pathogen in a latent phase, can subsequently become diseased after the appearance of new climatic factors and/or stress conditions for the host that favour disease development. The first record of a pathogen in a certain area does not necessarily mean that the crop cultivated in that place is the origin of the pathogen. Moreover, plant pathology is a relative modern science and a new disease record cannot always take into account the various displacements that a host might have had in the past. However, in some cases it seems that there is sufficient evidence for an overlap in the centre of origin of the pathogen with the centre of origin of the crop.

Xanthomonas axonopodis pv. *citri* (Vauterin *et al.*), the causal agent of citrus canker, apparently originated in tropical Asia (i.e. southern China, India, Indonesia), and *Xanthomonas oryzae* pv. *oryzae* (Van den Mooter *et al.*), the causal agent of bacterial blight of rice, seems to have originated in south Asia. For *X.a.* pv. *citri*, convincing evidence came from Lee (1918) who found natural infection caused by this bacterium on *Fortunella hindsii*

(Champ. *ex* Brenth) Swingle, on mountain tops in southern China and from Fawcett and Jenkins (1932) who observed canker lesions, on specimens of *Citrus medica* L. collected in India on 1827-31, and of *Citrus aurantifolia* (Chrism. *et* Panz.) Swingle, collected in Indonesia on 1842-44, in herbaria kept at Kew, England.

X.o. pv. *oryzae*, which was reported for the first time on 1884 from Japan (Goto, 1992), is frequently found associated with aquatic weeds and rice straw in several areas of south-east Asia where rice (*Oryza sativa* L.) has been cultivated for several millennia (Thind and Brar, 1996) (an alternative hypothesis is given in the subsequent paragraph). These two examples may show co-evolution between pathogen and host plant both occurring in the areas where the crop was first cultivated, and also involving the wild ancestors.

By contrast, some other phytopathogenic bacteria are ubiquitous. Probably, the best examples are *Agrobacterium* spp., soil inhabitants reported world-wide, in different environments and from herbaceous and woody host plant species, cultivated or not (De Cleene and De Ley, 1976, 1981; De Cleene, 1985). For another important pathogen, *Ralstonia solanacearum* (Smith)Yabuuchi race 3, biovar 2, evidence (partly based on presence of resistance in wild *Solanum* species in the Andes region) for its endemic presence and possible origin in South America and subsequent clonal spread by the potato tuber are given by Janse (1996). The ubiquitous race 1 of *R. solanacearum* is genetically much more diverse and has a much wider host range (Prior *et al*., 1998).

By taking into consideration the origin and the historical geography of a crop, records of previously unreported sudden and severe epidemics, epidemiological data, and also the host range and population structure of the pathogen, it is possible, in some particular cases, to infer at least the continent where, presumably, a certain bacterium began to infect a crop. For the following three pathogens the continent of origin seems to be ascertained.

From historical data on pear and apple cultivation, the first record of the destructive "fire blight" symptoms, and absence of such symptoms over more than 2,000 years in pome fruit cultivation in Asia and Europe, it is likely that *Erwinia amylovora* (Burr.) Winslow, originated on the American continent, most probably from North America (Baker, 1971). Pear (*Pyrus communis* L.) and apple (*Malus pumila* Mill.; synonym *Malus domestica* Borkh.) originated in central Asia and were cultivated in western Asia and Europe after the invention of grafting, as documented in Greece by Theophrastus, 300 B.C.. Moreover, crab apple (*Malus sylvestris* Mill.), a wild species present all over Europe, has been widely utilised since prehistoric times either for fresh consumption or for cider production. The striking symptoms induced by *E. amylovora* and the rapid and destructive epidemics it causes could not have escaped the early observers such as Greeks, Romans and Arabs (Savastano, 1891) or the first European investigators of plant pathology (Fabricius, 1774), and, more convincingly, were not reported from Europe before 1957 (Crosse *et al*., 1958). *E. amylovora* seems to be endemic in the eastern United States (Arthur, 1886; Van der Zwet, 1968) where the bacterium, probably, coevolved with indigenous Rosaceous species such as wild crab apples, *Crataegus* spp, *Sorbus* spp., and *Amelanchier* spp. (Baker, 1971).

The American continent, most probably southern North America (Mortensen *et al*., 1977), is also the centre of origin of another important plant pathogenic bacterium, namely *Xylella fastidiosa* (Wells *et al*.). The most convincing evidence for this is the appearance of Pierce's disease soon after the repetitive introduction in North America of European grapevine (*Vitis vinifera* L.) cultivars during the late 18th Century. Cultivation of *V. vinifera* in Europe dates back to the early bronze age (i.e. between 4,500 and 2,000 B.C.) in northern Greece, whereas in Asia the earliest evidence of grapevine cultivation and of wine making is from Iran and Baluchistan and dates before 2,000 B.C. (Liuni, 1988). On the American continent, *V. vinifera* was first introduced to central and south America by the Spanish and Portuguese during the 16th Century. By the late 18th Century European cultivars reached Alta California where most attempts to establish the grapevine failed. Apart from some insects, *X. fastidiosa* was the main obstacle, as documented by Pierce (1882). Native Americans exploited about a dozen of native grapes; most widespread was the wild muscadine *Vitis rotundifolia* Michx.. Only after development of hybrids between the native grapes and *V. vinifera*, could viticulture develop.

Citrus spp., originating from Southeast Asia (Simmonds, 1986), were introduced to America by the Spanish and Portuguese and was contemporary to that of grapevine. From 1493 onward, the sweet orange (*Citrus sinensis* Osb.) and other citrus fruits reached Haiti, Mexico, Peru, Panama, Brazil, Cuba and Florida. Interestingly, the first record of *X. fastidiosa* infection on citrus in Brazil is quite recent (Rosetti *et al*., 1990), and the pathogen is not reported on *Citrus* spp. in North America (i.e. Florida), where the crop is also widely grown. Whether *X. fastidiosa* is endemic in South America also or the occurrence of citrus variegated chlorosis in Brazil is a case of pathogen movement by means of vector(s) and/or alternative hosts to a new area is not known.

A pathogen most probably originating from and still restricted to the East African highlands is *Xanthomonas axonopodis* pv. *cassavae* (Vauterin *et al*.), the causal agent of cassava (*Manihot esculenta* Crantz.) bacterial necrosis (Verdier *et al*., 1994). Cassava, also known as manioc, was domesticated in north-eastern South America, where first evidence of cultivation is from about 2,000 B.C. (Sauer, 1993). By the mid 17th Century, the Portuguese had introduced manioc to several places in West

Africa. For East Africa, the first definite record was made in 1739, when Reunion Island received manioc cuttings from Brazil. From there, introduction to Madagascar and the East African mainland took place (Sauer, 1993). Interestingly, in the center(s) of origin of manioc, another xanthomonad, namely *Xanthomonas axonopodis* pv. *manihotis* (Vauterin *et al.*) and not *X.a.* pv. *cassavae*, is widespread and the occurrence of *X.a.* pv. *cassavae* is not documented (Van den Mooter *et al.*, 1987; Verdier *et al.*, 1994). *X.a.* pv. *manihotis* is also commonly found in African countries, probably through distribution of clonally multiplied and contaminated planting material.

Fire blight, Pierce's disease, citrus variegated chlorosis and bacterial necrosis of cassava are examples of how man directly influences evolutionary processes by "simply" displacing the plant germplasm between continents and allowing endemic micro-organisms to adapt to the new environment, possibly utilising virulence factors previously (i.e. when the bacterium is coadapted to indigenous wild plants) not displayed in a destructive way.

THE OCCURRENCE OF NEW DISEASES

To investigate the origin of bacterial plant pathogens is not only a matter for speculation. The sudden appearance of a new disease in a crop can also be read in terms of adaptation of a bacterium. The evolution and adaptation of bacteria (i.e. increase of their genetic variability) to diverse and almost infinite environments, is achieved either by the classical means, such as conjugation, transformation, transduction, or by lateral or horizontal gene flow (a proportion of the genetic diversity of the bacterium has been obtained through the acquisition of sequences from related or distantly related micro-organisms). This means the acquisition from other bacterial strains or species, of accessory genetic elements such as plasmids, transposons, integrons and phages containing, in some cases, virulence factors or "pathogenicity islands" (Levin and Bergstrom, 2000; Ochman *et al.*, 2000; Spiers *et al.*, 2000; Vivian *et al.*, 2001). The response time in these cases may be very short, allowing virulence factor(s) acquisition in a few steps (Lee, 1996; Mecsas and Strauss, 1996). It has been shown that the presence of efficient donors in heterogeneous bacterial populations can accelerate plasmid transfer. Such donors allow other bacteria to acquire the plasmid in a matter of days, whereas in the absence of such strains, plasmid dissemination would take years (Dionisio *et al.*, 2002). Most of the horizontally transferred DNA is part of the "flexible" gene pool (Fig. 1). In addition, bacteria have specific loci that are highly mutable, the so-called "contingency genes". Such loci allow efficient exploration of solutions to new and unpredictable aspects of the host environment (Moxon *et al.*, 1994; Taddei *et al.*, 1997). The high frequency of "mutator" clones (lineages

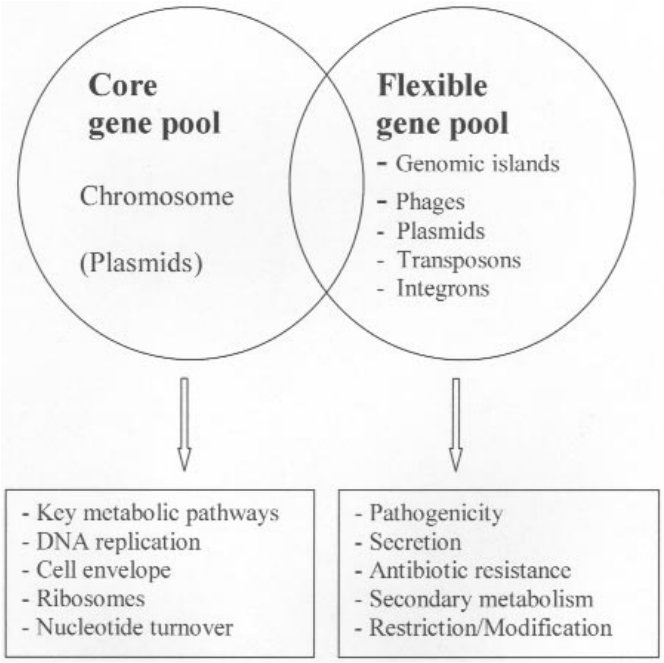

Fig. 1. Proposed model of the DNA pools in the genomes of prokaryotes. Some functions encoded by the pools are given in the lower part of the diagram. (Redrawn from: Hacker and Carniel, 2001).

having genes that increase the rate at which variation is generated by mutation) in pathogenic bacteria suggests that an increased mutation rate is important to the adaptation of parasites (Haraguchi and Sasaki, 1996). Genetic mechanisms such as the spontaneous inactivation of a transcriptional regulator gene, generating random phenotypic variation and genetic rearrangement at high frequency, have been found in *R. solanacearum* (Brumbley *et al.*, 1993; Nakatsu *et al.*, 1998). Changes in *X.a.* pv. *manihotis* haplotype frequencies within samples taken from cassava fields occurred in less than one year, and the appearance of a new population structure occurring during a single crop cycle were demonstrated (Restrepo *et al.*, 2004). Race shifts in *Xanthomonas axonopodis* pv. *vesicatoria* (Vauterin *et al.*) strains occurred within a season in pepper fields (Kousik and Ritchie, 1996). This was explained by an apparent loss of the plasmid carrying an avirulence gene and/or acquisition of an insertion element inactivating an avirulence gene carried by the host (Kousik and Ritchie, 1996).

Presence of the *hrp* gene cluster and other virulence genes on a plasmid, the relevant similarity and co-linearity of *hrp* operons between *Pantoea agglomerans* pv. *gypsophilae* (Gavini *et al.*), *Pantoea agglomerans* pv. *betae* (Gavini *et al.*), *E. amylovora*, and *Pantoea stewartii* subsp. s*tewartii* (Mergaert *et al.*), allowed Manulis and Barash (2003) to postulate that the two gall-inducing *P. agglomerans* pathovars evolved recently.

Also the widespread existence of apparently non-pathogenic xanthomonads associated with different

symptomless plant species (Vauterin *et al*., 1996) raises questions about their origins. Indeed, the authors argued that some isolates might be pathogenic *Xanthomonas* strains with as yet unknown host plant(s), but able to live as epiphytes on many plants. What induces epiphytic xanthomonads to become putative pathogens is still unknown. Since rice seed does not appear to be the original source of bacterial blight (Gonzalez *et al*., 1991), the possibility that epiphytic and/or endophytic xanthomonads associated with perennial or aquatic weeds can infect cultivated rice, after the acquisition of virulence genes, might explain the presence of *X.o.* pv. *oryzae* in most rice-growing areas of the world.

An example, known personally to the author, of the apparently sudden and recent appearance of a new pathogen, is the occurrence of *Pseudomonas avellanae* (Janse *et al*.) on hazelnut (*Corylus avellana* L.) both in northern Greece and central Italy. Questions to answer, in an ecological and adaptive context, are the following: i) is the pathogen a common, possibly endophytic, inhabitant of the plant species that has become pathogenic after extensive cultivation of the crop in subacidic soils that stressed the metabolism of the plant?; ii) did the pathogen escape from the submediterranean forests where it lives coadapted with wild hazelnut, and has it found in the cultivated trees a more suitable niche?; iii) were the virulence factors of *P. avellanae* acquired by lateral gene transfer from other pseudomonads?.

THE POPULATION STRUCTURE OF PLANT PATHOGENIC BACTERIA IN RELATION TO THE GENETIC DIVERSITY OF THE CROP AND TO CHANGES IN CLIMATE AND WEATHER

Geographical genetic structure is apparent for many plant pathogenic bacteria. It has been clearly demonstrated for *X.o.* pv. *oryzae* (Adhikari *et al*., 1995), *X.a.* pv. *manihotis* (Restrepo and Verdier, 1997; Wydra *et al*., 1998), *X.a.* pv. *vesicatoria* (Bouzar *et al*., 1999), *X.a.* pv. *citri* (Cubero and Graham, 2002), *Xanthomonas campestris* pv. *campestris* (Dowson) (Massomo *et al*., 2003), *Xanthomonas* sp. pv. *mangiferaeindicae* (Vauterin *et al*.) (Gagnevin *et al*., 1997), *Pseudomonas syringae* pv. *persicae* (Prunier *et al*.) (Young *et al*., 1996), *P. avellanae* (Scortichini *et al*., 2002), *E. amylovora* (Zhang and Geider, 1997), *R. solanacearum* (Poussier *et al*., 2000) and *X. fastidiosa* (Coletta-Filho and Machado, 2003), although the proportion of genetic variation explainable by the geographic effect (the geographic origin of the host is able to explain a significant amount of the total genetic variation obtained) was not determined.

For some bacteria, such as *X.o.* pv. *oryzae*, *X.a.* pv. *citri* and *X.a.* pv. *manihotis*, the host (plant species or cultivar) seems to influence the composition of the different bacterial lineages (host selection). By contrast, this strict influence seems less evident for *X. fastidiosa* and sweet orange (Coletta-Filho and Machado, 2002). In fact, the genetic variation observed in the bacterium was not related to or dependent on the different sweet orange cultivars from which the strains were obtained. Conversely, the different population dynamics of sharpshooter vectors and the alternative hosts (weeds) where the bacterium can survive, may also play a significant role in structuring the population of the pathogen on a geographical scale (Coletta-Filho and Machado, 2003).

The persistence of different *X.o.* pv. *oryzae* populations in Asia can be explained by: i) spatial partitioning of the host genotypes; ii) slow migration or dispersal of the pathogen; iii) physical geographic barriers (Adhikari *et al*., 1995). Host selection combined with different rice cultivation practices and different weather conditions also seem to play an important role in determining the population structure of this pathogen in south Asia. In fact, the different *X.o.* pv. *oryzae* populations found in the Philippines seem related to the different rice cultivation ecozones: one single traditional cultivar in the cool mountainous highlands *versus* two or three semidwarf, early-ripening rice cultivars in the tropical lowlands (Ardales *et al*., 1996). In addition, the absence in the Phillippines of typhoons affecting Northeast and Southeast Asia that spread the bacterial inocula, would contribute to limit the genetic diversity of the pathogen (Choi *et al*., 1998).

Host selection would also appear very important in structuring the genetic diversity of other xanthomonads. *Xanthomonas arboricola* pv. *pruni* (Vauterin *et al*.), the causative agent of bacterial leaf spot of stone fruit, is characterized by limited variability, and partitioning of strains on a geographic scale was not observed (Zaccardelli *et al*., 1999; Louws *et al*., 2001). This might be due to the extensive distribution of the same peach and Japanese plum cultivars in all areas of cultivation and also to the very limited genetic variability of the host itself. In fact, concerning peach (*Prunus persica* Batsch), a species self-compatible and tolerant to inbreeding, most modern cultivars have originated from breeding programs in the U.S.A. during the early twentieth century, using a very limited number of parents (Aranzana *et al*., 2003); this has drastically eroded the crop's genetic variability. By contrast, where a similar pathogen, such as *Xanthomonas arboricola* pv. *juglandis* (Vauterin *et al*.), has exploited a wider genetic variability of the host, walnut (*Juglans regia* L.), which is traditionally grown in Asia and Europe from seedlings taken from local trees (Malvolti *et al*., 1998), the genetic variability is considerable, and clustering of strains on a geographic scale is possible (Loreti *et al*., 2001; Scortichini *et al*., 2001).

Also environmental factors (temperature and rainfall) may have a major influence on the bacterial population structure fluctuation. Restrepo *et al*. (2004) found that some *X.a.* pv. *manihotis* haplotypes disappeared while

new haplotypes and pathotypes appeared within some cassava fields in a short time period. The authors argued that as a consequence of the El Nino phenomenon, weather conditions (i.e. low rainfall) were unfavourable for the development of the disease. Thus the occurrence of previously undetected bacterial lineages was possible.

Following two sudden minor tropical storms, Gagnevin and Pruvost (2001) found a predominant haplotype (94% of the isolates) of *Xanthomonas* sp. pv. *mangiferaeindicae* occurring in a mango (*Mangifera indica* L.) orchard, established using disease-free material. The same haplotype occurred on a severely infected mango tree, 250 m upwind of the orchard. This predominant haplotype, associated with migration *via* wind-driven rains, probably substituted other haplotypes belonging to epiphytic populations of *Xanthomonas* sp. pv. *mangiferaeindicae* previously present in the orchard.

CONCLUDING REMARKS

Nowadays, agricultural systems may change quite rapidly. Change in climate, introduction of new germplasm, and even "simple" modification of a fertilization scheme may dramatically alter the equilibrium between the pathogen and the host plant. A well-documented recent example comes from rice cultivation in Asia. As stated by Mew (1987), "bacterial blight is not a new disease of rice. Its importance to rice production in tropical Asia, however, was recognized only after the introduction of modern cultivars, which are highly responsive to nitrogen fertilisers. It is now a major rice disease throughout Asia."

Prediction, when possible, of the consequences of genetic change such as described above is fundamental for disease management. Therefore the combination of ecologic and genetic studies of phytopathogenic bacteria and the crop are of great importance. Indeed, from apparently theoretical studies, insight into the spread and control of pathogens can be obtained. Nuismer and Kirkpatrick (2003) have shown that host gene flow is an important determinant of the geographic range of the pathogen and that fluctuation in the rate of host gene flow causes shifts in pathogen population densities. With vector-borne pathogens, reduction in the rate of vector gene flow, due to habitat fragmentation or vector control programs, may have the unwelcome side effect of leading to increases in pathogen density or geographic range.

Even if a disease cannot be totally eliminated by natural selection (i.e. the genes conferring complete resistance cannot become fixed) (Roy and Kirchner, 2000), host plant resistance is still desirable (Leach *et al*., 2001). Needless to say, the durability of host resistance is the outcome of a process evolving in an ecological and adaptive frame.

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